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THE PRESENT MENACE OF TYPHUS FEVER IN EUROPE AND THE MEANS OF COMBATING IT[1]

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SUMMARY

[1] Acknowledgement is made to authors who, during the preparation of this study, have been good enough to keep us informed of their work, acquainting us with their MSS. and observations, and in particular to Professors Ludwik Anigstein, Georges Blanc, Michel Ciuca, Jean Laigret, Edmond Sergent. We also wish to acknowledge our indebtedness to the late Hans Zinsser, to whose memory we would pay a sincere tribute.

Public opinion and health authorities in various countries are at present concerned at the possibility of communicable diseases becoming epidemic on account of war conditions.

The memory of the innumerable victims of typhus fever in Eastern Europe during the first World War gives that disease a place in the forefront of the public mind. The many requests for information concerning typhus that we have received during the past months prompt us to publish here a brief summary of recent information received by the Epidemiological Service of the League, followed by a short account of recent developments in research concerning the methods of combating typhus, and more particularly in regard to vaccines against that disease.

I. Present Epidemiological Situation with regard to Typhus in Europe

A. Historical background

In order to appreciate the significance of the present situation with regard to typhus in Europe we must glance at the past. It is not necessary to go far back in the course of history, as Zinsser did in his attractive but sinister book *Rats, Lice and History*, to realise the dangerous and almost "explosive" character that typhus fever may assume in wartime, i.e. during periods of exodus, privation and famine.

From 1915 to 1920, typhus showed, in the Balkans, in Poland, and even more in Russia, its tremendous force of expansion and destruction.

The epidemic will be remembered, for instance, which decimated the Serbian population and army during the 1915 retreat. It is estimated that one person out of five was then attacked by the disease, which caused over 135,000 deaths in a population of 2½ millions. In the course of the same epidemic 70% of the Austrian soldiers captured by the Serbian Army fell victims to typhus. The war, the invasion and the privations that ensued gave rise in Roumania from 1916–1919 to an epidemic of typhus in which hundreds of thousands of people died—and Roumania then had a population of only about 7½ millions. An eye-witness described to us the horrible picture that certain villages presented during the retreat. The men had left for the army, the women were either dead or dying on their beds, while the children, resistant to the disease, cried with hunger and misery until they were collected and fed by the retreating troops.

In Russia, though the proportions were similar, the absolute figures were of a still more impressive order. From the very full and well-informed report presented by Tarassevitch (1922) to the Health Committee of the League of Nations on "Epidemics in Russia since 1914," it appears that, from the beginning of that war, typhus, which was endemic throughout the country, increased markedly and became epidemic from the time of the Revolution at the end of 1917. From an average of 150,000 a year before the war, the number of reported cases rose to 700,000 in 1918 and to 6,600,000 in 1919, at which figure they remained in 1920 (maximum in February 1920). In 1921 the number declined to 1,200,000. Tarassevitch is of opinion that the actual number of cases during that period amounted to about 25 millions, i.e. nearly one-quarter of the population for which statistics were available.[1]

In spite of their magnitude, these figures do not give a complete picture of the thorough impregnation of the population by the virus at that time. The figures, indeed, refer essentially to adults, since infection in children is not as a rule revealed by symptoms. Moreover, a large proportion of

[1] This estimate is based on the notifications received by health authorities (6,056,000 from October 1st, 1918, to October 1st, 1920), and on correction coefficients varying according to the period and the region, account being taken of the areas for which notifications were entirely lacking and of the incompleteness of notifications during the Civil War.

adults did not develop the disease, not through their escaping infection, but merely because they were immune as the result of a previous attack.

If, nevertheless, some 30% of the adults went through an attack of typhus, then it may reasonably be thought that a considerable proportion of the children who were in contact with them acquired at that time an infection which, although without symptoms, nevertheless resulted in immunity.[1]

A detailed study of the incidence of the disease month by month and province by province shows that it was closely linked up at first with the movements of troops and refugees and later with the extent of famine. The latter is responsible for the revival of the epidemic in 1922, during which year reported cases exceeded 1,300,000.

Since that time, the incidence of typhus fever declined in all countries of Europe, very rapidly at first, then more slowly, as peace permitted the return to a state of social and economic balance.

Typhus lost the epidemic character it had in time of crisis; it became endemic, sporadic, and even disappeared completely, according to the social and economic level of the various countries of Europe where it had prevailed. In Eastern Europe, as in French North Africa, in Egypt, in the Union of South Africa and in Chile, the world economic depression, which began in 1929, influenced the typhus curve, but the fluctuations had nothing like the extent of the wave caused by the World War.[2]

The Civil War which ravaged Spain from 1936 gave rise to the fear of an epidemic outbreak in that country.[3] This did not occur, however, except for a small number of cases in 1939, until the beginning of 1941. It was not so much the direct result of the war as of widespread destitution.

During the first World War no typhus epidemic occurred in France, in spite of louse infestation of the troops, because the country had been previously free from the disease. Similarly, the Civil War did not lead to the expected epidemic because typhus, during the preceding ten years, had only manifested itself in the form of isolated cases, very largely imported from Morocco.

B. The conditions for an epidemic

A number of well-determined conditions are, in fact, necessary for the outbreak of an epidemic.

1. The presence of the virus is necessary, whether it be pre-existing in an area where typhus was either endemic or sporadic, or imported by patients or infected clothing.

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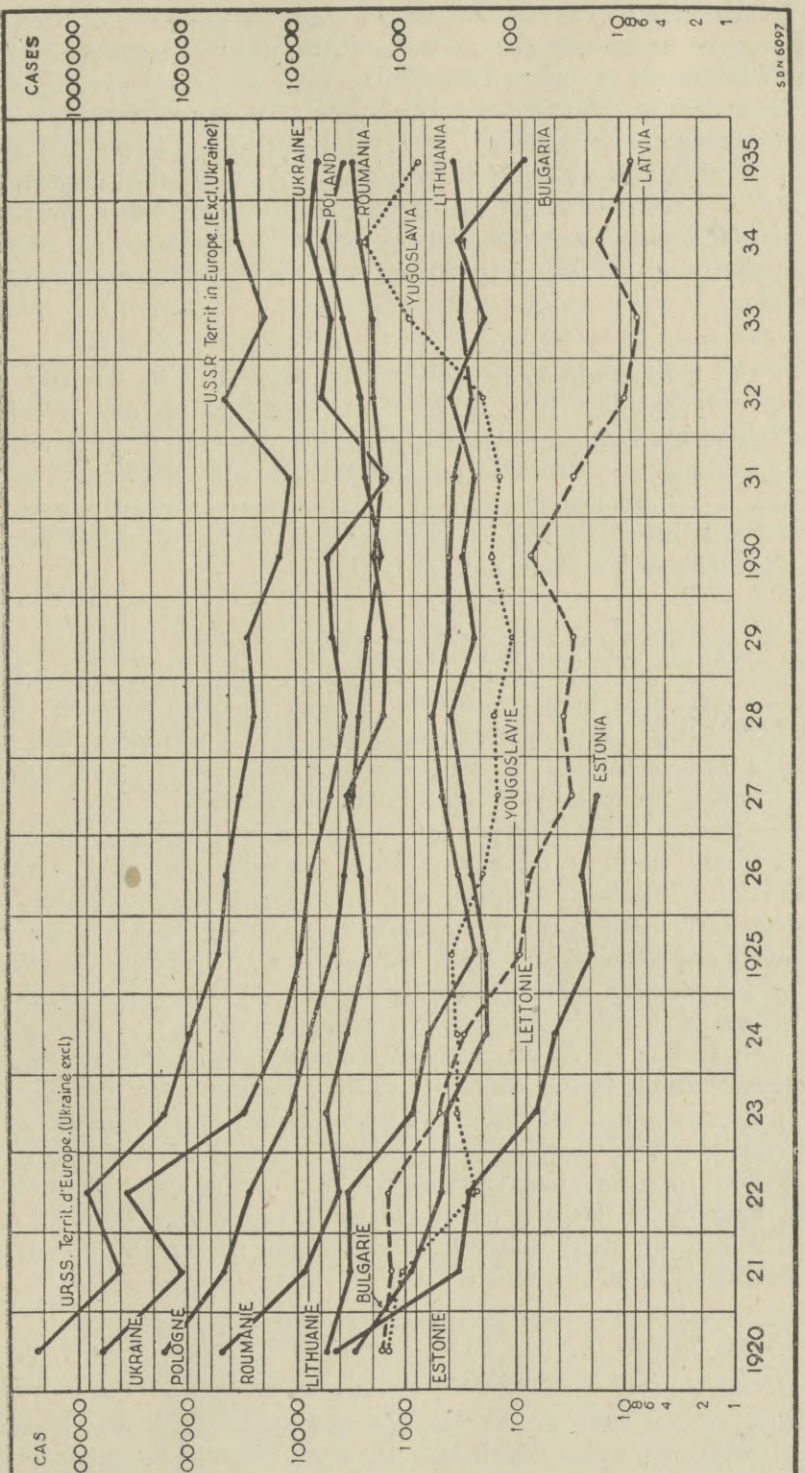
[1] According to the 1920 Census of the U.S.S.R., the number of children under 15 was 53,781,000; the number of persons above 15 was 90,506,000.

[2] The post-war trend of typhus in Europe may be studied in the series of articles published in the *Epidemiological Reports* of the League, and particularly in Nos. 133 (December 1929), p. 480 *et seq.*, and 181 (March 1936), p. 1 *et seq.*

[3] It was because of that fear that the League of Nations sent a technical mission to Spain in December 1936, and, in February 1937, convened in Geneva a group of experts on typhus prevention. This group included, among others, the five inventors of methods of vaccination then tried against the European type of typhus. The practical recommendations of these experts were published in the April 1937 number of the *Bulletin of the Health Organisation of the League* (volume 6, extract No. 6).


CASES
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URSS. Territ. d'Europe. (Ukraine excl)
U.S.S.R. Territ. in Europe. (Excl. Ukraine)
UKRAINE
POLOGNE
ROUMANIE
LITHUANIE
BULGARIE
ESTONIE
LETTONIE
YOUGOSLAVIE
POLAND
ROUMANIA
YUGOSLAVIA
LITHUANIA
BULGARIA
ESTONIA
LATVIA
1920
21
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1925
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1930
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34
1935
SDN 6097

2. The vector of the disease must be sufficiently abundant, i.e. the population must be infested with *Pediculus vestimenti* to an appreciable extent. Such infestation is rapidly produced by overcrowding in sleeping-places, lack of clean linen and spare clothing, lack of soap and material facilities for enduring body cleanliness. It is evident that troops in the field during winter, when soldiers must sleep in dugouts or crowded cantonments where it is difficult to get rid of vermin, are in the best possible conditions for louse infestation and, by way of consequence, for infection by rickettsiae, if military operations occur in a country where typhus is endemic.

3. The third condition for an epidemic outbreak is the existence of a receptive population. This is, of course, generally the case where the population has not been subjected to a recent pandemic or to an effective mass vaccination. Protection acquired through a previous attack of the disease is not absolute, and it is likely that hunger or excessive hardships or any other debilitating cause may bring about a revival of a latent infection through the loss of an immunity balance.

This hypothesis was put forward by ZINSSER (1934) to explain the cases of "Brill's disease" occurring in New York City and Boston among Jews, several years after their arrival from Russian or Polish zones of typhus endemic. This suggestion was made again by Ed. SERGENT, who considered that one form of individual resistance to typhus is "premunition," i.e. a state of lasting *latent* infection, following an attack.

This hypothesis would explain, to a certain extent at any rate, the revival of sporadic typhus at the end of winter, and also the development of epidemics, spontaneous in appearance, in regions of standing endemic, in periods of food shortage—for instance, through crop failure owing to a season of exceptional drought, as in North Africa. Whether the mechanism of the infection is a rupture of the "premunitory" balance in the meaning of Sergent, with a fresh growth and dissemination of the virus from within the individual, or merely the loss of the immunity acquired in the course of a previous attack, leaving him liable to fresh contamination from without, the fact remains that privation considerably facilitates the extension of an epidemic in a zone where typhus is endemic. Individuals coming from an area which for a long time has been free from typhus are, without exception, receptive to infection unless previously vaccinated. Their age and general state of health will determine, *not* the infection itself, but the seriousness of its manifestations.

C. Interpretation of recent events in the light of these conditions

These general rules of epidemiology applied to typhus enable us to understand certain facts which have occurred since the beginning of the second World War.

1. *The campaign of September* 1939 *in Poland* was not accompanied nor immediately followed by an outbreak of typhus,[1] at any rate in the central and eastern parts of the country occupied by German troops. This fact can be explained not only by the short duration of operations and the rapidity of movements, which left no time for the troops to become louse-infested in cantonments or shelters, as might have been the case if these had been overcrowded and in continual use, but also by the fact that the typhus virus was rare. The months of August and September are those when typhus reaches its lowest ebb in Poland and the European part of Russia. In September, the number of cases notified falls in normal years to a dozen or more, against five to six hundred and more (i.e. thirty times more) in May, when the endemic reaches its peak. Moreover, the western provinces of Poland (Pomerania, Posnania, Silesia) were, as a rule, free, while the Voivodies of Lodz and Warsaw in the centre of the country showed but a low endemic or sporadic incidence of typhus.

2. *Increase in typhus endemic in Poland.* Though the conditions necessary for an epidemic did not exist during the campaign, the latter left sequels distinctly favourable to the development of the typhus endemic. Invasion had caused masses of the inhabitants of Posnania and Polish Pomerania and Silesia to flee eastward. The destruction of certain towns close to Warsaw increased congestion, which later on was made worse by the occupation authorities' systematic spoliation of Polish proprietors in certain areas in the north-west. There resulted very severe overcrowding. Thus, in Warsaw, which before the war had some 1,300,000 inhabitants, the population rose to 1,800,000 at the end of 1940, and they were obliged to crowd themselves in that half of the dwellings which had been spared by the shelling.[2] Shortage of soap, fuel and food completed the series of conditions favouring typhus.

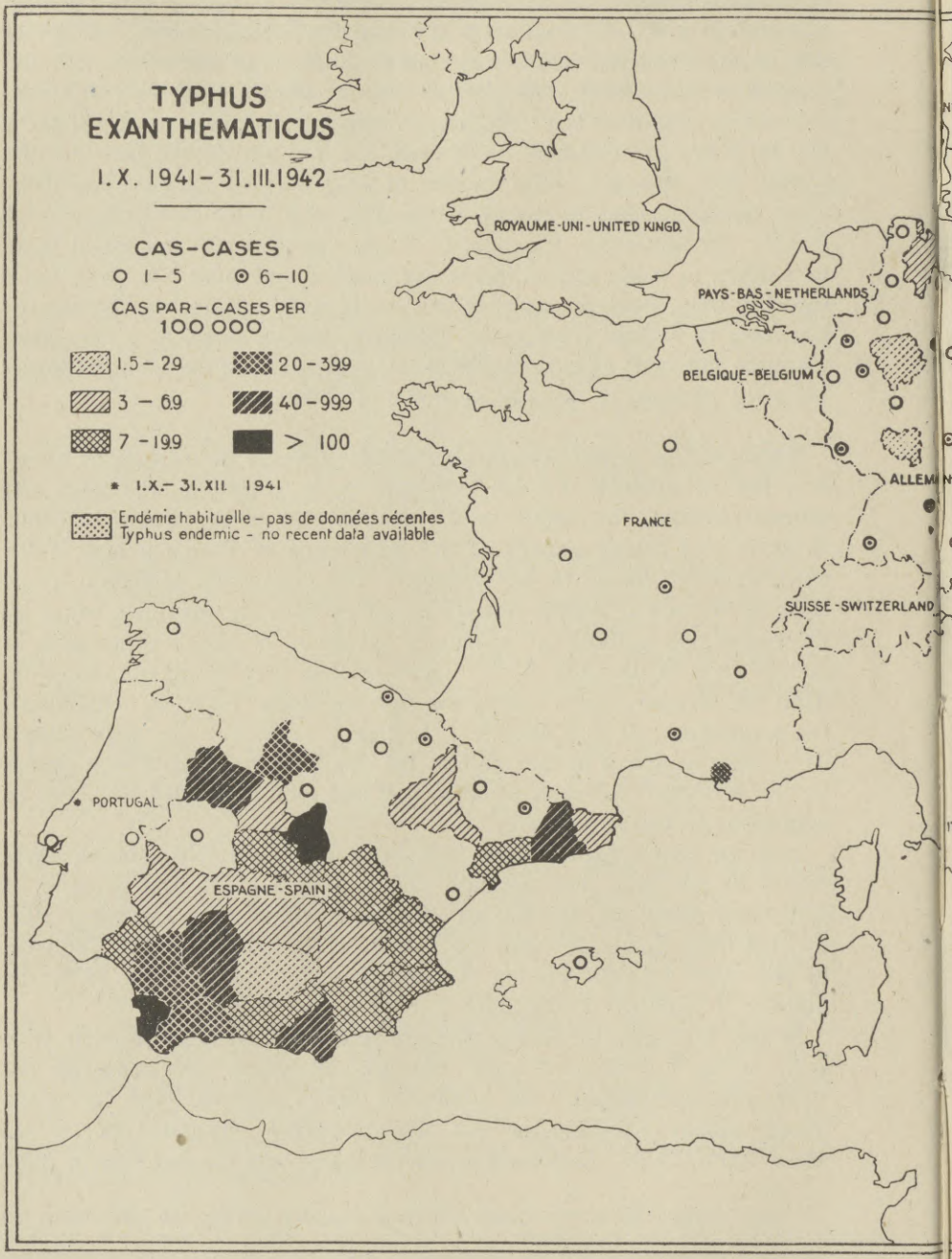
It is not surprising, therefore, that, in spite of extremely rigorous segregation of the sick, their contacts and suspected cases, and in spite of systematic delousing, the number of cases reported from December 1939 to June 1940 reached 3,976 in the district of Warsaw, of which 1,746 were in the town proper. During the corresponding half-year of 1939, the number of cases had not exceeded 33 (with a single death).

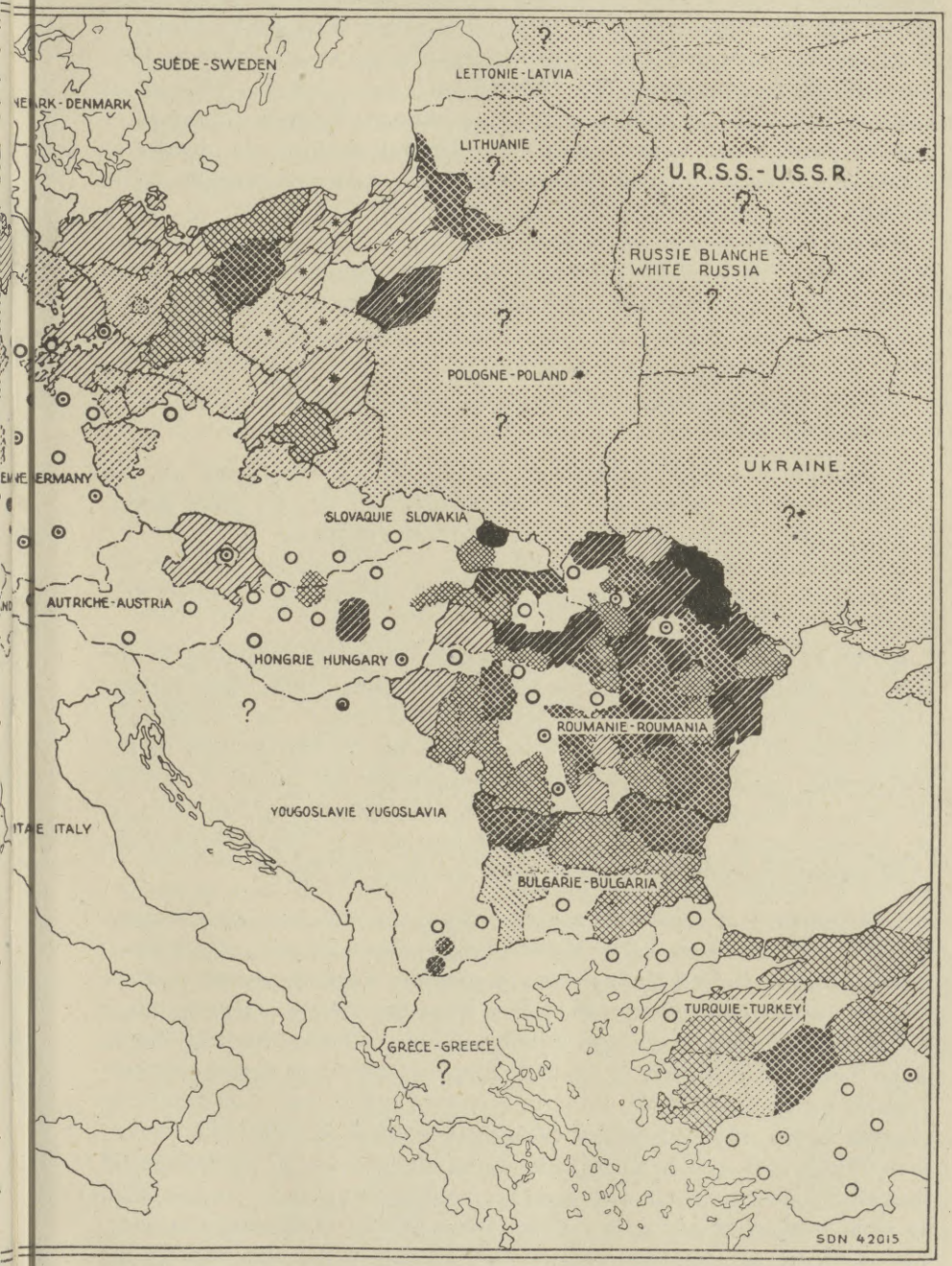
In the Voivodies of Kielce, Krakow and Lublin which, together with parts of the Voivodies of Lodz, Warsaw and Lwow, now constitute the "Generalgouvernement," the number of typhus cases recorded during the fifteen months preceding the war ranged between 300 and 1,000; it was about 700 in 1938. According to Zimmermann,[3] the number rose to 7,900

[1] It was owing to faulty translations of news agency telegrams that the press announced in October 1939 that an epidemic of several thousand cases of "typhus" was raging at Warsaw. This epidemic was not exanthematic typhus but typhoid (*Typhus abdominalis*), due to the destruction of the water mains during the shelling of the town.

[2] According to Kaminski, *D. med. Wschr.*, 14.XI.41, p. 1276, out of 19,025, 2,200 were completely destroyed, 7,800 severely damaged, of which 40% were entirely beyond repair; flats with four rooms sheltered 60 people; a street with 25 houses, 25,000.

[3] Zimmermann, E.: "Zur Epidemologie des Fleckfiebers im Generalgouvernement." *Z. f. Hyg.*, **123**, 552–7, 16.III.1942.


TYPHUS
EXANTHEMATICUS
I. X. 1941 – 31. III. 1942
CAS-CASES
1 – 5
6 – 10
CAS PAR – CASES PER
100 000
1.5 – 2.9
2.0 – 3.99
3 – 6.9
4.0 – 9.99
7 – 19.9
> 100
* I.X.– 31. XII. 1941
Endémie habituelle – pas de données récentes
Typhus endemic – no recent data available
ROYAUME-UNI - UNITED KINGD.
PAYS-BAS - NETHERLANDS
BELGIQUE - BELGIUM
FRANCE
SUISSE - SWITZERLAND
PORTUGAL
ESPAGNE - SPAIN


SUÈDE - SWEDEN
LETTONIE - LATVIA
LITHUANIE
U.R.S.S. - U.S.S.R.
RUSSIE BLANCHE
WHITE RUSSIA
POLOGNE - POLAND
UKRAINE
SLOVAQUIE SLOVAKIA
AUTRICHE - AUSTRIA
HONGRIE HUNGARY
ROUMANIE - ROUMANIA
YOUGOSLAVIE YUGOSLAVIA
BULGARIE - BULGARIA
TURQUIE - TURKEY
GRÈCE - GREECE
SDN 42015

in 1940 and, again according to the same authority, this figure should be doubled or trebled to take account of the light cases which escaped notification. In the western part of Poland, now called Wartheland, where, as stated above, typhus was, if not unknown, at least very rare, there were some 40 cases reported in 1938. This figure rose to 486 in 1940 and to 1,241 in 1941. In the nothern part of the Warsaw Voivodie—annexed to Eastern Prussia with a part of that of Bielystok to form the district of Zichenau (Ciechanow)—some dozens of cases were recorded annually before the war. There were eight in 1940 and 550 in 1941.

For the rest of Poland, we have no official figures for 1941 or the beginning of 1942, but available unofficial information showed that the upward trend of the disease has been maintained. It must be acknowledged that, although the rise observed is disquieting, the figures known still fall far below those showing the situation at the end of the first World War. Thus, in the area now corresponding to the Generalgouvernement, 44,000 cases were reported in 1919.

3. *Increase of typhus prevalence in the areas of Central Europe and the Balkans where the disease was endemosporadic.* In normal years typhus occurs in sporadic form in Bulgaria, Greece and Yugoslavia.

In Roumania, the disease is usually rare or even absent in Transylvania; it is endemic in Bessarabia and Bukovina, and sporadic in the other provinces.

In the Balkans, typhus has been increasing since the beginning of the war, the seasonal peak in the spring of 1942 being higher than in past years, at least in those areas for which figures are available. In Transylvania, both in the part occupied by the Roumanians and in that occupied by the Hungarians, typhus not only made its appearance but spread extensively in 1941. In Sub-Carpathian Ruthenia, a territory which formerly belonged to Czechoslovakia and is now under Hungarian rule, where typhus used to be endemo-sporadic, its prevalence has increased markedly.

4. *Appearance of secondary sporadic typhus in Hungary and Germany.* In 1941, and even more early in 1942, typhus appeared not only in Transylvania and in Sub-Carpathia, but, in sporadic form, in several parts of *Hungary* which had hitherto been free from the disease. This occurred also in *Germany* during the later months of 1941. Outside those territories taken from Poland where, as we have seen, the spread of typhus was particularly marked in 1940–41, no civilian case of typhus was recorded in the German Reich in 1939; only 6 cases were notified in 1940, but no less than 395 in 1941, nearly all of them during the months of November and December. During those two months, typhus occurred in no less than 44 administrative divisions out of the 74 comprised in Greater Germany. The incidence of cases was considerably greater in the east than in the central, western and southern parts of the country. During the first two quarters of 1942, when 459 and 1,273 cases respectively were reported, the distribution was more uniform, foci occurring even in the north-western part of the country.

The cases reported related exclusively to civilians. In view of the traditional cleanliness of the German civilian population, the widespread and rapid distribution of typhus among them can be explained only by the existence of active foci of infection, either within the country among the military population, for which figures are not published (German and foreign troops and, in particular, Russian prisoners), or outside in Poland or in the zone of operations in the U.S.S.R.

During active military operations involving the movements and relief of many divisions and the transportation of hundreds of thousands of wounded, prisoners and civilian workers, it must obviously be difficult to prevent the entry into Germany (or *mutatis mutandis* into Hungary) of carriers of lice and even carriers of the exanthematic virus itself coming from the zone of operations where typhus is endemic.

5. *Typhus endemicity in the U.S.S.R.* The map on pages 8–9 shows that typhus is endemic not only in Eastern Poland and Bessarabia, the bases from which operations were directed against the U.S.S.R., but also in the territories of the Soviet Union itself. Figures for years subsequent to 1937 are unfortunately lacking for that country. Those available for the previous fifty years, however, leave us in no doubt whatever concerning the existence of typhus endemicity there.

Moreover, the mass exodus of civilians during the Russian retreat, their precarious settlement away from their homes, military movements of considerable magnitude, and the hardships of the winter campaign of 1941–42 are powerful factors tending to aggravate the pre-existing typhus endemic. In areas where active operations take place, the systematic or accidental destruction of houses also plays its part, as it entails the crowding together of the remaining civilians and of the invading armies in the few habitable lodgings and houses.

The possibilities of the spread of typhus differ greatly in Russia as regards Germans and Russians. It is, of course, practically impossible to keep either German or Russian troops at the front absolutely free from lice, especially in winter, but whilst the chances of exanthematic virus being brought to the Russian troops are doubtless greater, the German troops are practically all receptive to it, whereas Russian troops are partially resistant, on account of the immunity acquired in past years by a large number of them, as we have shown above.

6. *The Spanish epidemic.* During the first two decades of the twentieth century the number of deaths caused by typhus in Spain could be counted each year by dozens or at all events by hundreds. From 1920 to 1929, the annual number of cases did not exceed a dozen, and from 1930 the annual figure did not reach ten.

The endemo-sporadicity which prevailed early in the century both in Madrid and in Andalusia had gradually changed to sporadicity and was nearing disappearance immediately before the Civil War in 1936. It is owing to this almost complete disappearance of the virus that, in spite of

the louse infestation of the troops in the trenches during the siege of Madrid and also of the civilian population of the capital, typhus did not break out then in the country. No case was reported in the Franco zone which, indeed, did not include the former endemic foci, and this in spite of the bringing over of Moroccan troops.

On the Republican side, it seems that there were false alarms due to relapsing fever, but while there was no true focus or epidemic of typhus, there may have been isolated cases in the provinces of Murcia and Madrid (PALANCA and MARTINEZ FORTUN, 1941).

It was only in April 1939, after the collapse of the Republican front, that typhus made a definite appearance. The first focus appears to have been Cuenca, from which city the infection spread to Madrid. It also manifested itself in the neighbouring provinces, but the majority of early cases originated in the south-eastern provinces. Indeed, it is from these provinces, into which it had been driven or evacuated, that the population of Madrid and other cities in the central part of the country was then flocking homewards. This first outbreak did not exceed 70 cases, and it died out in January 1940. It was only a year later, in January 1941, that the disease made its reappearance, and it did so almost simultaneously in its old endemic haunts of Andalusia and in Madrid. There the epidemic reached the highest incidence. Indeed, it scarcely affected other areas. Of the 7,000 or so cases recorded, no less than 2,100 were reported in Madrid, 960 in Malaga, 800 at Seville, 570 at Granada, the same number at Cadiz, 280 at Murcia, and 260 at Valencia. Thus, Madrid had over a quarter of the total number of cases, whilst the southern large towns aggregated over a half.

It may be wondered if this distribution was due to chance or whether, in former endemic zones, there was not a revival of previously cured infections, as a result of the destitution due to the war and the severe economic disturbances that followed it. The war favoured the development of louse infestation, but destitution increased it still further. The same cause would seem to have brought out the virus that was latent in the old endemic areas, thus bringing together all the conditions necessary for an epidemic.[1]

7. *The hyperendemic wave in North Africa.* Typhus is endemic in all North African countries, but the endemic level varies markedly from year to year, according to the economic and nutritional situation of the population. Thus, war conditions brought about a high incidence of typhus in Egypt from 1914 to 1920, the number of cases reported exceeding 30,000 in 1916. Similarly, poverty caused by the fall in cotton and sugar prices produced a further rise in the endemic level from 1932 to 1935.

In Morocco, Algeria and Tunis, which do not benefit from the regulating influence of the Nile on crops, the level of the endemic depends not so much on the world economic market as on the food production itself, and

[1] In 1942, the epidemic was still going on and invaded Catalonia. During the first quarter, 3,604 cases were reported in the whole of Spain, of which 1,064 were in Madrid and 636 in Barcelona. The epidemic receded, however, in April and May and nearly disappeared in the summer.

this, in turn, depends directly on the rainfall. If the rains fail, and particularly if they fail several years in succession, typhus appears again, concurrently with the impoverishment and hunger of the natives, and the migratory movements of the tribes in quest of food naturally favour the extension of the disease. 1937 and 1938 thus witnessed epidemic outbreaks. In Morocco, mass vaccination campaigns with BLANC's biliated vaccine checked the epidemic.[1] In Algeria and Tunis, on the other hand, it spread increasingly. The figures for 1941 are more than double those recorded in any of the preceding twenty years, and those for the beginning of 1942 exceed them again markedly. The food shortage resulting from abnormal exports and from unbalanced trade, both internal and external, seems to be a major factor in the present typhus situation.

The gradual rise of the typhus curve in French North Africa and the fact that it follows the normal seasonal rhythm and that the disease is not localised but distributed in a comparatively even manner,[2] suggest the existence of a typhus hyperendemic wave rather than of an epidemic in the usual sense.

In the case of communities as in that of individuals, disease often appears to have an inside rather than an outside origin.

The hypothesis, convincingly put forward by Edmond SERGENT and PARROT (1941), of the revival of a cured infection that has been latent, as a result of the rupture of the immunity balance owing to the present state of want, appears very plausible in existing circumstances. The same may be true in regard to some North African victims to the disease in France during the first half of 1942.

8. *Appearance of typhus in sporadic form in France.* In France, which for many years had been quite free from typhus, sporadic cases were reported recently in the central and southern part of the country (Loire, Corrèze, Gard, Bouches-du-Rhône, Allier, Hérault). These cases occurred not only amongst individuals arriving from Algeria, Morocco or Tunis, but also amongst natives from those countries working in France.

Apart from some cases of what one might call direct contagion, i.e. contagion by the parasites of the patients, of which physicians and nurses were the victims, the disease did not spread to the surrounding French population, as most of them were quite free from lice.

The only secondary foci recorded occurred in the prisons of Marseilles, where obviously lice infestation was rife. The number of cases there far exceeded one hundred. Once more, typhus deserved its old name of "gaol fever."

It would be of very great interest to ascertain whether all natives of North Africa who suffered from typhus in France in the beginning of 1942 had been in direct or indirect contact with North African foci of the disease; whether they all carried lice; whether, consequently, in all cases the virus

[1] Cf. GAUD (1938, 1939).

[2] For the details of the evolution and distribution of typhus in North Africa, see *Weekly Epid. Record*, **17**, pp. 86–8, 2.IV.42.

which caused their illness was imported either by themselves or by fellow-workers; or whether these were cases of endogenous revival of typhus infection due to food privations.

The contrast between the absence of typhus among the French population and its comparative frequency among North Africans living under similar conditions is somewhat striking, whether it be explained by their freedom from parasites or by freedom from previous infection.

D. Summary of the present typhus situation in Europe

To sum up, the military campaign in Poland in 1939 and the campaigns in Western Europe in 1940, in the Balkans in the spring of 1941 and in Russia during the summer of the same year, were not accompanied by any outbreak of typhus fever. In Western Europe, this fact is easily explained by the absence both of the typhus virus and of louse infestation among the population and most of the troops. In Eastern Europe, the explanation might be the rarity of the virus during the summer season, at a time when the typhus endemic reaches its lowest level, and also the comparative freedom from lice of troops in rapid but brief operations.

The situation altered later. Destitution following forced migrations and privations caused by foreign occupation produced a marked rise in the typhus endemic level in Poland. This phenomenon occurred during the winter of 1939–40 and became more pronounced during the following two winters.

The prolongation, without respite, of the struggle in the U.S.S.R. during the autumn of 1941 and the following winter was bound to result in louse infestation among the troops from Central Europe and also in their typhus contamination in the endemic area of Poland and the invaded Soviet territories. To the movements of troops across Europe, and of their prisoners both civil and military, must be ascribed the appearance of typhus in sporadic form in countries or parts of countries hitherto free from the disease, such as Germany, Austria, the plains of Hungary, Transylvania, and even certain occupied countries in Western Europe.

The question may be raised whether the marked increase in the usual typhus endemo-sporadicity in Roumania, Bulgaria and Sub-Carpathia is due exclusively to the same cause, i.e. military contamination, or whether as is likely, the effects of food and other restrictions make themselves felt in addition.

The violent epidemic which prevailed in Spain in 1941 and in the beginning of 1942 seems to be the result also of destitution engendered by the Civil War and the severe economic disturbances to which it gave rise.

The appearance of typhus in France is very clearly due to the typhus hyperendemic in North Africa. The latter is connected with the food shortage which by repercussion affects that area. The Spanish epidemic, on the other hand, does not seem to be due to importation of the virus from outside, but to a revival of endemo-sporadic typhus which had almost disappeared in the years preceding 1939.

II. Possible Extension of Typhus and Tasks of Health Authorities

A. The threat of epidemics and the measures called for

The future development of endemic and sporadic typhus in Europe is bound up, on the one hand, with the general course of the war, on the economic and military planes strictly so called, and, on the other hand, with the efficiency of the health services, their energetic action in countries already suffering from typhus and their organising foresight in countries still uninvaded.

A rapid termination of the war would considerably reduce the risks of a pandemic extension of typhus fever; it would not, however, suppress all danger, because it would occasion large troop movements, and it would, moreover, be necessary to ensure that prisoners and demobilised soldiers do not return home carrying infected lice.

Populations of war-devastated regions and reconstruction squads must also be the object of continuous sanitary precautions.

By reducing the means of agricultural production (labour, horses, machinery, etc.), by increasing the weight of military requisitions, by aggravating the unequal distribution of commodities resulting from political decisions and inadequacy of transport, the intensification and prolongation of the war may increase the destitution of populations of regions where typhus has appeared (Central and Eastern Europe in particular) to such a degree that the endemo-sporadicity may degenerate into an endemic, and this endemic into an epidemic.

Such a prolongation may also result in the spread of pediculosis in Western European countries free from typhus, by increasing destitution and thus putting the population at the mercy of an importation of virus.

On the military plane, an increase in the movements of troops, of wounded and of prisoners between zones where typhus is endemic and zones free from typhus, may cause an extension of sporadicity where the population is free from lice, and real epidemics where it is lousy, whether these movements result in fact from an intensification of the war or from its cessation.

The seriousness of the importation of virus into uncontaminated regions will depend on the steps taken by the health services of the armies carrying out troop movements. Experience of past wars shows that, though an army in good order may succeed in protecting itself from typhus, and even in ridding itself of it, the same is not true of an army in disorder, or even merely in retreat, since adequate delousing becomes impossible under such conditions. The efficiency of the civilian health services of the countries traversed or occupied, as well as of those of the belligerent armies, may have considerable bearing on the evolution of typhus in the circumstances outlined above.

We have seen above (page 4) that the three factors conditioning the appearance and development of typhus fever are the presence of the virus,

the presence of its vector, the body-louse, and the existence of a receptive population.

The first of these factors is directly dependent on military operations. It is obvious that strategic considerations will outweigh sanitary considerations when the necessity arises for moving troops from one front to another, even though this means importing lice and typhus from an endemic zone to an uncontaminated one. Under such conditions, sanitary barriers erected by civilian authorities will not be respected and precautionary measures prescribed by military sanitary services will be applied only in so far as they do not lead to too great a loss of time in the movements of troops. Only the sanitary authorities of the rare European countries which have been able to keep their neutrality can contemplate protection against typhus fever through measures designed to prevent the importation of the typhus virus.

For the same reasons as apply in regard to typhus, it is doubtless impossible for most health administrations to prevent the importation of lice. Nevertheless, they can undertake an extremely efficacious prophylactic action either by keeping the population free from lice, where it is uninfested, or by reducing louse infestation as much as possible. If it is possible to keep infestation down to zero, or nearly so, the importations of the virus by infected troops will have only limited effects and cause only sporadic cases. This preventive delousing of the population would have the advantage of training the staff and perfecting the material organisation which, without such a preliminary exercise, might prove to be insufficiently prepared in the event of an immediate menace of epidemic.

Though preventive delousing should be applied first of all to persons most exposed to infestation—vagrants, prisoners, internees, soldiers, etc.—it should be extended to members of the general population, beginning with those whose poverty makes cleanliness of body and clothing difficult (particularly through want of body-linen and spare clothes). It would be advisable to sample different communities and social strata. Infestation (or non-infestation) of persons attending clinics and out-patient departments of hospitals can easily be verified and they may be taken as representative of the mass of the population. Louse infestation is often unsuspected by the host, particularly when he belongs to a class of society in which lice are ordinarily unknown. For example, cases have recently been reported from France of people with irreproachable personal habits who have contracted pediculosis and have consulted doctors for dermatoses. Shortage of soap and fuel, which makes ablutions and the sterilisation of linen more difficult in winter, encourages pediculous infestation to a marked degree.

The continuation of the war and the aggravation of economic restrictions which it entails, not only in regard to soap and fuel, but also in regard to clothes and linen, may obviously favour the spread of pediculosis and render more necessary than ever arrangements for the preventive systematic delousing of the population. If such preventive systematic delousing is desirable in countries free from typhus, in order to limit the diffusibility of the disease, in the event of its being introduced, it is still more necessary

in countries where typhus prevails or has formerly prevailed in endemic or even in sporadic form.

Observations made during recent years have in fact shown that during epidemics, side by side with clear cases of typhus, there have been atypical cases or, more exactly, cases of persons having a symptomless typhus infection.[1]

Infection without symptoms may also be revealed in some cases several months after the characteristic illness.[2] Finally, in a certain proportion of individuals, the illness may make its appearance or reappearance after several years of latency, as the result of excessive fatigue, nutritional deficiencies or other causes of lowered resistance. It is conceivable that proliferation of the virus when the immunity balance is upset may lead to the infection of lice carried by patients and consequently to the contamination of their contacts.

In view of the unfavourable nutritional and economic conditions created by the war or aggravated by it, against which they are relatively powerless, health authorities would be well advised to intensify preventive delousing in zones where typhus has prevailed in order to prevent the revival of endogenous foci.

It is not necessary to recall here the standard methods of delousing and of disinsectisation of clothing, which have been abundantly described by sanitary authorities in official instructions.[3] The following few observations may, however, be made:

(1) The correct application of the technique recommended for each method is more important than the choice of method itself.

(2) In any delousing campaign, unless urgency and lack of materials compel the adoption of a specific method, that which does not damage the objects to be disinsectisised (clothing, shoes, etc.) should always be chosen. (Do not heat more or longer than necessary. Do not use antiseptics with a persistent odour, such as carbolic acid, etc.).

The careful handling of objects to be disinfected is important not only from the economic point of view, in order to avoid the deterioration of commodities difficult to replace, but also from the sanitary point of view itself. Without it, the co-operation of the owners will not be obtained and many articles will be hidden and will escape sanitary measures. Thus, deloused persons will be reinfested almost at once from contact with their infested property.

The necessity for the simultaneous disinsectisation not only of individuals and the clothes they are wearing, but also of their other belongings, bedding, etc., is well known to hygienists in endemic countries.

We have drawn attention to the necessity for a systematic organisation of delousing, including the detection of pediculosis. This should be accom-

[1] Cf. BIRAUD and DEUTSCHMAN (1936), pp. 135–6.

[2] It is mild enough not to infect lice, but it may be revealed by the inoculation of the blood to a guinea-pig.

[3] See references respectively under: Deutsches Reich (1939), England and Wales (1939), France (1939), Suisse (1942).

panied by the organisation of measures for diagnosing typhus cases, which would be particularly helpful in countries where this disease is unknown to the average practitioner, as is the case in most countries of Western Europe.

On the one hand there is the task of *educating* the medical profession, and this should be undertaken by the medical press, official or unofficial,[1] through the publication of articles on the diagnosis and treatment of typhus. On the other hand, it would be well to designate in advance, in every large town or region, doctors with personal experience of typhus fever, acquired abroad, in the colonies, etc., able to act as consulting diagnosticians in doubtful cases. Such an organisation of consultants, whose fees would be paid by the State, has been set up in England (cf. ENGLAND AND WALES).

The experts consulted by the Health Organisation of the League of Nations in 1937 had also recommended, in the event of a threatened epidemic, the "preparation of a plan for the isolation of patients, including their conveyance in vehicles easy to rid of insects, and their treatment in hospitals." This last requires not only the designation of hospital premises, but possibly also their extension.

In regard to hospital organisation for typhus fever patients, mention should be made, among the numerous practical suggestions made by Melville MACKENZIE (1941), of that concerning the employment of as young a staff as possible (less than 30 years of age)—if former cured typhus patients are not available—to care for the patients, the seriousness of the illness being directly proportionate to the age of the patient. The protection of medical and sanitary personnel, and in the first place of those responsible for receiving patients and treating contagious persons in the hospitals, should, moreover, be ensured by *vaccination* before any epidemic breaks out.

As this is merely a precautionary measure to be applied to comparatively few and fixed persons, vaccination by *killed vaccines* in multiple injections is particularly suitable. Systematic revaccination should be practised every six months or so. Vaccination should not be limited to doctors and nurses only, but should be extended to all the auxiliary hospital staff, orderlies, and ward maids, ambulance drivers, laundry workers, bath attendants, disinfectors, and the first-aid personnel who might be called upon should an epidemic break out (St. John Ambulance, etc.). The absence of reactions with killed vaccines facilitates their use when circumstances do not constitute an emergency.

Consideration should be given before any epidemic outbreak not only to the vaccination of medico-sanitary personnel, but also to the constitution of stocks of vaccine which would, if necessary, permit of the vaccination of all or part of the population, should an epidemic break out.

In the following chapter, we review the different vaccines, killed virus-

[1] In the List of References, under the name of each country, will be found the titles of a certain number of publications of this kind issued in Germany (Deutsches Reich), France and Switzerland. Among unofficial publications in French, the excellent monograph of DANIELOPOLU and his collaborators (1940) on the characteristics of war typhus merits special mention. Attention should also be drawn to those of CRAMER (1942), GIRAUD (1941), MOOSER (1942).

vaccines as well as live virus-vaccines, which are of proved efficiency and are now available for use. For each one we shall indicate its respective advantages and disadvantages, as regards the facility with which it is obtained, stored and used, its harmlessness and its preventive efficacy. These different qualities will indicate the circumstances in which the various vaccines may appropriately be used, according to the different situations that may arise—a distant or immediate threat of epidemic, the presence of a limited or extensive epidemic—and to these indications we shall revert briefly in our conclusions.

Even before the appearance of an epidemic, consideration should be given to the organisation of a service for the collection of convalescents' serum in case of need, and to the storing of the necessary apparatus and material. For use at the beginning of an epidemic, the preparation of hyper-immunised animals' serum, that of horses, for instance, should also be considered. Although far from having a curative action comparable, for instance, with that of a diphtheria antitoxin, in strong doses it seems to have a favourable influence on the course of the illness, if applied at an early stage. It acts still better as a prophylactic or semi-prophylactic, i.e. when given to persons who have been in contact with diagnosed patients and are probably in the incubation stage.

We can only touch on this question of anti-typhus serum here, and would refer the reader to articles concerning it, among recent ones those of LEMAIRE (1938), DURAND and BALOZET (1940, 1941), KUROTCHKIN, van der SCHEER and WYCKLOFF (1940), and to the observations of CIUCA and JONESCO-MIHAESTI (1941).

B. Measures to be applied in the event of an epidemic

The 1937 Meeting of Experts outlined the tasks incumbent on health authorities in case of an epidemic—tasks, it should be noted, that will be all the easier to carry out satisfactorily the more thorough the preparation during the pre-epidemic phase. These tasks are as follows:

1. The intensification of measures for discovering louse infestation and for delousing;
2. The isolation of the sick (transfer to hospitals) and of the foci of infection (the establishment of sanitary cordons and the victualling of populations in isolation);
3. The collection and use of convalescents' serum (particularly among non-vaccinated subjects in contact with the sick);[1]
4. The mass vaccination of populations in infected localities.

In 1937 *killed* vaccines against historical typhus fever of proved efficacy and capable of being prepared in quantities were not available, and the

[1] Serum should, nevertheless, be administered to persons whose vaccination with a killed vaccine is not complete or which took place less than a month previously, as they may have been contaminated before immunity was complete.

experts were therefore of opinion that "use might be made of living virus vaccines which can be prepared rapidly in quantities sufficient to meet all requirements." The experts added "that the health administration concerned would be responsible for deciding on the use of these vaccines, the efficacy of which has been proved, but which are still in the experimental stage."

Conditions have changed since this opinion was expressed. It is possible to obtain killed vaccines more easily and promptly than at that time but, on the other hand, live vaccines have been improved and their dangers have greatly decreased. A choice still lies before health authorities, but the elements on which their decision will be based are no longer the same. They are given in the following chapter.

III. Anti-Typhus Vaccines and their Respective Applications

A. General observations on killed and live anti-typhus vaccines and their respective advantages

In spite of their diversity of origin and preparation, available anti-typhus vaccines may be classified in two categories:[1]

(*a*) Killed vaccines;
(*b*) Live virus vaccines.

(*a*) *Killed* vaccines have the merit of being almost completely harmless; in normal doses injections of suspensions of rickettsiae cause local reactions only, generally light, much lighter than those resulting from anti-typhoid vaccine, for instance. As the suspension contains an antiseptic—carbolic acid or formalin—it cannot cause either an inoculation typhus or an infection due to an accidental contamination of the culture. The absence of inoculation typhus *ipso facto* excludes any further transmission of the virus to the parasites (if any) of the vaccinated person, which would have made him a source of contamination to others.

Nevertheless, these vaccines have the following drawbacks, which are inherent in all killed vaccines. They require inoculation of large quantities of the germs, and that at repeated intervals, as these germs do not multiply in the body. Three injections are necessary, at intervals of from seven to ten days, i.e. a total period of from three weeks to a month, for killed vaccines to produce their maximum immunising effect. The time thus required to achieve immunity with a killed vaccine may be a very serious obstacle to prompt intervention in a declared epidemic. Similarly, the necessity to get into touch, at fixed intervals, with the persons to be vaccinated would make the application of this process very difficult in time of crisis, epidemics occurring so often in periods of confusion and of shifting populations.

[1] Among recent general studies on typhus vaccination, we may mention those of Findlay (1941) and of Murgatroyd (1940).

On the other hand, the drawback of multiple injections is negligible when immunisation is carried out well before the appearance of any epidemic, in easily controlled military or hospital communities.

Another drawback of killed vaccines is the relatively short duration of the protection they confer—not longer than a year—unless kept up, either by accidental contamination, probably without symptoms, in an epidemic environment, or by re-vaccination.

If made up from a single species of rickettsia (*Rickettsia prowazeki*), killed vaccines differ little from one another, as they can be brought to the same concentration of antigenic elements.

Their specificity, however, is very exclusive; a killed vaccine prepared with a certain strain affords good protection against that strain but little against others. For that reason, Weigl, who observed this fact in regard to his own vaccine, recommends the use of local strains, if possible several of them, or of strains prevailing in the country from which typhus importation is anticipated. This specificity, this narrowness in immunising power, of the killed antigens is quantitative. Fairly strong immunity against heterologous strain may be obtained by multiplying the number of injections and increasing the doses. From this fact, Mooser draws one of his arguments in favour of the fundamental unity of murine and historical typhuses (Mooser, 1941, Mooser and Leeman, 1941).

(*b*) There are more differences, on the contrary, between *live virus-vaccines*, although they are all at present made up from murine strains (*R. mooseri*), because the attenuation of their virulence and, consequently, their relative harmlessness and efficacy are more complex phenomena than mere antigenic concentration. Each vaccine should therefore form the subject of a special study. Nevertheless, they present certain characteristics in common, specified as follows by the Conference of Experts in 1937:

1. Living virus-vaccines confer *early* protection, whether by premunition[1] only, or by premunition followed by immunity.
2. Living virus-vaccines, provided they actually induce infection, confer *greater* and hence *wider* protection—i.e. protection applying to a greater number of strains or species—than that which can be conferred by the same viruses when killed.

The early establishment of immunity, whether or not due to a state of "precedence" (*préséance*),[2] as certain authors[3] believe, is an important advantage in times of epidemic, an advantage, moreover, combined with that of the single inoculation, which makes live virus vaccination suitable for application to any—even a floating—population.

The *degree* of immunity conferred by injections of live virus-vaccine is seen not only in its polyvalence with regard to multiple strains of rickettsiae

[1] Premunition is the form of immunity depending on the presence of the germ in the body.

[2] "Precedence" (*préséance*) is a condition in which the presence of a live germ prevents infection by further doses of germs of the same species (Blanc, Nowry and Baltazard, 1935).

[3] On the mechanism of immunity in typhus see Ruiz Castañeda (1937).

—a property which allows of the use of murine strains, comparatively mild for man, as vaccinating strains—but also in its long duration, clearly demonstrated by animal experiment and human cases.

In typhus, as in other diseases where live virus-vaccines are available, the immunity obtained is more lasting than that produced by a killed antigen. If the lasting and strong character of the immunity conferred by an attack of typhus is related to the latent survival of the germ (premunition), it is understandable that only live virus-vaccines are capable of inducing a similar immunity.

The possibility of using only one small dose of virus for each inoculation means an enormous economy in vaccines, and consequently the rapid acquisition of the quantities of vaccine necessary to protect large populations, e.g. the entire populations of whole countries. In this respect the Moroccan experiment is conclusive.

Because of their numerous theoretical and practical advantages, live virus-vaccines would undoubtedly have been adopted on a large and exclusive scale were it not for the fact that, on the other hand, they present certain risks.

It is, no doubt, this theoretical risk that the inoculated virus-vaccine circulating in the blood of the vaccinated person might infect possible parasites and thus make of them sources of contagion, that has prejudiced people's minds most against the use of live vaccines. In countries free from typhus, the introduction of the *live* typhus virus used in the vaccines has been feared, whilst in countries where historical typhus prevailed the *murine* character of this virus has been feared.

It must, however, be recognised that numerous attempts, particularly those by Blanc, to infect lice by feeding them on subjects inoculated with live murine virus have all failed, even where the vaccinated subjects contracted a murine inoculation typhus. The same negative results were obtained with fleas.

The *disappearance* of typhus—which regularly follows the mass use of live vaccines in lousy populations, even where the scarcity of food is making itself felt—affords a striking confirmation of these laboratory experiments (BLANC, GAUD, BONJEAN, LAIGRET, etc.). These facts show that there is no practical foundation for the objection raised.

In our opinion, the drawbacks of live vaccines, at least of those first used, lie elsewhere. Although, in general, harmless and effective, live virus-vaccines have sometimes revealed irregularities of action, ranging from insufficient attenuation—with the appearance of an appreciable proportion of murine vaccinal typhus in inoculated subjects—to excessive attenuation, as a result of which the vaccine loses all prophylactic efficacy. Besides a quantity of observations on collective vaccinations which proved to be remarkably effective and without casualties in those vaccinated, some accounts have been published of vaccination setbacks and mishaps, the latter occurring chiefly among Europeans, who are particularly susceptible to typhus virus.

These facts have caused live vaccines to be regarded with suspicion, but

they have also led to further research, which has resulted in the elaboration of techniques that permit of the titration of the activity of live viruses, and thus the prevention of the irregularities of action formerly noted.

B. Detailed study of killed vaccines

The general setback[1] attending attempts at prolonged cultivation of typhus viruses on the usual amorphous media, and even on those which do not contain living cells, has led to research with a view to obtaining appreciable quantities of these viruses either from the tissue of animals normally infected or from the organs of animals inoculated for this purpose.

1. *Vaccines prepared with rickettsiae from the louse; Weigl's vaccine*

During the first world war, DA ROCHA LIMA tried the immunising effect of a suspension of infected body lice in guinea-pigs, but the results were irregular, doubtless owing to differences in the abundance of the virus in the different lice used.

WEIGL conceived the idea of remedying this defect by inoculating lice artificially by intrarectal injections of a suspension of rickettsiae by means of a fine pipette.[2] The rickettsiae invade the intestinal mucosa and proliferate there. After some days the rickettsiae-crammed cells burst and the intestinal lumen contains a pure culture. Their number reaches from 10 to 100 million in the intestine of an adult female; less in that of a male; with an average of some 50 million germs per intestine at the stage of maximum development, i.e. about 8 days after the inoculation. The intestine is detached from the louse by sterile microdissection; it is ground and emulsified in saline solution containing 0·5% of carbolic acid.[3, 4]

Use and dosage

When first applying his process, Weigl gave the following doses in three injections at intervals of from 3 to 5 days:

1st injection: 0·25 c.c. = 1,250 millions of germs = 25 lice
2nd injection: 0·50 c.c. = 2,500 millions of germs = 50 lice
3rd injection: 1 c.c. = 5,000 millions of germs = 100 lice

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[1] It must be recalled, however, that ANIGSTEIN and LAWCOWICZ (1939) had succeeded in cultivating the murine strain "Tunis I" and the historical strain "Wet" on Noguchi's semi-solid medium, which they modify by replacing the saline solution it contained by tap water. The haemoglobin of the blood corpuscles of the rabbit were thus laked and diffused in the medium. The formula of the medium was the following:

Plain sterile water	80 c.c.
Defibrinated rabbit blood	3 c.c.
Fresh rabbit serum	10 c.c.
Agar 12% (pH 7·4)	10 to 12 c.c.

[2] The lice inoculated are fed morning and evening for eight days on immune individuals.

[3] A good description in French of Weigl's technique has been published by NICOLLE and SPARROW (1932).

[4] Recently NAUCK and WEYER (1941) have shown that one could not obtain any appreciable reproduction of rickettsiae in the tissues of lice *in vitro*.

i.e. a total of 175 louse intestinal contents (WEIGL, 1930[b]). Weigl afterwards reduced the dose to 90 lice for a normal vaccination. He experimented with smaller doses on 23,000 persons in an endemic area in Poland, but found that at least 1 to 6 louse intestines were necessary to achieve the slightest degree of immunity, and that, as the duration of the immunity depends on the dose, protection was only transient.

These attempts to reduce the antigenic dose obviously had their cause in the small output of the production laboratory, owing to the delicacy of the necessary manipulations, which only trained technicians can carry out (anal injection of the louse and dissection of its intestine). In 1937 Professor Weigl's Lwow laboratory succeeded in producing only 2,000 "normal" doses (of 90 lice each) a month, at a cost of about 3 gold francs a dose.

Reactions to the inoculation

The reactions to inoculation with Weigl's vaccine are mild and, according to Weigl, depend on the doses used. He claims that hyperthermia regularly occurs only with doses exceeding 200 louse intestines, doses which exceed considerably those now used.

In the cases observed by MARIANI (1939), who, moreover, used weaker doses than Weigl (10 and 15 and 25 lice), local and general reactions (hyperthermia exceeding 1° C.) were noticeable only in 5% of the vaccinated persons. On the whole, therefore, these reactions are negligible and, as this virus is carbolic-killed, its innocuousness can be considered as complete.

Applications of Weigl's vaccine

Naturally, it is in Poland that Weigl's vaccine has been most widely employed, chiefly among medical and sanitary personnel, and also among contacts of the patients. The results recorded by CHODZKO (1933), WEIGL himself (1933), and his pupil HERZIG (1939) have been distinctly favourable.

According to MOSING (1938), 120,000 persons had been vaccinated in Poland since 1929, and amongst them only a few dozen cases had been recorded, a proportion of only 0·09% according to PIETRASZEWSKI.[1] According to ANIGSTEIN (L.o.N. Document CH1236[a]), among 10,000 members of the medico-sanitary professions vaccinated, 15 cases of typhus were recorded at the beginning of 1937, some during the first days following the vaccination—this having been done during the incubation—the others during the 11th or 12th month following, which would indicate that the duration of effective immunity may be taken as about a year.

RADLO (1937) has recorded that the vaccination of 13,980 inhabitants of the endemic zones of the Jaworow district near Lwow, where typhus was endemic and accounted for an average of 200 cases each year, resulted in the number of cases falling to 12. Of these 12, 4 had been vaccinated from two months to three years previously. The limited duration of immunity observed agrees with the results of the serological studies made by LIU,

[1] Cited by MOSING (1938).

ZIA and WANG (1938), on vaccinated persons in North China. These authors found, in fact, that if the anti-rickettsiae agglutinins appear early after Weigl vaccinations, they disappear quickly, more quickly than the agglutinins against the *Proteus* OX_{19}. A more limited but, by reason of the degree of intensity of the risk of contamination to which the vaccinated persons were exposed, more conclusive experiment was afforded by the vaccination of 200 Belgian missionaries of Sheut in North China. Four or five of these missionaries died of typhus every year and that disease alone caused 83% of their mortality. Its inroads among these missionaries were stopped outright (no death in five years) while they continued unchecked among the other missionary orders in the region, who were not vaccinated (TCHANG and LOTSONG, 1934) (RUTTEN 1936).

This success was sufficiently conclusive to result in Weigl's method being used in a university laboratory in Peiping (LIU, ZIA and WANG, 1938). However, the easier technique of culture on chicken embryo led to the abandoning of Weigl's method in this centre for that of Cox (TCHANG and MATHEWS, 1940) (PANG and ZIA, 1940).

In Czechoslovakia, Weigl's vaccine has been prepared in small quantities by the Prague Institute of Hygiene and used among doctors, nurses and contacts of the sick in Sub-Carpathian Ruthenia.

In Egypt, no evidence was furnished by the vaccination of half the inhabitants of Esbel village, as the illness did not appear among the other half either (SHAHIN PACHA, 1935).

In Ethiopia, in 1938, MARIANI (1939) vaccinated 13,076 persons, soldiers, hospital staffs and officials particularly exposed to the disease, using Weigl's method. He has recorded only 8 cases among the vaccinated, of which 5 occurred before the third injection and most of the others during the days immediately following the third injection; only one vaccinated person died. MARIANI (1938) had to abandon the breeding of lice because of epizoötics caused among these lice by rickettsiae other than *R. prowazeki.*

In Germany, the army medical services have been vaccinated with a vaccine prepared at Cracow by EYER (1941), using Weigl's method, and he has declared himself very satisfied with it. EYER, PRZYBYLKIEWICZ and DILLENBERG (1940) observed 15 cases of laboratory typhus in persons, themselves vaccinated, working on the preparation of Weigl vaccine with infected lice. Weigl had formerly called attention to other cases, including his own. But it is obvious that, in these laboratory infections, contamination takes place with doses considerably higher than those to which one is ordinarily exposed, and that these laboratory typhus cases do not justify the condemnation of the vaccine as ineffective.

Weigl's vaccine affords poor protection against strains of virus other than that with which it has been prepared. Recognising this, Weigl recommends that local and multiple strains should be used in its preparation. This recommendation should also be applied to the preparation of all killed vaccines, as they share with Weigl's this characteristic narrowness in their specificity.

Conclusions

Weigl's vaccine has abundantly proved its harmlessness and efficacy. Its essential drawback, unfortunately a serious one, is the delicacy of manipulation required for its preparation, which limits the amount that can be produced, even by expert hands, and consequently limits its application to the minority of persons particularly exposed to the disease, such as the sanitary personnel and persons in contact with the sick. Among these contacts, if the vaccine is applied soon after contamination, it seems to lessen the seriousness of the infection (MOSING, 1938). Because of its intrinsic worth, a certain number of Institutes with specialised assistants are continuing to prepare Weigl's vaccine; others have abandoned it in favour of vaccines that are easier to prepare in quantities. Most Institutes that have recently begun to produce anti-typhus vaccines have also chosen easier and more productive methods of preparation.

With Weigl's vaccine may be associated also that experimented with by his pupils CHRZANOWSKI and MOSING (1933)—a carbolised suspension of dejections of lice artificially inoculated by Weigl's method. We have not seen any accounts of extensive human vaccinations by this process, but, nevertheless, it appears *a priori* to be satisfactory.

2. *Killed vaccines from murine virus* (*Mooser*, *Zinsser*, *Castañeda*)

In a study dealing chiefly with typhus in Europe and, consequently, with the prophylaxis of historical typhus, it is not possible to describe in detail the numerous examples of work undertaken in the New World with a view to obtaining an effective vaccine against murine typhus, and the typhuses resembling it, prevailing in Central and North America. We shall mention them briefly, however, as a tribute to the fertility of invention and the patience of the authors of the various methods evolved.

We would recall the vaccines prepared by MOOSER, by ZINSSER and by RUIZ CASTAÑEDA with formol-killed rickettsiae of the Mexican murine virus, rickettsiae obtained from the tunica of infected guinea-pigs (1931), from the peritoneal exudate of guinea-pigs submitted to a vitamin-deficient diet (1931), or from the peritoneum of rats subjected to benzol poisoning, to intense exposure to X-rays (VARELA, PARADA GAY and RAMOS, 1932), or to repeated injections of blood (VARELA and PARADA GAY, 1934), all processes aiming at lowering the resistance of the inoculated animals and consequently increasing the abundance of the rickettsiae to be obtained from them. These vaccines appear to have given good results in arresting epidemics of tabardillo in Mexico (in the hands of RUIZ CASTAÑEDA) and in Bolivia (in those of VEINTEMILLAS), etc.

Yet murine viruses cultivated on the rat in this way have shown themselves incapable of protecting effectively against historical virus[1]—an instance of the exclusive specificity of killed vaccines—and, moreover, in spite of the

[1] FINLAYSON and GROBLER (1940, 1941), for instance, insist on the necessity for strong and repeated doses of the Zinsser–Castañeda murine vaccine in order to protect the guinea-pig—and that only partially—against the historical strains of North Africa.

numerous trials made by ZINSSER and his pupils, it has not been found possible to cultivate the historical virus on the rat.[1] For this reason, and also because he found mass production of vaccine to be impossible by these methods, ZINSSER turned to the production of rickettsiae on tissue cultures.

3. *Killed vaccines from tissue cultures in vitro*

As early as 1930, NIGG and LANDSTEINER grew rickettsiae of the European type on tissue cultures using a modified MAITLAND–RIVERS technique, and found that virulence could be maintained through a series of sub-cultures (1932) and for months at a temperature of 37° C. or at — 20° C. (NIGG, 1935, 1936).

KLIGLER and ASCHNER (1933, 1934), on the other hand, using the tunica of a normal guinea-pig macerated in Tyrode solution, obtained a culture of *R. prowazeki* in from 8 to 30 days abundant enough for use, formalinised as a vaccine which proved effective in the guinea-pig.

With MACCHIAVELLO (1936), and afterwards with other collaborators, ZINSSER devoted himself to perfecting these techniques in order to increase production. He succeeded in this by abandoning the big Maitland flasks for Kolle flasks containing a special agar medium, on the surface of which he increased the quantity of tissue (guinea-pig tunica, mouse embryo), keeping the culture in a nearly anaerobic condition, allowing the access of air only through a glass tube with a fine point, thus reducing cell metabolism to the minimum (ZINSSER, WEI and FITZPATRICK, 1937, 1938); (ZINSSER, FITZPATRICK and WEI, 1939). We cannot mention here all the modifications applied to those of MAITLAND and MAITLAND by the ingenuity of authors.[2] We would indicate that of HITZ (1938), however, which replaces the Tyrode solution by ascites fluid.

As Zinsser's technique has undergone frequent modification, we think it would be useful to give below the description this worker and his collaborators FITZPATRICK and WEI gave in the *Journal of Experimental Medicine* of February 1939. The success of the technique depends in a large measure on the quality of the nutritive medium. This is an isotonic agar, partly buffered, the pH of which is adjusted by the addition of Tyrode solution and horse or ox serum. When the living fragments of tissue are placed on this agar the diffusion in the medium affords the necessary conditions for the removal of metabolic products and for the supplying of the material necessary for the life of the cells.

The quality of the agar being one of the most important factors for the success of the method, its preparation is given in detail:

1. A 3% agar solution is made in distilled water. The agar used is the Difco granular variety. This agar solution is dissolved at 15 pounds for 15 minutes in the autoclave, then cooled and kept at 45° C. in a water-bath.

[1] LIU, SNYDER and ENDERS (1941), however, have recently succeeded in killing irradiated white mice by intraperitoneal inoculation of historical virus.

[2] For the growth of historical virus on tissue cultures see JAZIMIRSKA-KRONTOWSKA, SOLITERMAN and SCHWEDKOWA-ROCHE (1937).

2. A double strength Tyrode solution is made corresponding in every way to the original formula except that 2 g. of sodium bicarbonate are used per litre. To 300 c.c. of this double-strength Tyrode are added 200 c.c. of the appropriate serum—in rickettsiae work, horse serum—and 16 c.c. of 0·04 solution of phenol red. This Tyrode-serum mixture is filtered through a Seitz filter. Zinsser finds it important to wash the Seitz filter discs rapidly with distilled water at least three times in order to diminish alkalinity. These washed discs are dried slowly in the incubator and then sterilised, in the holders, in the autoclave.

The Tyrode-serum mixture, as given above, after filtration, is warmed to 45° C. and mixed at this temperature with 300 c.c. of the 3% agar. The mixture is then poured into 6 × 1 inch test tubes, with precautions for sterility (use of a sterile siphon system) in 12 c.c. amounts. The tubes are slanted and left at room temperature to harden. Then they are stoppered with sterile No. 4 stoppers. The final reaction should be about 7·8. The slants should be kept upright at room temperature for two or three days so that the water of condensation may sink to the bottom to be removed with a capillary pipette before the tubes are planted, since excessive moisture interferes with growth. They are kept at room temperature for an additional check on possible contamination and, after this, can be kept in the ice chest until used.

The tissue at first used for rickettsia cultivation was guinea-pig tunica of the testicle.

The tunica is removed from animals infected with passage virus on the sixth or seventh day after intraperitoneal infection. It is removed under sterile precautions, is finely minced in a test tube with a few drops of Tyrode-serum mixture, and gently laid on the surface of an agar slant as described above so that about one-third of the surface is covered. Culture takes place at 37° C.

Subsequent transfers are made at intervals of anywhere from the sixth to the tenth days by removing the tissue from a previously inoculated tube, mixing it with a fresh normal tunica, mincing them together, and allowing them to stand for 10 minutes before inoculating other slants. It is wise, at first, not to attempt to make more than three new slants from a single old one. Later, as growth picks up, five or six transplants can be made from a single seed tube.

In the course of these researches Zinsser found he could replace the tunica tissue by young mouse embryo tissue, and finally by chick embryo.[1] Ultimately he succeeded in inoculating these tubes of agar-tissue with virus taken from an egg[2] in order to sustain and even augment the virulence of the rickettsiae which repeated subculturing on agar-tissue, on the contrary, weakened (ZINSSER, PLOTZ and ENDERS, 1940). Zinsser did not claim that the vaccine thus obtained was superior in quality to Weigl's, or to that obtained by Maitland's original method. He considered his technique of agar-tissue culture offered the sole advantage of quantity. Yet, although Zinsser described it as being "mass production," relatively speaking, it was still very moderate, since Zinsser considered that a staff of one bacteriologist and two technicians could obtain weekly about a litre of rickettsiae emulsion, which would permit the vaccination of 300 people.

Moreover, in February 1939 he added, very rightly, that only field application on a large scale would enable the value of his vaccine to be judged.

As far as we are aware, this large-scale application has not been made.

[1] Ordinary ten-day chick embryo.
[2] Allantoid sac of hen eggs inoculated four days previously.

and these culture vaccines have not as yet undergone the crucial test of practice.

4. *Killed vaccines from rickettsiae cultivated on chick-embryo—Cox's vaccine*

Already in 1934 ZIA successfully inoculated the chorio-allantoid membrane of fertile hen eggs with fragments of the tunica of guinea-pigs infected with Mexican or European virus (1934), but he was not able to obtain a quantity of rickettsiae sufficient for practical use.

BENGSTON and DYER (1935) used the same medium to grow rickettsiae of Rocky Mountain spotted fever.

In 1938, Herald R. COX was the first to succeed in producing any considerable quantities of rickettsiae by inoculating the egg directly in the yolk.

Although, at first, the rickettsiae of historical typhus (Breinl strain)—unlike those of endemic typhus (Wilmington strain)—were scarcely visible in the yolk, and only revealed themselves by the virulence of the vitelline sac on inoculation of the guinea-pig (COX, 1938), this difference in behaviour disappeared in subcultures and *R. prowazeki* showed up in as large numbers as by inoculations of *R. mooseri* (Cox, 1939).

Thereafter, Cox devoted himself to determining which part of the egg contained the highest numbers of rickettsiae in proportion to its protein bulk, in order to facilitate the purification of the vaccine by eliminating most of these proteins. He therefore prepared vaccines with

(1) The vitelline sac alone (''*ep. ty.* 26 and 27'');

(2) The vitelline sac plus the chorio-allantoid membrane (''*ep. ty.* 20'');

(3) The two membranes, plus the embryo itself, i.e. the total tissues of the egg (''*ep. ty.* 6–1, 6–2, 6–3'').

Several methods were tried by COX and BELL (1940) and, though the vaccines they yield differ as regards quantity, they are practically identical from the point of view of their experimental prophylactic efficacy. Cox and his collaborator describe the methods as follows:

> Before inoculation, fertile eggs were incubated 6 to 7 days at 39° C. The inoculum, 0·5 to 1·0 c.c. of a 10% yolk-sac suspension in Tyrode's ascitic fluid, was injected into the yolk by means of a 1½ inch, 21-gauge needle introduced through the air-sac end of the egg. The eggs were then placed in a 37° C. incubator until death of the embryo, which usually occurred in 5 to 7 days. In every instance the tissues were used for vaccine preparation within 12 hours after death of the embryo.
>
> For the preparation of vaccines, the embryonic tissue or tissues used were completely removed aseptically from a number of eggs of the same transfer and washed once or twice with sterile saline[1] to remove any yolk or other fluids that might be present. They were then drained free of excess moisture, pooled, weighed, and ground with sterile alundum to a homogeneous mixture. Sterile saline was added to make a 10% suspension. A portion was reserved for titration and to

[1] All saline used for re-suspending the various vaccine fractions, as well as that used in the final product, contained phenol at 0·4% and formalin at 0·1% concentration.

the remainder was added phenol to 1·0%, and formalin to 0·5% concentration. The suspension was then vigorously shaken on a shaking-machine for 1 hour and stored at 2° C. (6 to 76 days) before being refined for use.

Vaccines *ep. ty.* 26 and 27 were similarly prepared from yolk-sac alone by the following method:

The 10% suspension was allowed to stand at 2° C. for 6 days and was then centrifuged (a 51° angle centrifuge was used in all experiments) at 4,500 to 5,000 r.p.m. for 45 minutes to an hour. The precipitate was reground with alundum, resuspended in approximately the same volume of saline[1], and again centrifuged as above. This precipitate was also resuspended, this time in one-half the original volume of saline, and then centrifuged at 1,000 r.p.m. for 10 minutes. The supernatant fluid thus obtained constituted the vaccine.

Practically all the lipoids along with some protein were eliminated in the first two supernatant fluids, while the great bulk of cellular debris was thrown down in the final precipitate. The final supernatant fluid, which constituted the vaccine, contained rickettsiae in profusion with relatively little detritus. Further clearing may be obtained by fractional centrifugation.

Vaccine *ep. ty.* 20 was similarly prepared except that it was made from yolk-sac and chorio-allantois and the crude suspension was kept at 2° C. for 25 days before being refined.

Vaccines *ep. ty.* 6–1, 6–2, and 6–3 were prepared somewhat differently. The crude suspension (yolk-sac, chorio-allantois, and embryo) was kept at 2° C. for 76 days and then treated as follows:

(*a*) A portion was diluted with saline (containing no phenol or formalin) to make a final tissue concentration of 2%. The suspension was centrifuged at 1,000 r.p.m. for 10 minutes and the supernatant fluid thus obtained constituted vaccine *ep. ty.* 6-1.

(*b*) The remaining portion of the crude suspension was diluted with saline (containing no phenol or formalin) to make a final tissue concentration of 4%. This suspension was centrifuged at 5,000 r.p.m. for 1 hour and the supernatant fluid thus obtained used as vaccine *ep. ty.* 6–2.

(*c*) The resulting precipitate was resuspended in one-fifth of the original volume of saline (containing 0·4% phenol and 0·1% formalin), centrifuged at 1,000 r.p.m. for 10 minutes, and this supernatant fluid was used as vaccine *ep. ty.* 6-3.

Vaccines prepared by these different methods all conferred a certain amount of protection to *all* the guinea-pigs used experimentally (106/106), and *completely* protected, against a very active virus, 82 of 106 of them. The results of the test and the quantities of vaccine obtained with the different methods are set out in the table below.

It will be seen that, in spite of the unequal yield of the different methods used, as Cox says, each of them can be used for the practical production of vaccine.[2] This fact, in conjunction with the prophylactic efficacy revealed by control tests, not only in guinea-pigs but also in monkeys, contributed to the extension of the use of Cox's method.

[1] Phenolised at 0·4% and formalinised at 0·1% concentration.

[2] According to Cox (1941), 226 litres, i.e. the dose for 75,000 persons, were used in 1940.

Type of Vaccine	Tissues used	Protection of the Guinea Pig obtained		Yield of Vaccine in c.c.	
		Certain	Total		Per Egg
I. *Ep. ty.* 26 and 27	Yolk sac alone	53/53	34/53	85–100 c.c. for 14 eggs	6–7 c.c.
II. *Ep. ty.* 20	Yolk sac and chorio-allantois	14/14	13/14	200 c.c. for 14 eggs	15 c.c.
III.	All embryonic tissues	39/39	35/39		
Ep. ty. 6–1	„ „ concentration of 2% centrifugation at 1,000 r.p.m. × 10 minutes	14/14	12/14	550 c.c. for 2 eggs	275 c.c.
Ep. ty. 6–2	„ „ concentration of 4% centrifugation at 5,000 r.p.m. × 60 minutes	12/12	12/12	275 c.c. for 2 eggs	137 c.c.
Ep. ty. 6–3	„ „ precipitate resuspended and centrifuged at 1,000 r.p.m. × 10 minutes	13/13	11/13	50–60 c.c. for 2 eggs	25–30 c.c.

Application of Cox's method

Although trials of Cox's method against historical typhus on man in Europe have only been effected on a limited number of subjects and under unfavourable conditions owing to present circumstances (Sub-Carpathia in 1939; Hungary and Roumania in 1940; Spain in 1941 (PRIMITIVO de la QUINTANA, 1942); U.S.S.R. in 1941–42, it has been chosen by American laboratories for the manufacture of quantities of vaccine despatched, on the one hand, to Europe, particularly to Great Britain, and set aside, on the other hand, for vaccinating American troops destined for foreign theatres of war where typhus may constitute a risk for them.

The official circular (1942) on the use of this vaccine lays down an initial immunisation consisting of 3 subcutaneous injections of 1 c.c. each, spaced at 7 to 10 days, maintained by subsequent doses, also of 1 c.c., to be repeated every 4 to 6 months as long as the risk of contamination lasts.

KUROTCHKIN and WYCKOFF (1941), who gained experience of Zinsser's method at the Lederle Laboratories, Pearl River (N.Y.), consider that Cox's vaccine, tried on a guinea-pig against 1,000 infectious doses, is at least as effective as Zinsser's tissue-agar vaccine.

It has been introduced in *Germany* by OTTO (1941, 1942) and WOHLRAB of the Frankfurt a.M. Forschungsinstitut für Chemotherapie, and has also been employed by GILDEMEISTER and HAAGEN (1942), who, whilst considering Cox's technique as being more practical than Weigl's, are of opinion that it can produce only restricted amounts, and accordingly cannot supply the material necessary for mass vaccination.

In *China*, we have seen that TCHANG (CHANG) and MATHEWS (1940),

PANG and ZIA (1940) have put Cox's method into practice, and that it has shown itself perfectly suited to the virus of North China.

This method of culture on chick embryo has also been applied successfully by GEAR (1940)[1] for South African typhus. It is interesting to see that this worker has given up Zinsser's (agar-tissue) method for that of Cox, because there is less risk of culture contamination and virulence is maintained and, above all, because of the handsome crop of rickettsiae which can be obtained by a single bacteriologist, working without complicated apparatus or the necessity for using animals.

In *Australia* BURNET and FREEMAN (1941) have successfully applied chorio-allantois cultivation to the virus of Queensland typhus (Q fever), as also to Dyer's X virus, its North American equivalent (Montana) confirming COX and BELL'S observation (1939). These workers have all noted the increased virulence of *R. diaporica* (or *R. burneti*) in passages on chick embryo.

In the Netherlands Indies, GISPEN (1941) has cultivated the virus of Malaya Scrub typhus, *R. orientalis*, and of the mite fever of Sumatra (Mijtekoorts), not on hen- but on *duck*-egg. He considers that duck-egg presents appreciable advantages over that of the hen:

> The embryos are more viable under laboratory conditions;
> Rickettsiae infection kills them more slowly (10 days instead of 5 to 7);
> They furnish larger areas for cultivation because of their greater size.

On the other hand, the success achieved in the cultivation of murine virus on chick embryo, not only by ZIA but by numerous workers in Central and South America (VEINTEMILLAS, 1938), points to the fact that this method of obtaining all kinds of rickettsiae is widely established in the practice of laboratories and also in human use, in spite of the scanty facts published regarding the results.

5. *Killed vaccines from rickettsiae developed in the lungs of animals inoculated in the respiratory tract*

(*a*) *Mouse-vaccines of Ruiz Castañeda and of Durand–Sparrow*

Once more, in 1939, RUIZ CASTAÑEDA opened up a new line of enquiry by recording the considerable development of rickettsiae in the pulmonary tissues of rodents—rats, mice and rabbits—and even sheep, inoculated in the nasal passages while under ether anaesthesia. He used a Mexican strain of murine type which had undergone 150 passages on rodents, regularly provoking the Neill–Mooser scrotal reaction. To prepare an anti-typhus vaccine, RUIZ CASTAÑEDA (1941) made use of the rickettsiae inoculation pneumonia in the rat. HUDSON (1940) showed the efficacy of this vaccine against Mexican typhus in the guinea-pig.

Working independently, Paul DURAND and Hélène SPARROW (1940) also

[1] GEAR gives an excellent description of the method of culture on chick embryo. Many practical details on the organisation of the laboratory for cultivation on chick embryo will also be found in ANDERSON (1941).

obtained a rickettsiae hepatisation of mouse and rabbit lungs by nasal instillation of a strain of historical typhus, cultivated in the intestine of the louse and not causing orchitis.

DURAND and SPARROW (1940) found that white mice,[1] particularly the so-called Swiss mice and also *Mus musculus gentilis* Brandt, *Mus musculus* Lataste, and the striped rat *Arvicanthus barbarus* L., were particularly receptive to this mode of inoculation and, considering their weight, capable of supplying regularly and rapidly a large quantity of rickettsiae.

The technique applied by these workers was as follows:

> The mice are placed one by one in a glass jar containing a plug of cotton-wool imbibed with ether until they fall motionless. They are then seized by the ears and held nose upwards. Four or 5 drops of virulent material are then dropped into the nose with a very fine pipette. This corresponds to about 0·10 c.c. The virus is, to start with, an emulsion of intestines of lice inoculated *per anum* and dissected on the 7th day. The dilution is made in saline solution containing 1/5th of normal human or horse serum. Each mouse receives a dose of 1 to 3 louse intestines.
>
> Anaesthesia being light, the mice awake very rapidly. Mice inoculated die within 40 to 60 hours. Illness begins 24 hours after inoculation and is characterised by considerable hypothermia. The lungs show haemorrhagic hepatisation. Smears from the lungs, stained with Giemsa's or Castañeda's techniques, show enormous quantities of rickettsiae, most of them extra-cellular owing probably to the bursting of the cells in which they grew, when the mouse dies or is killed in the state of hypothermia 48 hours after the inoculation. In general the lungs yield a pure culture of rickettsiae.

DURAND and GIROUD (1940) give the following details concerning the technique used for preparing vaccine on the lung:

> The lungs, which a rapid examination through Ruiz Castañeda's staining has shown to contain an abundance of rickettsiae, are ground, without further addition, as finely as possible in a special grinder of Pyrex glass, or in a grinder containing stainless steel beads. They are then emulsified in normal human or horse serum, diluted to a proportion of 1 to 5 in normal saline (preferably buffered at a pH of 7·4 with a small quantity of a mixture of phosphates). To this diluted serum is added, immediately before use, 2 c.c. per 1,000 of formalin at 40% previously neutralised.
>
> The lung emulsion is centrifuged for five minutes in order to separate cells or cell debris, which fall to the bottom. They are again ground and the process repeated. After the second centrifugation, the supernatant fluid is gathered and thoroughly centrifuged for 1 hour at a speed of at least 7,000 r.p.m., a rise in temperature during that process being carefully avoided.
>
> The final sediment obtained is greyish-white; the fluid above it, which contains haemoglobin in solution, is thrown away, together with the thin layer of fat which floats at its surface.
>
> A smear stained with cyanoquine or with Castañeda's stain, or examination with the ultra-microscope, must show that 9/10ths at least of the volume of the sediment is made up of rickettsiae. If this proportion is not obtained, purification is repeated by means of fractional centrifugation.
>
> A homogeneous emulsion of the mass of rickettsiae is made in diluted and formalinised serum identical to the one which was used in the first manipulation,

[1] For the technique of breeding white mice for vaccine production see, for instance MATHIS (1942), REMLINGER and BAILLY (1942).

and note is taken of the titre of the dilution with regard to the number of lungs used or, rather, the weight of those lungs.[1] The vaccine is distributed in ampoules and kept in the ice-chest for at least 5 days before use.

DURAND and GIROUD and DURAND and PANTHIER (1942[a] and 1942[c]) obtained a very strong protection against virulent inoculation made subcutaneously in the guinea-pig, in the monkey (baboon) and, finally, in man.

In their first trials, these authors made, at 5-day intervals, 3 injections of formalinised vaccines at doses corresponding to 7/100, 12/100 and 18/100 of mouse-lung. The serum of 11 individuals out of 12 acted as the serum of typhus convalescents, neutralising the virus completely, or almost completely.[2]

In a following series, in which the vaccine doses were reduced by 2/3rds, the neutralising antibodies were still revealed constantly 20 days after vaccination and the Weigl–Félix reaction turned positive in 2 cases out of 3.

Guinea-pigs protected by 3 doses of formalinised vaccine did not react to the inoculation of 1,000 infecting doses (GIROUD and PANTHIER, 1942[c]).

(*b*) *Vaccine obtained from rabbit-lungs by Durand–Giroud.*

In spite of the abundance of rickettsiae found in the lungs of mice inoculated through the nasal route, the slight volume of the lungs in so small an animal as the mouse induced DURAND and GIROUD to try the possibilities of obtaining a greater yield on the lungs of rabbits after tracheal inoculation, as certain organs of this animal had been proved capable of growing rickettsiae in large numbers (cf. VIOLLE, 1937). This attempt proved fruitful (1941). Indeed, although the lung of the rabbit yields per unit of weight only half that of the mouse, the pulmonary organ of the rabbit (40 g.) will give 660 c.c. of rickettsiae emulsion[3] of the same concentration as the 20 c.c. yielded by the lungs of a mouse (0·30 g.). This result, however, is not obtained without a previous adaptation of the virus to the rabbit and, for this purpose, the experimental animal's resistance must be lowered. Various means are used: X-ray exposure, splenectomy, anaesthesia, bleeding and, more often, toxics, and shaving with exposure to the cold.[4] Thus prepared, the rabbit is submitted to ether anaesthesia and intratracheal inoculation. The virus used for this inoculation is made up of the lungs of a mouse ground and diluted in 5 to 10 c.c. of a mixture of Tyrode solution and horse serum in a proportion of 1 : 6 (later an emulsion of rabbit-lung may be used). The disease induced in the rabbit develops somewhat irregularly. However, the temperature reaches and even exceeds 40° C. on the 3rd day and remains high during 5 days, if death does not occur on the 7th or 6th day in hypothermia. Pulmonary infection reaches its maximum between the 5th and the 7th day.

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[1] The average weight of the hepatised lungs varies from 0·30 g. to 0·40 g. for young mice weighing 16 to 18 g.

[2] For the "sero-protection" dermal test, see GIROUD, 1938[a] and 1938[b].

[3] At the Pasteur Institute of Algiers Ed. Sergent obtains 900 to 1,000 c.c. of vaccine from a 4-lb. rabbit, an amount sufficient for about 300 vaccinations.

[4] At the Cantacuzene Institute Dial poisoning and cold are used.

After being ground with Latapie's apparatus with steel beads, the organs are suspended in saline solution containing 2 per 1,000 of formalin. They are then submitted to fractional centrifugation and the sediment which contains the rickettsiae is diluted and formalinised at 2 per 1,000.

The formalinised suspensions of rabbit rickettsiae, even when used a considerable time after preparation, give to the guinea-pig efficient protection against the intraperitoneal inoculation of some 500 infecting doses. Their potency is practically equal to that of formalinised or phenolised suspension of mouse-lung.

In man (14 individuals from France proper), rickettsiae suspensions from rabbit-lungs have given rise to the production of antibodies exactly as mouse-lungs have—the same agglutinins against the Prœteus X, the same rickettsiolysines, as revealed by the dermal sero-protection test. It is to be noted that, at the doses used (0·2 c.c. or 1 c.c.), the injection of the vaccine did not cause a general reaction.

Whether dealing with mice or rabbits, it may happen that inoculation does not produce in the first animals of the series complete pulmonary hepatisation, or the appearance of abundant rickettsiae in their bacillary form. There is then found in the lung only red granular bodies and punctiform elements, either ruby-red or blue when stained with Macchiavello's method. GIROUD and PANTHIER (1942[b]) have carefully observed and interpreted the phenomenon. The stimulation of virulence, or rather the progressive adaptation of the typhus virus to the mouse or to the rabbit, that is produced by successive passages in those species gradually causes the disappearance of these forms of degeneration (homogeneous ruby-red bodies, pink inclusions), forms of agglutination (granular red bodies) or forms of resistance (punctiform elements) and, inversely, favours the multiplication of the bacillary forms of rickettsiae which alone constitute good antigens.

The knowledge of the significance of the various forms of the virus in the course of its adaptation to an animal species has therefore an undoubted practical interest.

The same authors' observation that preservation of the infected lungs, whether in the cold state or in the dried state, causes them to lose their virulence for the guinea-pig and their antigenic properties is also of practical importance.

(*c*) *Vaccines obtained from dog-lungs by Combiesco, Zotta and others*

COMBIESCO and his co-workers ZOTTA, MANCIULESCO, POP and TASCAU (1941, 1942) succeeded in their endeavours to cultivate rickettsiae in large numbers on the lungs of puppies inoculated through the respiratory tract and kept in a cold room (at about 0° C.).

The yield obtained by these authors was about the same as that which Durand and Giroud obtained with rabbit-lung, i.e. proportionately about half that of the mouse-lung but, in fact, considerably greater, in view of the weight of the animal used.

The formalinised vaccine prepared with rickettsiae obtained from the lungs of dogs gave a strong protection to the guinea-pig. The results of the trials on man that the authors had in mind have not yet been published.

(d) General observations on lung-vaccines

1. *Dangers of the culture of rickettsiae on the lung*

The inoculation and use of mouse-lungs to prepare rickettsial emulsions present a serious drawback, i.e. the danger they entail for the laboratory worker. Thus, at the Hygiene Institute of Zürich, Mooser's six collaborators acquired a laboratory typhus from a murine strain inoculated on the lungs of mice; 3 proved severe. Of the 7 persons exposed to infection through the air polluted by the sneezing of the animals during the nasal inoculation, the only one who did not show a clear-cut form of typhus, but merely an influenza-like syndrome, had contracted typhus 14 years previously in handling the same virus (LÖFFLER and MOOSER, 1942).

At the Cantacuzene Institute at Bucharest, all members of the two teams of 6 and 4 physicians respectively which in succession prepared lung-vaccine (historical strain) suffered from an attack of typhus in spite of the usual precautions for the first team, reinforced for the second. The presumed portal of entry was, in two cases, the skin of the hands (microscopic abrasions by brushing) and, in the others, the ocular and respiratory mucous membrane. It is known that the instillation of a drop of virulent emulsion on the nasal mucosa (NICOLLE and SPARROW, 1932[b]), or on the conjunctiva, regularly induces in man a general typhus infection without any local reaction (NICOLLE and SPARROW, 1935; SPARROW and MARESCHAL, 1938). BLANC, BALTAZARD and DONNADIEU (1938) consider that the mucous membranes are a frequent portal of entry for typhus infection. It seems likely, therefore, that this is the explanation of the cases cited above.

The prophylaxis of these accidents in the laboratory thus seems to comprise, in addition to the reinforcement of ordinary precautions, the wearing of a mask with eye-glasses. The case of an individual infected merely by entering the room in which nasal inoculations of mice had taken place shortly before, reported by LÖFFLER and MOOSER, suggests that the masks ought to be worn for some time after the operation, unless the room is abandoned for some time until infective droplets in the air have been deposited.

In the Bucharest cases, previous vaccination of the workers with Weigl's vaccine did not prevent infection but it seems to have reduced its severity, especially in the case where vaccination was recent. It appears therefore essential to vaccinate and, if necessary to re-vaccinate all workers likely to have contact in the laboratory with typhus virus. It must be realised however, that the enormous virulent doses they have to handle can break down the most solidly established immunity. Such is the opinion of Weigl, in whose laboratory 22 cases of typhus have been contracted.

The auto-observation published by FINDLAY (1941[b]) is illustrative of this

point. This author received 3 injections of Weigl's vaccine in January 1940 and 4 injections of mouse-lung vaccine in April, after which his serum gave a positive Giroud's dermal test. He contracted a mild form of typhus in June 1940, a murine strain being recovered from his blood on the second day.

The typhus epidemic, described by EYER, PRZYBYKIEVICZ and DILLENBERG (1940) in a laboratory preparing Weigl's vaccine, showed also the relation between the severity of the disease and the time elapsing between vaccination and infection, as well as the infectiousness of dry virus (faeces of lice) as compared with virus in the organs of the guinea-pig.

2. *Application of lung-vaccines*

The risks involved in cultivating typhus virus on lungs have not, in practice, prevented the experimental study, if not the large-scale production, of lung-vaccines.

We know that Giroud at the Pasteur Institute in Paris prepares large quantities of rabbit-lung vaccine. Ed. SERGENT at the Pasteur Institute of Algiers has recently set up a service for the preparation of the same Durand–Giroud vaccine. MOOSER at Zürich, JONESCO-MIHAESTI, M. CIUCA and their co-workers at Bucharest, and Et. BURNET at the Pasteur Institute of Tunis are preparing mouse-lung vaccines (Mooser alone using the murine strain).

Human vaccinations in quantities that are not negligible have been carried out on medical and sanitary personnel, both in Poland and in Germany, in prisoners' camps. Circumstances have not so far permitted the scientific study of these vaccinations, and still less their publication. Their innocuousness is, however, well-established, and their preventive efficacy, which has been proved in animals, may be inferred both from the human trials made and from analogy with other killed vaccines; since this is a case of homologous vaccination, i.e. historical killed virus against historical typhus, Mooser's murine vaccine excepted. We know that, in Roumania, mouse-lung vaccine has been applied on a large scale to individuals greatly exposed to typhus contamination and that it has given satisfaction: the number of typhus cases among the vaccinated was small and in those cases the severity of the disease was distinctly lessened.[1]

3. *Conclusions regarding lung-vaccines*

Owing to their innocuousness and their efficacy and to the possibilities of their large-scale production, vaccines made from lung-culture may have a wide practical application for prophylaxis, in spite of the risks involved in their preparation. It may be hoped that publicity will one day be given to the results of comparative trials of these vaccines on a large scale, together with those of Cox and Weigl, and that precise data may be made available

[1] Personal communication from Professor M. Ciuca.

concerning the actual yield and cost of the various processes. As production is, at present, very largely confined to military or militarised laboratories no definite information is available.

C. Detailed study of live vaccines

1. *Historical*

It may not be superfluous to cast a brief glance backwards at the long series of attempts to use for vaccination the live historical typhus virus. The record presents, unfortunately, a long series of failures.

NICOLLE, as early as 1916, SPARROW in 1924, NICOLLE, SPARROW and CONSEIL in 1926, had attempted *dilution* of the virulent material in order to obtain an immunising effect from the use of very small doses of infective material. These attempts had to be abandoned, in spite of some experimental successes, owing to the irregularity of the results and the dangers involved in their failure, classical typhus in man never being benign.

The attempts to *attenuate* historical virus were not more fortunate; whether attenuation was attempted by cold (NICOLLE, SPARROW and CONSEIL, 1927), by the addition of phenol, of formalin, of iron hydroxide (BALTEANU and CONSTANTINESCO, 1937) or by coating first in lanoline and afterwards in olive oil (COMBIESCO, 1937), failure has been constant. Virulence either disappears completely—and then no immunity results—or persists unaltered —and inoculation typhus occurs.

It must be added that, if the use of live virus of *historical* typhus presents risks for the vaccinated themselves, it is equally dangerous for the communities where vaccination is carried out, if the population carries lice. In certain cases, indeed, the historical virus inoculated in that fashion may produce no symptoms in the individual vaccinated, but may nevertheless enter his circulation and eventually infect lice. RAMSINE (1929), YU (1931), KUTEISCHIKOFF, DOSSER and BERNHOFF (1933), BALTEANU and CONSTANTINESCO (1937) have proved the virulence of the blood in certain individuals inoculated with historical virus, in spite of the absence of morbid symptoms (unapparent typhus).

The fact that certain strains of murine typhus gave a comparatively mild disease in man, as shown in America by MOOSER, MAXCY, DYER, and in Africa by the Tunis workers, and, on the other hand, the proved existence of a cross-immunity between its various types (Toulon ship fever, Mexican tabardillo, murine typhus of Tunis and Casablanca) and also of a cross-immunity between historical and murine typhus suggested the use of a murine virus of low initial virulence, artificially attenuated, for the vaccination of man against classical typhus. There was reason to hope, on the one hand, that the possible failures of the methods would not entail serious risks for the vaccinated or for the community, owing to the low virulence of the strain used. On the other hand, it might be expected from the intensity and long duration of the immunity engendered by live viruses that a live vaccination, even cross-vaccination (heterologous), would give a strong and lasting protection. Since, however, no strain of murine typhus

had been found of sufficiently low virulence for man to be employed directly as a live virus-vaccine, a practical method for attenuating the available murine strains had still to be discovered.

2. *G. Blanc's biliated murine virus-vaccines*

Georges BLANC, who, at Athens, had, by adding bile to the virus of dengue, modified it in such a way as to make it capable of inducing immunity without producing a clinical disease (1930), thought of applying the same substance to the virus of murine typhus.

Many experiments on the guinea-pig, followed by trials on man, showed him that typhus virus acted in the same way and that this virus, mixed in suitable proportion with bile, conferred immunity on man without causing in him a febrile disease.

Action of bile

What is the mechanism by which bile acts in this case? Blanc and his co-workers humbly confess that they cannot explain it with certainty. They think that it is a physical-chemical action and that bile constitutes a kind of coating for the virus. They showed that bile did not kill any part of the virus; that it did not act as a mere diluting agent: a dose of murine virus which infects the guinea-pig intraperitoneally is always infective for man (far more receptive to typhus virus than are rodents). On the other hand, the same dose, biliated, while remaining infective for the guinea-pig, ceases to produce a febrile reaction in man, although still conferring immunity on him. One cannot speak, moreover, of an *attenuation* of the *strain*, in the strict sense of the word; that is to say, an attenuation that can be transmitted, since the virus inoculated to the guinea-pig after this passage retains its original virulence unabated.

BLANC and BALTAZARD (1937[a]) have shown that the virus-bile complex acted more like a physical-chemical than a biological combination, and hence the importance of a possible dissociation of that complex:

> The virus, inoculated pure, in the skin of the guinea-pig, produces a strong local reaction, followed by a general infection with the usual scrotal reaction.
>
> The virus, mixed with bile, inoculated under similar conditions, does *not* produce any local skin reaction, nor a febrile reaction. It produces, however, an unapparent infection, followed by immunity.
>
> The same mixture of bile and virus, inoculated into the *peritoneum* of the guinea-pig, acts as a pure virus (unbiliated) through dissociation, as it seems, of the bile-virus complex in the peritoneum.

For a phenomenon of physical-chemical balance of this kind, the relative doses of bile[1] and virus are of importance. In his first method of vaccination, using the organs of infected guinea-pigs as virus, BLANC used a 5% bile content in its guinea-pig virus suspension at 1/2,000 (emulsion of the virulent organs of *one* guinea-pig in 2,000 c.c. of normal saline solution).

[1] The bile used is prepared as follows: Gall-bladders of oxen are removed immediately after slaughtering. The bile is precipitated by being kept for half an hour in an autoclave at the temperature of 120° C., filtered on moist filter-paper, distributed in ampoules of 20 c.c., which are later sealed and sterilised in the autoclave at 110° C.

(*a*) *First method of Blanc, using organs of guinea-pig inoculated with murine virus*

Blanc used, as a source of virus, a strain isolated from a rat taken at Casablanca which he called T.M.C. III (typhus murin de Casablanca III). This strain was kept by passage on male guinea-pigs among which it caused regularly Neill–Mooser's scrotal reaction. These animals are inoculated intraperitoneally with a mixture of ground spleen, tunica and adrenals.

Blanc has given his technique in great detail in the *Revue d'Hygiène* (vol. 58, pp. 252–272, 1936), to which the reader is referred. We shall merely transcribe the summary Blanc gave in *Le Maroc médical* in June 1937:

> A guinea-pig, inoculated with murine typhus, is killed during the febrile period, at a time when the scrotal reaction is well marked. The tunica, the spleen and the adrenals are removed aseptically and carefully ground; the pulp obtained is diluted in 2 litres of saline solution and filtered on fine gauze. This is a virulent dilution at 1/2,000 and is used to prepare the vaccine.
>
> For this purpose, shortly before use, there is added to 95 c.c. of virulent dilution 5 c.c. of sterilised ox bile; the fluids are left in contact for 15 minutes and the individuals to be vaccinated receive the biliated mixture in the deltoid muscle: 1 c.c. for adults, 0·5 c.c. for children between 8 and 15, and 0·25 c.c. for children between 1 and 8.

This technique of extemporaneous preparation, giving 2,000 doses at least in one operation, is particularly well-adapted to mass vaccination. Thus it is that such vaccinations were carried out in Morocco from 1934 to 1938 by hundreds, and later by thousands, *at a time.*

Effects of the inoculation

For a method which has received as wide an application as that of the guinea-pig organs vaccine of Blanc, it is not necessary to enumerate the series of convincing animal experiments which led to its use in man. It may be useful, however, to recall the first individual trials of the vaccine in man, in view of their clear-cut experimental character, and the light this throws on the collective trials made later, which brought about the extinction of epidemics.

Nineteen individuals (North Africans) received the biliated emulsion of organs of guinea-pigs infected with the T.M.C. III strain. They gave no general reaction. Twenty-five days later, they were subjected to inoculation of virulent murine virus. None reacted. They were therefore immunised against the homologous virus. Six of the vaccinated were later tested with classical human virus; they resisted infection. They were therefore immune to the heterologous strain. Inoculation to three vaccinated individuals, volunteers, of 600 infective doses of historical virus caused no reaction amongst them, while a control monkey died in 17 days with bulbar paralysis and presented in its central nervous system the typical nodules of classical typhus.

Vaccination of the inmates of jails, in which typhus prevailed endemically, at Adir (1934), and Ali-Moumin (1936) was carried out, under conditions of strict observation, on 723 and 823 individuals respectively. It brought

about complete disappearance of typhus in these institutions, without any change in the living conditions or in the louse-infestation of the prisoners. There again, control virulent inoculation of volunteers showed the effectiveness and intensity of the protection conferred on the vaccinated.

Among the prisoners at Adir, it was possible to determine with precision the proportion of reactors to vaccination, i.e. of the vaccinal infections showing symptoms; it was 1·5%, a proportion later confirmed by many series of vaccinations carried out amongst natives of Morocco.

The effectiveness of mass-vaccination in stopping and eradicating typhus in an epidemic focus prevailing in a free population was first proved on a large scale in the town of Petitjean in Morocco. The population, numbering 8,234, was systematically vaccinated; on March 27, 1935, 8,122 natives and 12 Europeans were inoculated. As reported by GAUD (1935):

> After the mass vaccination of the population, and without any other measure being taken, the epidemic, which had already caused 22 known cases and 6 deaths, stopped abruptly, and this stoppage occurred within the limits of time exactly necessary to the development of immunity amongst the vaccinated.

After a similar experiment, carried out on the 12,000 members of the Abd-el-Ahmed tribe, the health authorities of Morocco were of opinion that the experimental stage was ended, and since, as a result of drought, a serious epidemic prevailed in the country, vaccinations were undertaken on a large scale. In less than three months, over 20,000 vaccinations were carried out at Casablanca and 75,000 in the surrounding districts, 168,000 at Marrakesh, and 70,000 in the neighbouring country, etc. Rather more than a million vaccinations were effected between November 1937 and June 1938, in addition to the 276,000 carried out previously.

The epidemic wave was broken (cf. GAUD, 1938, 1939).

The magnitude of these figures clearly shows that, generally speaking, the results were found satisfactory. These results, however, were not absolutely uniform; different lots of biliated vaccine sometimes differed rather markedly from one another in the percentage of febrile reactions they caused, and in their prophylactic efficacy.

Individual reactions to the inoculation of biliated guinea-pig organs vaccine

A clear distinction must be made between the reactions that the vaccine produces among Moroccans living in the native environment—whether they are Berbers, Arabs, Jews, or Negroids—and among the Europeans. In the Moroccans, among and for whom the method was evolved, inoculation of the emulsion of biliated organs of guinea-pigs at 1/2,000 and even 1/1,000 and 1/500 (BONJEAN, L.o.N. Document C.H.1236(*a*), p. 26) used experimentally in the beginning, did not give immediate reactions. Thus, the individuals vaccinated in a jail were able to resume work the very day on which they were vaccinated (BLANC, C.H.1236(*a*), p. 25). Febrile reactions appear 10 days at least after inoculation in a proportion ranging from 1 to 1·5% amongst Moroccan natives. These reactions correspond to the evolution of a more or less attenuated murine typhus. The temperature

rises as from the 12th or even the 15th day after inoculation, and exceeds 38° C. (100·4° F.), sometimes 39° C. (102·2° F.), for about a week.

> No clinical or other sign[1] except complete absence of stupor and the excellent general condition of the patient enables "vaccinal reaction," i.e. murine typhus, to be distinguished with certainty from true typhus (BALTAZARD, 1938).

In view of the fact that the incubation of classical typhus lasts from 10 to 12 days, it may easily happen that, in an epidemic focus, authentic fatal classical typhus cases, contracted at about the time of vaccination, may be labelled as "vaccinal reaction," since these cases occur precisely at a time when true vaccinal reactions are to be expected (BALTAZARD, *loc. cit.*).

In Europeans, the evolution of vaccinal reactions is about the same as in the natives. However, their frequency is very much greater and, in certain cases, has even amounted to as much as one-third of the vaccinated. For this reason, BLANC has "except in emergencies, given up systematic vaccination of Europeans living in Morocco who are not directly exposed to contamination" (i.e. other than physicians, nursing staff and certain officials).

An attempt to use biliated vaccine during the 1936 typhus epidemic in Chile gave results in sharp contrast with those of the vaccination carried out in Morocco. Of 550 individuals inoculated with Casablanca II virus, 227 showed febrile reactions, 5 of which were fatal.

As no impartial and strictly scientific study has been published concerning this unfortunate attempt, such as the one by which Bruno LANGE rehabilitated BCG after the Lübeck tragedy, we cannot gauge the significance of the accidents caused by biliated vaccine in Chile. We may note, however, that according to the statements of SUAREZ at the Typhus Conference of 1937 (L.o.N. Document C.H.1236(*a*), pp. 23–24, 28, 41–42), vaccinations in Chile were carried out by a person who had not had sufficient experience and who employed suspensions of 1/1,000 and even 1/800 of guinea-pig, instead of 1/2,000.

Suarez was the first to call attention to one objection that might be brought against Blanc's method, viz. the impossibility, owing to lack of time, of checking the bacterial purity of the rickettsial emulsions and, consequently, the possibility of their contamination by accidental *Pasteurella* or *Salmonella* infections in the guinea-pigs.

At the same Typhus Conference, ZINSSER drew attention to another and cognate disadvantage of the method—viz. the impossibility of titrating the murine virus in the guinea-pigs used for vaccine preparation. Generally speaking, the emulsion is made up from a single guinea-pig and, even if the latter was killed at what seemed to be the height of the infection, there may be marked differences in the intensity of that infection as between one animal and another. Consequently the virulence and antigenic potency of the emulsion used may vary from one vaccination series to another.

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[1] JULLIARD and HENAFF (1939) have called attention to the constancy of low level of sodium-chloride excretion in historical typhus and the rarity or insignificance of that symptom in murine typhus. It is, however, merely a question of degree.

Danger to the community resulting from the use of a live virus-vaccine

With regard to the objections brought against the use of biliated vaccine, as against that of any other live typhus vaccine, mention must finally be made of the danger to the community occasioned by individuals who, with or without symptoms, might carry in their circulation inoculated murine virus, which might *a priori* be picked up by parasites (lice or fleas) and transmitted to other persons. The fear of this was, from the very beginning, in the minds of Blanc and his co-workers, and of the Morocco health authorities, who made a point of conducting many controlled experiments in order to satisfy themselves that this fear, though founded in theory, was not, in actual fact, justified.

> A series of volunteers, purposely selected as carrying large numbers of lice, were inoculated with murine typhus virus. Every second day during the incubation period, and during the disease and convalescence, lice were taken from them and were ground and inoculated to guinea-pigs. These lice never transmitted infection. Furthermore, a series of experiments on the transmission of murine typhus from man to man by its normal vector, the flea, also proved negative (BALTAZARD, 1938).

If such negative results are obtained with a non-biliated murine virus, the risk with a biliated virus is clearly far less. Indeed, inoculation to Moroccan natives with a *normal* murine virus induces inoculation typhus in 90% of cases, whereas the inoculation of a *biliated* murine virus produces a febrile reaction, that is to say, inoculation typhus, in less than 1·5% of cases.

In point of fact, the strongest argument against the view that secondary typhus foci are created by vaccination, is furnished by the observation of vaccinated communities. Even in penitentiaries, or in tribes suffering from food scarcity, cold and under-nutrition, vaccination leads, not to the appearance, but to the disappearance of typhus foci. Even in Chile, according to SUAREZ, not a single secondary case of typhus was observed among contacts of patients (L.o.N. Document C.H.1236(*a*), p. 41).

However that may be, certain authors consider it undesirable to import murine virus, such as a North African virus, into a country where this virus does not already exist, and where the population might be more receptive and more sensitive to murine typhus than are North Africans. Thus, CIUCA and JONESCO-MIHAESTI (1941) are opposed to the importation of African murine viruses into Roumania, although historical typhus prevails endemically in a part of that country.

Other authors object to the use of a live murine virus for the opposite reason that it might, once it had been introduced into a country by vaccination, lose its original character and become classical virus (MOOSER, 1941).

Efficacy of vaccination, appearance and duration of immunity

Immunity appears early. It is already established by the 10th day, since individuals inoculated with pure virus 10 days after vaccination do not become infected by it. Immunity is perhaps obtained earlier, but proof

is not forthcoming (BLANC, L.o.N. Document C.H.1236(*a*), p. 20). Moreover, when vaccination is started in an infected environment, the epidemic ceases, as a rule, after 2 or 3 weeks.

Epidemiological observation seems to indicate that the protection conferred persists for several years (non-reappearance of typhus at Petitjean, for instance, several years after mass-vaccination had been carried out there). The tests of the immunity provided by virulent inoculations, which BLANC and BALTAZARD reported to the French Academy of Science in August 1939, showed that febrile murine typhus gave man perfect immunity against both murine and epidemic typhus for at least 5 years. Further, *unapparent* murine infection through biliated murine vaccinations protected against epidemic typhus for at least 5 years and 5 months. Thus, even if all persons vaccinated with biliated virus are not immunised, those at any rate who have received an unapparent infection are protected for a considerable period of time. Biliated vaccine, indeed, does not, any more than any other vaccine, give immunity in 100% of cases.

As regards troops, whose health is under particularly careful observation, the number of failures of vaccination amongst Moroccan soldiers vaccinated during the epidemic of 1937–38 (that is to say, the number of cases of epidemic typhus occurring over 28 days after vaccination) was 0·1% (2/1,949) in the garrison of Casablanca (LAURENS, FORT and BERNIER, 1939) and 1·5% (28/2,021) in that of Marrakesh (JULLIARD, HENAFF and POUBLAN, 1939). The proportion of vaccinal murine typhus cases (admitted to hospital) was, in these garrisons, 1·22% and 1·38% respectively. It was 1·08% for the whole of the 8,389 soldiers vaccinated in Morocco.[1]

(*b*) *Second method of Blanc, using dried murine virus obtained from infected fleas*

Realising that they could not obtain from guinea-pig organs material for immunising inoculation possessing constant virulence and capable of conservation, BLANC and BALTAZARD (1938[b]) conceived the idea of trying to obtain such material from fleas infected with murine virus.

The fleas of the rat or of man are easily infected by biting a rat inoculated with murine typhus (DYER, WORKMAN and CEDER, 1932; MOOSER, 1932); they become virulent 48 hours after the infective meal and they remain so for the rest of their lives, their faeces being also infectious, as DYER had pointed out (1937[b], 1937[c]). These faeces, as was first noticed by ARKWRIGHT and BAGOT (1923), were found by Blanc and Baltazard to retain their virulence for a very long period—for two years at least.[2] Their infectiousness is very marked since, even after two years conservation, 1/100 milligramme, that is to say 1/10th of a single dejection, is sufficient to infect man, whilst 1,000 times that dose is necessary to infect the guinea-pig through the peritoneal route (cf. BLANC and BALTAZARD, 1938[a]). Since

[1] For the effect of vaccination in Morocco, see also DUPOUY (1938).

[2] STARZYK (1938) made the same observation concerning the *Rickettsia prowazeki* contained in the faeces of lice and in the dead lice.

the virulence of the virus of the dry faeces of lice remained constant, this virus could be titrated. The problem was to obtain faeces from fleas in which the virus content would be as high and as constant as possible. This result may be achieved by feeding fleas exclusively on rats at the height of typhus infection and constantly renewed, and by using the faeces from a very large number of fleas, thereby compensating any possible differences in the virus content of different samples.

(*c*) *Technique*

From the detailed description of his technique published by BLANC (1939/40), we extract the following data:

Large-scale breeding of rat fleas (*Xenopsylla cheopis*) is undertaken in a special room, well isolated, in large stoneware jars, the bottoms of which are covered with a layer of bran. Some of these are used for breeding fresh fleas, others for keeping infected fleas and rats for the purpose of collecting the virus.

> In a jar containing about 50,000 fresh *X. cheopis*, recently hatched and not having bitten as yet, are placed two white rats, the lower incisors of which have been cut, and which have been inoculated 48 hours previously with T.M.C. III murine virus, taken from a passage guinea-pig. These rats usually die, or are killed, after 48 hours. Their bodies are freed from their fleas and burned. Immediately afterwards, two other rats, inoculated in the same fashion, are placed in the jar, the same process being repeated every second day. From the end of the second week, when all the fleas have been duly infected, the collecting begins.
>
> Every 48 hours, the two dying rats are killed and carefully plucked. The hairs, matted with flea dejections, are placed in a dessicator in vacuum in contact with anhydrous calcium chloride. Next day, the dried dejecta are easily separated from the hair by screening on silk screens of increasingly fine mesh; as soon as it is collected, this material is distributed in ampoules, which are then sealed in the vacuum produced by a rotating pump giving 1/100 mm. pressure.
>
> Every fifteen days, infected fleas are transferred to another jar in order to prevent the hatching of fresh fleas. Throughout the process, the only rats placed in the jar will be rats infected with guinea-pig passage virus, so that the infection of the fleas will be continually reinforced.
>
> Thirty to forty collections, aggregating some 10 g. of dejecta at least, are thus made in each jar, after which, as the number of fleas declines, the jar is no longer used.
>
> Usually two jars are employed at the same time. If necessary, 40 g. of dried virus could be produced every month without difficulty. (This corresponds theoretically to 4 million vaccinating doses of 1/100th of a mg.)
>
> Although all the dejecta collected may be considered as of equal virulence, all the material collected from the first jars is mixed together to make up lots, of which the virulence is titrated. The stocks are kept, as a precaution, in the ice-box at + 4° C., in the dark, the sealed ampoules being kept *in vacuo* in desiccators containing calcium chloride.
>
> For use, the dried virus is dissolved in a buffer solution with a pH of 7·5 constituted by
>
> | | |
> |---|---|
> | Crystallised disodic phosphate .. .. .. | 17·91 g. |
> | Hydrochloric acid N/1 .. .. | 8 c.c. |
> | Distilled water .. .. | Q.S. for 1,000 c.c. |
>
> (i.e. $NAHPO^4$ M/5 250 c.c. + HCL M/5 40 c.c. + distilled water 710 c.c.).

To this solution sterile ox-bile is added in the proportion of 6·66 per 1,000. The biliated buffer solution is distributed in 100 c.c. bottles with a tight-fitting closing device. When filled, these bottles are sterilised. The virus powder is in ampoules sealed *in vacuo*, containing 1 mg., i.e. 100 vaccinating doses. A few c.c. of the buffer solution, introduced into the ampoule by means of a syringe, dissolve the virus, which is then emptied into the special bottle. The vaccine thus prepared can be used for several hours.

Application and results of Blanc's second method

The first trial of the new vaccine was made on 93 volunteers of a first group of 1,073 individuals inoculated in a relief shelter. These volunteers received, after vaccination, 100 virulent doses of dried virus or strong doses of guinea-pig virus (this control virus infected the guinea-pigs regularly). Only 3 individuals showed the febrile reaction of murine typhus.

Unfortunately, it was impossible to ascertain what proportion of individuals in the group had been previously vaccinated with the guinea-pig vaccine or had suffered from an attack of typhus.

A second trial experiment was therefore carried out in a penitentiary which had been free from typhus for several years, the inmates of which had not previously been subjected to vaccination. 60% of the individuals vaccinated with the dry vaccine and reinoculated with virulent material proved immune.

Although Blanc and Baltazard hoped to increase this proportion by improving their technique, they thought the result sufficiently satisfactory to justify the practical use of the new vaccine. Thus, in Morocco, 31,584 persons were vaccinated during the second half of 1938 and 212,400 in 1939 (GAUD, 1939). At the beginning of 1940, the number of vaccinated persons in Morocco aggregated 271,666. A large number of those vaccinated could be observed carefully, e.g. the troops (over 37,000), medical staff, inmates of prisons, etc. Reactions were exceptional—5 only, to the knowledge of the authors (1940). Of some 30 Europeans vaccinated, none showed a general or local reaction, in spite of the fact that some of them received doses three times larger than normal.

Mention should be made of the observations reported by GONNET (1942) who, in the Oujda region, where a vaccination campaign with the dried vaccine had been carried out in May 1941, saw cases (115) of benign typhus about mid-July in the tribes vaccinated. Some of these cases were of very short duration and suggested to him late vaccinal reactions. In 6 cases admitted to hospital, 5 of which were military cases, he diagnosed vaccinal murine typhus.

But, as the author points out, "no symptom or titration permits a diagnosis to be made with certainty as between historical typhus and vaccinal typhus," and since no result of animal inoculation is available to identify the virus in these cases, their nature is far from clear. More definite data would be required before vaccination could be held responsible for them, either on the score of its innocuousness (vaccinal reaction) or of its efficacy (historical typhus).

The new dried vaccine of Blanc can be transported and kept anywhere,

and can be used by any physician and not only by teams comprising laboratory technicians. It thus offers considerable practical advantages as compared with its predecessor, apart from its technical advantage of constant dosage.

The fact that this vaccine may be prepared rapidly in considerable quantities and may be kept in a very small space makes it possible to prepare stocks for the purpose of coping with epidemics on the largest scale. It is to be hoped, therefore, that many and precise observations will be published to verify its innocuousness for Europeans and for other persons living outside endemic zones, as well as the regularity of its prophylactic action amongst them. Experiments on natives in a country where typhus is endemic cannot, unfortunately, establish beyond discussion these essential points.

3. *Laigret's vaccines with murine virus in fatty coating*

(*a*) *First method of Nicolle-Laigret* (*brains of rats and guinea-pigs*)

Shortly after the publication of the results of the first vaccinations with the live murine virus by Blanc in Morocco, NICOLLE and LAIGRET (1935, 1936), at Tunis, who had invented a method of vaccination against yellow fever by fatty coating of the virus in the dried brains of mice, conceived the idea of applying the same method to the preparation of a vaccine against typhus.

Having found it difficult to preserve the typhus virus in the mouse, these authors used as virulent material the brains, first, of the guinea-pig and, later, of the rat, this animal being found more receptive.

The brains were removed at the height of fever following the intraperitoneal inoculation of the animal. In the view of the authors, the fatty coating was intended not to kill the virus used, nor even to attenuate it *stricto sensu*, but to retard its absorption by the human body and to enable the latter to produce antibodies before the virus could invade the whole organism.

Nicolle and Laigret tried first to emulsify the brains in egg-yolk, which they afterwards inoculated in the form of an aqueous suspension. They obtained 3 febrile reactions (murine typhus) among the 9 men inoculated; a simple oil emulsion of dried brain produced 10 reactions amongst 153 individuals inoculated. Finally 110 individuals were inoculated with brains emulsified in egg-yolk, suspended in olive oil, without giving a single reaction. A control virulent inoculation showed that individuals thus "vaccinated" had become immune. The virus employed had been isolated by SPARROW from a rat caught in the port of Tunis. It produces fever in the rat and, fairly regularly, a scrotal reaction in the guinea-pig: it is kept by passage on white rats.

At the outset, Nicolle and Laigret had adopted the following dosage:

1st dose: 1/200 of a guinea-pig brain; then, after 25 days,
2nd dose: 1/200 of a rat brain; then
3rd dose: 1/50 to 1/12 of a rat brain.

At the end of 1935, they vaccinated with 2 injections only (guinea-pig brain followed, after 20 days, by rat brain). As from March 1936, they used 1 injection only, with rat brain.

Technique of preparation

The technique of preparation established by LAIGRET and DURAND (1936) is described as follows by LAIGRET in 1937 (L.o.N. Document C.H.1230):

Removal of brains of the rats.—The passage rats are killed on the 2nd day of their fever; their blood is first taken, with a syringe, from the heart, in order to make a blood culture. This culture must remain sterile. The brain is then removed, under sterile conditions, and placed in a tube containing glycerol. The cerebellum is taken and ground, and the product of this grinding is used, first, to make cultures for the control of bacterial sterility, secondly, to inoculate guinea-pigs, to control the initial virulence. The tube containing the brain in glycerol is placed in a refrigerator, where it is to stay for 24 hours at a temperature of — 15° C.

Drying.—The brain is taken from the glycerol. It is dried on a sterile blotter and placed in a mortar containing 2·50 g. of sodium phosphate (a mixture of 100 parts of anhydrous disodic phosphate and 5 parts of anhydrous monopotassic phosphate). The brain is ground with this powder; it consists then of a pulp, of which a loop is cultured in broth and on agar slant (second control of bacterial sterility). It is placed in a glass desiccator, the bottom of which is abundantly covered with melted calcium chloride. (Neither sulphuric acid nor phosphoric anhydride is suitable.) The air is removed and the desiccator is placed in the ice-chest at a temperature of about + 5° C. until the next day. The powder obtained after this first desiccation is the *mother-powder* which will keep its virulence in the refrigerator (at — 15° C.) for at least two months and will eventually be used, according to requirements, to prepare the final vaccines. Two rats and two guinea-pigs are inoculated intraperitoneally with 5 cg. of this powder. They must develop typhus (second control of virulence).

Coating in egg-yolk.—All the powder obtained from the desiccation of one brain, i.e. about 2·50 g., is emptied into a sterile mortar, and 8 c.c. of egg-yolk are added drop by drop (sterilised beforehand by the Tyndall process). The whole is mixed and the mortar with its contents is placed for 24 hours in a calcium-chloride desiccator, where vacuum is applied. Next day all that remains to be done is to grind the dried product and pass it through a sieve in order to obtain the final powder. A third control of bacterial sterility is then made.

Putting up of vaccine in tablets.—At first the vaccine was kept and used in its powdered form, but it was later found more convenient to compress it into tablets of 20 doses, each tablet representing 1/10th of the virulent brain of a rat. The tablets are kept in the ice-chest in small tubes emptied of air.

Coating in oil and vaccination.—Just before use, a tablet is ground in a mortar with 20 c.c. of olive oil, in order to make up the 20 doses of vaccine. The technique of this grinding is as simple as that applied for dried small-pox vaccine, when ground extemporaneously in glycerol. Any practitioner is capable of carrying out the process; it is even carried out on most occasions by an orderly, under the supervision of the vaccinating physician. The vaccine coated with its oily excipient is injected in the region of the shoulder-blade at the dose of 1 c.c., which corresponds to 1/200 of virulent rat brain.

Conservation and transport of the vaccine.—The tablets are kept in a refrigerator at — 15° C. They retain their virulence for at least a month; in practice, all the stocks are exhausted before that time. At ordinary temperatures, preservation of the virulence has been proved after 48 hours, which makes possible postal despatch

without special wrapping. It is, however, far preferable always to transport the vaccine at a low temperature. This is done in Tunis by using thermos bottles with carbonic snow. The same bottles are to be used whenever possible for postal despatch.

Application and results

Nicolle–Laigret's vaccine has been used in Tunis since November 1935. From that time until June 1937, 32,481 vaccinations were carried out in that country. The results were summarised in a memorandum published by LAIGRET, DURAND, BELFORT and LEFAUCHEUR (1937).

This study comprises not only a general picture of the innocuousness and efficacy of the vaccine, but also a series of monographic notes on the use of the vaccine in a number of native communities, with the results obtained. These notes show more clearly than any general description the sterilising effect of mass-vaccination in local endemic or epidemic typhus foci. A single instance will suffice.

At Sidi Naceur, a village of 539 inhabitants, free from typhus for years, an epidemic broke out on December 11th, 1935. The first vaccinations (guinea-pig vaccine) were carried out on December 18th (155), December 30th (179), January 8th (89); second injections (rat vaccine) were given on January 8th and 15th (398).

Sixty-two cases of typhus (8 deaths) occurred among the non-vaccinated; 6 among the vaccinated within 6 days after the first inoculation; 48 individuals remained unvaccinated on January 15th, at the end of the vaccination campaign; 3 of them contracted typhus during the following seven months. No vaccinated individual contracted typhus although, on the 30th day, the grindings of 100 lice picked up from the straw bedding and clothing of the inhabitants (in spite of previous delousing) infected guinea-pigs and rats.

Even allowing for the fact that vaccination does not take place in a community until a certain number of cases have occurred—and these cases are bound to bear on the rate of the non-vaccinated—these figures show the efficacy of vaccination, since the virus was abundant in the community and no typhus case occurred except during the first six days following vaccination, i.e. in individuals who were already in the incubation stage when vaccinated.

Sixteen persons vaccinated showed illness during the course of their vaccination, including the 6 cases of historical typhus already mentioned, 2 cases of typhoid and 8 fevers of a benign character, of which 2 at most might, from certain symptoms, be considered to resemble a very benign vaccinal typhus.

In the Maharès epidemic, of 91 persons who had been in contact with typhus cases, those who had been neither deloused nor vaccinated contracted typhus in the proportion of 100%; the percentage was 72% for those who were deloused but not vaccinated; it was but 8% for those who were both vaccinated and deloused, and all of this 8% were individuals who were already in the stage of incubation when prophylactic measures were applied (LAIGRET, *loc. cit.*, p. 620).

Many similar instances might be quoted. What is common to all of them is the *disappearance* of the epidemic when the number of vaccinated persons reaches a certain proportion of the population. The absence of typhus attacks among the vaccinated once the time necessary for the establishment of immunity has elapsed is also a constant feature. Thus, of the 29 cases of historical typhus observed among the vaccinated in the whole series, 27 occurred within the first 15 days following vaccination, which shows how early protection is brought about.

The vaccinations covered by the report were carried out in scattered communities where typhus was either endemic or epidemic. Their population aggregated 212,170, among whom, during the 20 months considered, there occurred 2,634 cases of typhus, with 298 deaths. Among the 32,481 vaccinated, 3 benign cases were recorded—failures of vaccination, which took place 33 days, 57 days, and 4 months respectively after inoculation. No deaths from typhus occurred among the vaccinated after the 30th day following vaccination.

Incidents of vaccination included local abscesses in 0·1% of cases, chiefly among women or infants, whose clothing or movements might cause the contamination of the vaccinating needle. There were 5 clear cases of vaccinal murine typhus, 4 doubtful cases, and 63 cases of light fever. Vaccinal murine typhus was naturally more common among the Europeans, who are more receptive to the disease than the natives.

	Europeans	*Tunisians*	*Total*
Vaccinations	1,766	30,715	32,481
Clear murine typhus	4	1	5
Doubtful murine typhus	4	4	8
Murine typhus total	8	5	13
Murine typhus total %	0·45	0·016	0·04

It must be noted that all 13 cases of vaccinal typhus were observed in towns.[1]

The number of vaccinations grew in the following months, reaching a total of 100,000. However, while Laigret was absent on an anti-epidemic mission in China—where he launched his method—incidents occurred which caused the application of the method to be suspended in Tunis.

(*b*) *Second method of Laigret–René Durand* (*mouse-brain*)

On his return, Laigret tried to remedy the fundamental disadvantage of his first method—that is to say, the absence of a titration for determining the quantity of active virus contained in the dose of vaccine, a disadvantage which on two occasions had, with the use of rat-brain vaccine, resulted in an abnormal proportion of vaccinal reactions.

For the purpose of preparing his vaccine against yellow fever, Laigret had made use of the fact that mice showed paralysis after inoculation with passage yellow fever virus adapted to mice. This paralysis gave concrete

[1] On the innocuousness of the Nicolle-Laigret vaccine, see ET. BURNET (1939).

evidence of the evolution of infection of the mouse, and made it possible to use that animal for testing the activity of the viruses.

Laigret thought he might make his typhus strain a paralysing one by giving his mice a joint yellow fever and typhus infection. To his satisfaction, he found that some mice showed paralysis as early as at the first passage of murine typhus virus alone (strains Port I or Marché), without the addition of yellow fever virus.

These passages are made from brain to peritoneum or from brain to brain. The intracerebral route produces a marked increase of virulence.[1] At the 19th passage, the virulence reached 10 million mouse-units, which means that 1 c.c. of an emulsion of mouse-brain in 10,000 litres of water would still contain active virus.

In fact, Laigret does not dare to use, for his vaccine, virus of such exceedingly high activity passed directly from brain to brain; on the contrary, he uses virus the virulence of which has been reduced by peritoneal passages.

Titration of mouse-virus

According to the adaptation of the strains, titration may be effected either through the peritoneal or through the cerebral route; both give practically identical results when they are employed in parallel.

> In order to titrate a fresh brain, it is ground in a mortar in 10 c.c. of saline solution. From this first suspension 1/10, 1/100, etc., dilutions are prepared. Each dilution is inoculated to at least two mice. 0·5 c.c. of each dilution is used for inoculation in the peritoneum, and it is easy to compute that the mice thus receive 1/20, 1/200, 1/2,000, etc., of a brain. Through the cerebral route 0·01 c.c. is inoculated (with a tuberculin syringe) so that the mouse receives 1/1,000, 1/10,000, etc., of a brain.
>
> The inoculated mice are observed and the appearance of paralyses noted. These occur after an incubation of from 5 to 6 days. This observation requires particular care, since generally the paralytic phase is of short duration and frequently the animal dies within a few hours. The control ends on the 12th day. Its result indicates the highest dilution with which the virus remains sufficiently active to cause paralysis in the mouse. The virus content of the material submitted to titration is thus expressed in *mouse-units*.

This titration is, of course, applicable only to those strains adapted to the mouse, which is the case for the mouse-brain vaccine-virus.

It should be added that the virus, which may be present in enormous quantities in the brains of mice, may remain active for a long time, at least 3 months in the refrigerator and at least 33 days at a temperature of 20° C. to 25° C. In suspensions, even in saline solution, the virus retains its activity for 15 days at 5° C.

This keeping quality of the virus is of obvious importance for the practical use of the vaccine.

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[1] Starting from the rat, however, the virus must be passed several times through the peritoneum before the strain is adapted to the mouse.

Preparation and titration of the mouse-vaccine[1]

As shown above, brains of mice inoculated through the peritoneal route are used. One brain is sufficient for preparing 1,000 doses of vaccine. As soon as paralysis in the mouse is detected, the animal is killed. A rapid thionine staining (cf. LAIGRET and AUBERTIN, 1938) shows the presence of rickettsiae in large numbers in the peritoneal exudate:

> The brain, removed aseptically, is placed in a sterile tube in the refrigerator until its bacterial control on agar and broth is completed. The brain is then ground with 40 c.c. of egg-yolk and 12·50 g. of anhydrous sodium phosphate (the whole corresponding to 50 g. of dried product and to 1,000 doses of 0·05 g.). After 48 hours in a vacuum desiccator in the ice-chest, the powder is ground again, sieved, submitted to fresh bacterial control on ordinary media and titrated.
>
> The titration is carried out by diluting a dose, i.e. 1,000th of a mouse brain, in 10, 100, 1,000 c.c. of saline solution and inoculating 0·5 c.c. of each dilution into the peritoneum of three lots of mice. According to whether these are paralysed by the first, second, or third dilution, it is ascertained that 0·05 g. of vaccine contains 20, 200, or 2,000 units.

LAIGRET finally adopted for practical use (1940) (cf. LAIGRET *et al.*, 1941) a virulence of 200 mouse-units per dose. The vaccines which do not reach or which exceed that dose are mixed with others so that the virulence is standardised at 200 units per dose.

Once titrated, the vaccine batches are kept in the refrigerator; they are later distributed in ampoules of 1, 2, 5, 10 and even 20 doses, as required.

Before use, the vaccine tablets are suspended in saline solution, and no longer in oil as in the original method. Laigret is of opinion, indeed, that the preliminary titration now carried out obviates the necessity for the precaution of using oil as an excipient. Oil retards the absorption of the vaccine but also slows up the vaccination itself and necessitates the use of a large calibre needle which is painful to the vaccinated person.

For practical reasons also, vaccination in two injections has been abandoned since May 1936, a single injection of 1 c.c. containing 200 mouse-units being used in preference. A team, consisting of two vaccinating physicians, two orderlies preparing the dilutions, boiling the needles, etc., and two other persons applying iodine previous to vaccination and counting the vaccinated, can, in a room provided with separate entrance and exit, vaccinate 600 persons in an hour—4,000 per day.

In towns and among Europeans, i.e. among individuals who are particularly receptive, Laigret vaccinated, as a matter of precaution, with three injections: First, 1 c.c. of a vaccine with a high antigen content (intracerebral passage) but killed by heat; this was followed a week later by the injection of 1 c.c. of live vaccine corresponding to 1 unit. After three weeks a third inoculation of 10 units of a live vaccine was carried out. The whole operation thus lasted about a month. Later experience showed that, even among Europeans, the new mouse-vaccine could be used at higher dosage and without the previous injection of killed virus. Experience showed also

[1] Cf. LAIGRET and RENÉ DURAND (1939).

that a lower dosage might be insufficient. Accordingly, Europeans now receive at first 1/10th of a dose, followed by a normal dose 25 days later.

However, 60 Europeans (physicians, policemen in Tunis, etc.) were vaccinated in 1939 with one injection of the normal dose of mouse-vaccine, without any appreciable reaction.

In July 1942, LAIGRET, FABIANI and VARGUES published the results of the experimental use of Laigret–Durand's vaccine through skin scarifications, a method which had already proved its efficacy in the case of other live vaccines (smallpox lymph, Laigret's yellow fever vaccine in the hands of Peltier, BCG tuberculosis vaccine (Rosenthal's method)).

No incident occurred in 316 persons vaccinated in Tunis in 1939, and a single case of vaccinal murine typhus showed itself among 991 individuals vaccinated in the neighbourhood of Algiers in March and April 1942. Nearly one-half of the 24 sera of individuals vaccinated showed rickettsiolysins when submitted to the serum neutralisation test in the skin of rabbits.

Encouraged by these results, Laigret carried out without accidents, in June 1942, in 116 adults, a mixed vaccination, against typhus and smallpox, through scarifications.

For the typhus vaccination, the technique is very simple: a tablet of dried virus of 20 doses is crushed in 20 drops of distilled or boiled water and with the pulp thus formed 20 vaccinostyles are loaded.

Application and results

From May 22nd, 1939, to May 9th, 1940, 200,488 vaccinations in one injection in aqueous excipient were carried out. *Not a single case* of vaccinal typhus was observed among the persons vaccinated. "Innocuousness was complete and constant." Confirmed historical typhus cases, appearing after an interval sufficient to eliminate those already incubating at the time of vaccination, did not amount to 20, i.e. 0·08%. Regarded as failures of the method, this is a strikingly small proportion for an endemo-epidemic milieu.[1] The results were sufficiently favourable for Laigret and his co-workers to consider that, with their new vaccine, the dangers of vaccinal murine typhus no longer exist and can therefore no longer be weighed in the balance against the dangers, which are always grave, of epidemic typhus.

With regard to vaccination by scarifications, time and more numerous observations will be necessary to judge of its value.

Notwithstanding the kindness of the authors in communicating the results of their work to us last year, even before publication, we are not, owing to present circumstances, in possession of the most recent observations concerning the use of live vaccines in North Africa during the last epidemic year. The publication of the results of the comparative experiment[2] con-

[1] In October 1941 LAIGRET and DURAND reported the continuation of the satisfactory results obtained with 220,000 vaccinated persons.

[2] Comparative tests of the different vaccines used in Algeria have just been published (DURAND, BEGUET, HORRENBERGER and RENOUX, 1942), but they relate only to the rickettsiolytic action of the serum of vaccinated persons, and not to the immunity conferred in a wide sense.

ducted in Algeria, the plan of which was drawn up by Professor Ed. Sergent (cf. *Bull. Org. Hyg.*, S.d.N., 6, 233–234) in agreement with Dr. G. Blanc and Dr. J. Laigret, would, we consider, be particularly desirable. Precise clinical observations, backed by laboratory tests, concerning Europeans would be of special interest at the present time when the vaccination of millions of Europeans may have to be envisaged.

IV. Summary and Conclusions

1. By comparison with the years preceding the present war, typhus is very markedly on the increase in the endemic countries of Eastern Europe, Spain and North Africa; moreover, in regions of Central and Western Europe hitherto free from the disease, it has made its appearance in sporadic form.

In Eastern Europe, however, it is far from having attained the epidemic gravity it assumed at the end of the first World War, and in the West it does not constitute a serious danger so long as the population continues on the whole to be free from lice.

Nevertheless, the increased destitution that might result from the prolongation and aggravation of the present economic disorder in Europe might, on the one hand, change the character of typhus, in those regions where it prevails in an endemo-sporadic form, into an endemo-epidemic one, and might, on the other hand, render regions habitually free from typhus liable to epidemics.

2. Lacking the power to influence economic conditions, or to prevent the risks of importation of virus as a result of troop movements between infected and healthy zones, health administrations should strive to make the populations under their care as unreceptive to the disease and as resistant as possible by preventive delousing and by vaccination.

Delousing should first of all be carried out among those elements of the population which are most exposed—vagrants, refugees, poor folk generally, and military and civilian communities. Vaccination should, in the first instance, be applied to medical and sanitary personnel. This merely precautionary vaccination should be effected with a killed vaccine.

3. If an epidemic breaks out, health authorities must further be prepared for the extension both of delousing and of vaccination measures.

If epidemic foci are few, if the population is stable and free from lice, and if sufficient stock is available, recourse should be had to a killed vaccine, beginning with the persons in contact with the sick and infected communities.

If, on the contrary, the epidemic is widespread and the population is a shifting one (troops, refugees, etc.) and is lousy, live vaccine should be used because of its rapid preventive action following on a single injection. In circumstances such as these, the very real dangers of historical typhus, both to communities and to individuals, far outweigh the risks of reactions to vaccination with an attenuated murine virus.

If stocks of killed vaccines are available, they could with advantage be used in zones less immediately exposed to epidemic invasion where, as the population is stable, time and material will permit of carrying out methodically, and at the proper intervals, the three injections necessary for an efficient use of killed vaccines.

4. Vaccination is to be carried out as an effective measure for the protection of the *community*, capable of preventing epidemics (either by reducing the susceptibility of the population or by increasing amongst it the number of immune persons).

Certain and absolute protection of all *individuals* vaccinated must not, however, be expected.

Killed vaccines do, indeed, give to all a certain amount of protection, but this protection, which generally reduces the severity of typhus infection, is not always sufficient to prevent infection.

As for *live vaccines*, the protection they confer is, as a rule, greater, but, unfortunately, appears to be less constant.

5. In case of emergency the selection of the *killed vaccines* will obviously be determined by the available stocks of the various types. If there is sufficient time for their preparation, preference will be given, in endemic countries, to local multiple strains, and, in countries free from typhus, to strains from those regions whence an epidemic invasion may be anticipated.

The method used in preparing killed vaccines will, of course, depend on the animals and materials to hand, but even more on the technical preferences of the local bacteriologists. Rickettsiae cultures on chick embryo and on lungs of rodents are, in fact, equally capable of supplying vaccine in quantity and, at the present time, proof is not forthcoming that any one of the killed vaccines is superior to the others in field application.

In spite of the efficacy of the product, Weigl's method of culture on louse intestine cannot be contemplated where mass production is called for.

6. As regards *live vaccines*, the new method of Blanc (biliated flea virus) and that of Laigret–Durand (mouse-brain) are the only ones that can be considered for the preparation of vaccine intended for Europeans, since their products can be titrated and are less likely than their predecessors to induce serious vaccinal reactions. Both are capable of furnishing the quantities necessary for mass vaccination.

Although it seems that the time required for setting in motion the production of flea virus must be somewhat longer, this method should supply enormous quantities of a vaccine that may be easily preserved, remains active over a long period, and is particularly easy to employ.

Both types of vaccine seem to have given equally favourable results in suppressing epidemics in North Africa.

In short, we may conclude that the judicious use of the modern methods of vaccination at our disposal, concurrently with delousing, affords an effective protection against the epidemic menace that typhus fever now constitutes for Europe.

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CONTRIBUTION TO THE STUDY OF METABOLISM OF VITAMIN C AND ITS ELIMINATION IN THE URINE

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As early as the spring of 1936, as a complement to our surveys on the physical development and general state of health of children, we had undertaken a series of researches on the elimination of vitamin C in the urine, using the method of GANDER and NIEDERBERGER, slightly modified to permit of the titration in series of the urine of a large number of children.

The process was as follows:

> To 10 c.c. of urine in a graduated beaker, we added about 10 c.c. of water[1] and 1 c.c. of glacial acetic acid. For the titration of vitamin C, dichlorophenol-indophenol was used as a reagent. A freshly made solution of this reagent (2 tablets of 0·002 g. in 100 c.c. of water) was placed in the burette with which the titration was carried out. All that was then necessary was either to note the number of cubic millimetres of reagent used until coloration appeared, and to multiply this figure by 2 in order to find the quantity of vitamin C in mg. per 100 c.c. of urine, or, alternatively, to count the number of drops of the dichlorophenol-indophenol solution necessary to obtain coloration, which gives directly the quantity of vitamin C expressed in mg. per 100 c.c. of urine.

We have already published the results of the first series of our research[2] bearing on 7,464 titrations, which enabled us to establish that the mean content in vitamin C of the urine of Lausanne children was only 1·4 mg. per 100 c.c., whereas we had found an average of 1·55 mg. among 250 healthy adults.

Observations on children in four schools where the pupils came from families of different economic levels showed us that there existed a correlation between the quantity of vitamin C contained in their urine and their state of health. We not only checked the vitamin C found in the urine of children but, at the same time, compared over the period of a year the proportion of absences from school due to illness.

We also found that vitamin C deficiency was more marked during winter months, from December to March, and that there was an inverse and constant correlation between the incidence of infectious diseases and the

[1] It is not necessary to add water when the urine is pale, but 5 to 10 c.c. must be added when the urine is strongly coloured, in order to observe more easily the apparition of the red colour (temporary formation of a carmine-red disc).

[2] (*a*) MESSERLI, F.-M., and HEIMANN, F.: "Recherches sur la fréquence de l'hypovitaminose C chez les enfants de Lausanne." *Rev. Hyg.*, **60**, No. 1, January 1938. (*b*) MESSERLI, F.-M.: "Hygiène alimentaire—Recherches sur la fréquence des carences alimentaires chez les enfants et la population de Lausanne." *Schweiz. Med. Wschr.*, **69**, Nos. 38 and 39, 1939. (*c*) MESSERLI, F.-M.: "Recherches sur la fréquence des carences en vitamines." *Bull. Soc. vaud. Sci. nat.*, **61**, No. 253, 1940.

quantity of vitamin C eliminated; in other words, that a low vitamin C content corresponded to a high incidence of communicable disease. A fall in the vitamin C content of the urine was also observed during the incubation period of these diseases.

Schools	Vitamin C in mg. per 100 c.c. of urine	Proportion of children absent in the course of the year through illness	Proportion of children with no absences from school on account of illness
Bellevaux and Barre (poor districts) ..	1·37	94%	6%
Villamont-dessus and Ouchy (prosperous districts)	1·59	71%	29%

Finally, we made a number of studies on vitamin C saturation, giving the children *Redoxon*[1] and *Nestrovit.*

As a consequence of these researches, we proposed to prevent vitamin C insufficiency in children by giving them during the winter, from December to March, when fruit and vegetables are scarce, synthetic vitamin C in the form of *Redoxon* or some similar product.

We also considered it desirable to continue our studies on the following points:

(1) Determination of the mean rate of elimination among Lausanne children in 1940 and 1941;
(2) Influence of the body position on this rate;
(3) Action of exposure to sun on this rate;
(4) Rhythm of elimination of vitamin C in the course of saturation tests;
(5) Effect of preventive distribution of vitamin C on the incidence of communicable diseases;
(6) Finally we endeavoured, in the course of these studies, and particularly during saturation tests, to ascertain whether the administration of high doses of vitamin C would provoke the appearance of albuminuria in the children tested, or increase among them a pre-existing albuminuria.

1. Mean Rate of Elimination of Vitamin C in the Urine of Lausanne Children

As in 1936 and 1939, we carried out analyses in three groups of children, respectively drawn from different social strata; medium, poor and very poor.

The above table shows that the rate of excretion of vitamin C in the urine tallies exactly with the social status of the children and with their state of health. If to these figures are added the 7,464 analyses made between 1936 and 1939, we have a total of 16,329 analyses giving a mean rate of 1·4 mg. per 100 c.c. of urine.

[1] *Redoxon* is pure synthetic ascorbic acid, prepared by the firm of Hoffmann-La Roche & Cie. *Nestrovit*, a product of the same firm, is a complex of vitamins standardised at the following doses for each tablet: Vitamin A, 6,500 I.U.; B_1, 65 I.U.; C, 200 I.U.; D, 650 I.U. The quantities of *Redoxon* and dichlorophenol-indophenol required for the study and titrations recorded in the present paper were kindly supplied by F. Hoffmann-La Roche & Cie.

	1940		1941	
	Number of analyses	Mean rate in mg.	Number of analyses	Mean rate in mg.
(*a*) Children of medium social level (Oeuvre de Vidy-Plage) Boys ..	910	1·45	360	1·43
Girls ..	321	1·27	202	1·62
Children ..	1,231	1·40	562	1·50
(*b*) Children of poor families. Elementary schools (Prélaz School) ..	2,958	1·16	2,210	1·20
(*c*) Very poor children (orphanage) ..	1,368	0·7	536	1·10
	5,557	1·10	3,308	1·26

We had in 1936 and 1937 computed the quantity of vitamin C excreted by 4,233 Lausanne children distributed according to age; for purposes of comparison, we made a similar computation for the Vidy-Plage children in 1940 and 1941.

	Age (years)							
	7	8	9	10	11	12	13	14
1936 Girls ..	1·24	1·25	1·26	1·84	1·29	1·77	1·68	1·49
Boys ..	1·35	1·60	1·85	2·08	2·05	1·68	1·50	
1937 Girls ..	1·63	1·34	1·24	1·55	1·38	1·34	1·36	
Boys ..	1·69	1·11	1·54	1·88	1·36	1·09	2·05	
1940 Girls ..	1·34	1·33	1·30	1·65	1·67	2·26	1·44	1·65
Boys ..	1·11	1·16	1·20	1·40	1·10	1·20	1·25	
1941 Girls ..	1·50	1·58	1·80	1·35	1·98	1·55	1·62	
Boys ..	1·43	1·52	1·54	1·33	1·71	1·44	1·65	

It is difficult to draw conclusions from these figures; it seems, however, that the deficiency is greater between 7 and 9 years than above 10.

2. Influence of the Body Position on the Rate of Elimination of Vitamin C

We made three series of observations, placing 43, 27 and 31 children successively in various positions and analysing their urine every half-hour.

Samples were taken and titrated on their arrival (I), then each half-hour after the children had been placed in a lordotic standing position (''standing rigidly at attention'') (II), after they had been allowed to play freely (III), after they had

*

been kept lying on their backs (IV), or lying face downwards (V), and after letting them walk and run freely (VI).

Averages obtained from	I	II	III	IV	V	VI
43 analyses	1·3	0·9	1·0	0·9	1·0	0·9
27 analyses	0·7	0·9	0·8	0·8	0·9	0·8
31 analyses	0·9	1·0	0·9	0·7	0·8	0·9

From these figures it may be concluded that the position of the children has no influence on the rate of elimination of vitamin C.

3. Influence of Exposure to Sun

We checked the rate of vitamin C excretion at the beginning of the sun-cure—that is, before pigmentation occurred, and also during and after the cure. We also repeated titrations several times in the course of an afternoon, when the children were subjected to the action of the sun. All these analyses showed that the rate of elimination of vitamin C was not influenced by exposure to the sun.

4. Saturation Tests

We ascertained the rate of elimination of vitamin C in the urine of children after administration of various doses of *Redoxon*.

In the first test, we gave to three groups of girls 1, 2 or 3 tablets respectively of 0·05 g. of *Redoxon*, while to three control children none was given. Analysis of their urine was made at 2.30 p.m. (i.e. just before *Redoxon* was taken) and at 4.30 p.m. (i.e. 2 hours afterwards).

August 16th, 1940

		Age (years)	2.30 p.m.	Ingestion of *Redoxon*	4.30 p.m.
Girls	H. A. ..	9	1·6	0 tablet	0·8
	H. L. ..	8	1·6	0 tablet	0·8
	F. M. ..	6	1·2	0 tablet	1·0
			Average 1·4	0 tablet	0·9
	F. G. ..	11	1·0	1 tablet	2·2
	B. D. ..	10	0·6	1 tablet	2·0
	B. D. ..	8	1·2	1 tablet	2·4
			Average 0·9	1 tablet	2·3
	A. RM. ..	10	0·6	2 tablets	0·8
	A. Y. ..	9	0·6	2 tablets	1·2
	L. G. ..	10	0·8	2 tablets	0·8
			Average 0·65	2 tablets	0·93
	E. A. ..	7	0·6	3 tablets	0·6
	V. M. ..	6	0·6	3 tablets	0·6
	M. T. ..	9	0·8	3 tablets	1·8
			Average 0·65	3 tablets	1·0

The second analysis showed a marked increase in the vitamin C content of the urine, this increase being even greater in the girls who had received only one tablet that in those who had received two or three. This experiment could not be considered as conclusive, in view of the small number of observations. We therefore gave to five groups of six children each, 1, 2, 3, 4 and 5 tablets of 0·05 g. of *Redoxon* daily for five days; the children were observed during three days before taking the drug and again for four days afterwards. Nine other children served as controls throughout the period of the experiment. For various reasons, bad weather or absence from town, some children were sometimes absent. The general table of the analyses (Table I) gives, however, useful indications on vitamin C elimination. Each day the first sample was taken at 2.30 p.m., before *Redoxon* was given, the second two hours later, at 4.30 p.m.

It appears from this table that, during the first days of observation, before *Redoxon* was taken, the quantity of vitamin C found in the urine was practically the same in all groups, and did not show much day-to-day variation. The urine of the children who did not receive *Redoxon* continued to show the same amount, while there was a marked increase among the children who took the drug. It is noteworthy that the increase was similar among all children taking the drug, whether they took 1, 2, 3, 4 or 5 tablets. This shows that, at the beginning of the experiment, they were in a state of vitamin balance. Among all these children there was a marked increase of vitamin C excretion in the urine after two hours. The first day, the mean rose from 1·0 to 2·47, the second day from 1·63 to 2·75, the third day from 1·8 to 4·5, the fourth day from 1·88 to 3·95, the fifth day from 2·4 to 4·22. After this sharp increase, the vitamin content of the urine gradually fell back to the levels prevailing before the experiment, both among the children receiving several tablets and among those who received only one. This proves that, to be efficacious, *Redoxon* administration must not be stopped after a few days, but must be continued for a longer period.

These observations were supplemented by the determination of the excretion rates of vitamin C among 32 children from the Lausanne orphanage; these children came from very poor families. Samples were taken, for six days in succession, every half-hour from 8 to 11.30 a.m.

> The amount of urine excreted, the amount of vitamin C eliminated per 100 c.c. of urine and the presence of albumin, if any, were noted. The first sample was taken before the children had eaten at 8 o'clock. The children drank 100 c.c. of water at 8, 8.30, 9, 9.30 (the first day, however, they drank 100 c.c. of water every half-hour from 8 to 11). After three days' observation (Monday to Wednesday) *Redoxon* was given on two days, Thursday and Friday, at 8 in the morning, in doses of 0·05 g. tablets to 7 children, 2 tablets to 7 others, and 3, 4 or 5 tablets to three further groups of 6 children each. On the sixth day, *Redoxon* was not given and the urine vitamin C excretion was again determined under the same conditions as on the second and third days. On the fifth day, a further determination of excretion was carried out at 2.30 and 4.30 p.m.

The analyses showed that the mean rate of vitamin C excretion by these children was very low (0·70 mg.), which corresponded to vitamin insuffi-

Table I

Determination of Vitamin C Saturation

Date 1940		31st July		2nd August		3rd August		5th August		6th August		7th August		8th August		9th August		12th August		13th August		14th August		15th August	
Time, p.m.		2.30	4.30	2.30	4.30	2.30	4.30	2.30	4.30	2.30	4.30	2.30	4.30	2.30	4.30	2.30	4.30	2.30	4.30	2.30	4.30	2.30	4.30	2.30	4.30
Group of 6 children receiving *Redoxon* from 5th to 9th August, 1940	0·05 g.	1·43	1·30	1·23	1·26	1·12	1·44	1·16	2·22	1·96	2·93	2·20	2·90	1·52	4·16	2·53	3·13	1·80	1·66	1·55	1·55	1·00	1·30	1·00	1·50
	0·10 g.	1·06	1·23	1·13	1·33	1·06	1·10	1·10	2·43	1·40	2·04	1·12	4·16	1·36	3·23	2·33	4·90	1·60	1·43	1·40	1·20	1·24	1·12	1·28	0·76
	0·15 g.	1·03	1·06	1·26	1·03	1·00	0·97	0·86	3·16	2·00	3·06	2·52	8·28	2·08	2·68	1·68	3·53	0·73	1·22	1·04	1·00	1·15	1·60	0·96	0·93
	0·20 g.	1·00	0·93	0·90	0·96	0·90	0·86	0·80	2·10	1·90	3·06	1·24	3·44	2·66	5·20	2·00	3·00	1·00	1·12	0·80	1·08	1·07	0·93	0·80	1·04
	0·25 g.	0·90	0·96	1·18	0·93	1·13	0·90	0·83	2·44	1·00	2·63	1·88	4·08	1·76	4·36	1·50	6·55	1·60	1·10	0·85	1·05	1·00	0·95	1·15	0·85
Average of groups II and IV		1·07	1·10	1·14	1·10	1·04	1·05	1·00	2·47	1·63	2·75	1·80	4·50	1·88	3·95	2·40	4·22	1·55	1·50	1·15	1·18	1·03	1·26	1·04	1·04
Average of 9 control children receiving no *Redoxon*		1·15	1·35	1·20	1·15	1·22	1·04	1·00	0·90	1·08	1·84	1·50	1·94	1·42	1·62	0·84	1·20	1·35	1·10	0·76	0·83	1·20	0·96	0·66	0·93

ciency. Indeed, by giving on the fourth day 0·05 g. of *Redoxon* to the first group of seven children, no immediate increase of elimination took place that day; an increase of excretion was observed, however, in the other groups—very small among the children who received 0·10 g. of the drug (2 tablets) and greater among those who received from 0·15 g. to 0·25 g. of *Redoxon*. The elimination of vitamin C increased as early as half an hour after the ingestion of *Redoxon*; it reached its peak two hours afterwards. The same experiment was repeated on the fifth day; at 8 o'clock the sample taken of the night urine showed in all groups a higher vitamin content than had been found on the preceding days at the same time. This resulted from the absorption of *Redoxon* the day before. A further and marked increase of the amount was found two hours later. On the sixth day, when no more *Redoxon* was given, the first samples, taken at 8 o'clock, still showed a high vitamin content; there was later a rapid return to the average observed during the first days, before *Redoxon* was administered.

Thus, among these children showing vitamin C insufficiency, the effect of *Redoxon* administration was only temporary.

We conclude from these observations that, in cases of vitamin C insufficiency, high doses of *Redoxon* (at least 4 tablets of 0·05 g.) must be given at once and administration continued for some time.

The observations made prove, moreover, that *Redoxon* has no influence on the quantity of urine excreted.

5. Preventive Distribution of Vitamin C and its Effect on the Incidence of Illness

A small dose of *Redoxon*—i.e. 0·05 g. of ascorbic acid—was given daily for a month, as a preventive, to Lausanne school children. The state of health of these children was compared with that of an identical group of controls. By agreement with Dr. Wintsch, school physician, four classes of children (two of boys, two of girls) from 11 to 13 years of age were selected in the Prélaz school, which serves poor districts of Lausanne. The teachers of these classes divided the children into two groups, who respectively received or did not receive *Redoxon*.

> Thirty-six boys received a daily dose of 0·05 g. of *Redoxon* from Monday to Friday of each week, between February 25th and March 24th, 1941, while 35 boys received none.
>
> Similarly, 42 girls received *Redoxon*, while 41 had none.

The mean quantities of vitamin C found in the urine of these four groups of children are given in Table III.

The mean vitamin C content in the urine of the boys before taking *Redoxon* was 1·16 mg.; it remained unaltered among the 35 boys who took none, while, among those who took the drug, it rose successively to 1·30, 1·27, 2·03, 2·16 and 1·87 in the following weeks (with an average for the period of 1·73). In this class of 36 pupils, 16 absences of one half-day on account of illness were recorded during the month of March, while there were 27 such absences among the 35 boys not taking *Redoxon*.

Table II

Groups of Children who Received *Redoxon* on August 21st and 22nd

U. = Average quantity urine excreted.
Vit. = Average quantity Vitamin C, mg. per 100 c.c. of urine.

	Monday, August 19th, 1940									Tuesday, August 20th, 1940								
Time, a.m.	8	8.30	9	9.30	10	10.30	11	11.30	Aver.	8	8.30	9	9.30	10	10.30	11	11.30	Aver.
Water drunk in c.c. ..	100	100	100	100	100	100	100			100	100	100	100					
Ist group, 7 children .. U.	67·9	59·3	89·3	133·3	117·9	67·1	78·6	78·6	*86·5*	97·8	24·3	79·3	214·3	179·3	110·7	33·6	17·1	*95·8*
0·05 g. of *Redoxon* .. Vit.	0·65	0·62	0·60	0·46	0·42	0·48	0·60	0·57	**0·55**	0·65	0·63	0·54	0·40	0·43	0·51	0·50	0·80	**0·55**
IInd group, 7 children: .. U.	117·9	54·3	78·6	102·1	84·3	67·1	86·4	94·3	*83·1*	103	30	109	200	129	124·3	30·2	15·7	*92·9*
0·10 g. of *Redoxon* .. Vit.	0·65	0·65	0·57	0·51	0·48	0·51	0·45	0·45	**0·53**	0·68	0·65	0·57	0·45	0·51	0·57	0·53	0·73	**0·57**
IIIrd group, 6 children: .. U.	70	63·3	38·3	113·3	108·3	65	60·8	85	*75·5*	78·5	27·8	80·8	106·7	13·8	98·5	17	10·2	*60·9*
0·15 g. of *Redoxon* .. Vit.	0·60	0·66	0·66	0·43	0·43	0·50	0·56	0·46	**0·53**	0·57	0·60	0·50	0·50	0·40	0·50	0·63	0·80	**0·53**
IVth group, 6 children: .. U.	85·8	48·3	85·8	134·2	109·2	80	111·7	85	*92·5*	118	74·3	128·6	180·7	133·5	95·7	23	17·1	*96·3*
0·20 g. of *Redoxon* .. Vit.	0·73	0·66	0·66	0·43	0·50	0·50	0·50	0·50	**0·56**	0·68	0·77	0·51	0·60	0·43	0·51	0·60	0·83	**0·62**
Vth group, 7 children: .. U.	59·2	31·7	39·2	90	131·7	80·9	97·5	83·3	*76·6*	61·4	13	48	137·1	115	60·7	12·1	10·7	*57·2*
0·25 g. of *Redoxon* .. Vit	0·73	0·83	0·66	0·50	0·46	0·46	0·46	0·50	**0·57**	0·50	0·51	0·40	0·32	0·32	0·40	0·56	0·57	**0·44**
Average quantity urine excreted..	80·1	51·3	66·7	114·5	110·2	61·2	87	85·2	*82·8*	91·7	33·8	89·1	178·5	114·1	97·9	23·1	12·1	*80·6*
Average quantity Vit. C., mg. ..	0·67	0·62	0·63	0·46	0·45	0·49	0·51	0·49	**0·54**	0·61	0·63	0·50	0·45	0·42	0·49	0·56	0·74	**0·54**

	Wednesday, August 21st, 1940									Thursday, August 22nd, 1940								
Time, a.m.	8	8.30	9	9.30	10	10.30	11	11.30	Aver.	8	8.30	9	9.30	10	10.30	11	11.30	Aver.
Water drunk, in c.c. ..	100	100	100	100						100	100	100	100					
Ist group, 7 children: .. U.	170·7	95·7	150	139·3	100·7	48·5	18·6	22	*93·2*	259	40	132	132	106	81	39	41	*103·7*
0·05 g. of *Redoxon* .. Vit.	0·68	0·83	0·57	0·54	0·54	0·60	0·65	0·68	**0·63**	0·77	0·77	0·84	1·0	1·0	0·80	0·77	0·72	**0·83**
IInd group, 7 children: .. U.	127·8	36·4	110·7	183	144·3	118·5	47·8	26·1	*99·3*	75	33	92	131	152	76	41	42	*80·3*
0·10 g. of *Redoxon* .. Vit.	0·60	0·64	0·48	0·47	0·43	0·41	0·55	0·68	**0·61**	0·71	0·94	0·94	1·01	1·14	0·94	0·84	1·02	**0·95**
IIIrd group, 6 children: .. U.	120	43·3	83	172	124·3	142	41·3	5·6	*90·1*	76	1·1	1·03	1·35	1·83	1·28	1·2	1·93	*1·21*

IVth group, 6 children: .. U.	130·7	52·1	137·1	172·8	135	73·5	54·3	30·5	*98·2*	138	49	123	267	138	74	53	32	*109·3*
0·20 g. of *Redoxon* .. Vit.	0·80	0·68	0·60	0·55	0·47	0·48	0·68	0·67	**0·61**	0·83	1·26	1·06	1·16	1·96	1·83	2·06	2·16	**1·54**
Vth group, 7 children: .. U.	138	31	106	179	181	70	42	21	*96*	174	83	105	207	162	84	41	34	*111·2*
0·25 g. of *Redoxon* .. Vit.	0·76	0·72	0·48	0·46	0·52	0·60	0·72	0·68	**0·62**	0·93	1·08	1·03	1·03	2·16	1·66	1·35	1·86	**1·39**
Average quantity urine excreted..	137·4	51·7	117·7	169·8	144·5	85·1	40·8	24·8	*96·5*	153·2	50·6	109·8	186·4	142·6	81	37·8	34·4	*99·5*
Average quantity Vit. C., mg. ..	0·71	0·71	0·55	0·51	0·49	0·53	0·63	0·67	**0·62**	0·80	1·03	0·98	1·11	1·62	1·30	1·24	1·55	**1·2**

	Friday, August 23rd, 1940											Saturday, August 24th, 1940								
Time, a.m.	8	8.30	9	9.30	10	10.30	11	11.30	Aver.	2.30 p.m.	4.30 p.m.	8	8.30	9	9.30	10	10.30	11	11.30	Aver.
Water drunk, in c.c. ..	100	100	100	100								100	100	100	100					
Ist group, 7 children: .. U.	235·5	53·5	82·8	18·3	113	92·1	14·3	20	*78·7*			182·1	54·3	90·8	102·9	87·8	50	21·4	9·07	*119·7*
0·05 g. of *Redoxon* .. Vit.	1·32	0·88	0·77	0·77	0·88	1·26	1·86	2·11	**1·23**	1·62	1·88	1·37	1·08	0·54	0·64	0·52	0·48	0·45	0·52	**0·63**
IInd group, 7 children: .. U.	73·2	42·8	98	165·7	176·4	128·5	42·8	26·4	*94·2*			79·4	30·7	121·4	109·2	112·8	67·1	87·8	23·4	*126·3*
0·10 g. of *Redoxon* .. Vit.	1·44	0·84	0·80	1·0	1·4	1·8	1·82	2·11	**1·40**	1·84	1·80	0·97	0·78	0·64	0·64	0·45	0·55	0·50	0·54	**0·57**
IIIrd group, 6 children: .. U.	121	81·6	108·3	180	120	141	12·5	11·6	*97*			80·8	16·8	93·3	123·3	133·3	62·5	26·6	6·3	*108·6*
0·15 g. of *Redoxon* .. Vit.	1·83	1·06	0·96	1·2	2·13	1·96	2·7	2·83	**1·83**	2·33	2·03	0·86	0·63	0·54	0·54	0·43	0·45	0·51	0·66	**0·58**
IVth group, 6 children: .. U.	122·5	43	109	210	181·6	94	11	19	*98·6*			122·5	22·6	121·6	135	109·1	73·3	27·5	31·7	*128·8*
0·20 g. of *Redoxon* .. Vit.	1·2	0·9	1·1	1·0	2·2	2·7	2·9	2·5	**1·81**	3·1	3·1	1·43	1·03	0·60	0·57	0·50	0·48	0·45	0·55	**0·70**
Vth group, 7 children: .. U.	175	47	69	184	145	93	31	31	*96·9*			253·5	63·3	103·3	151·6	155	68·3	25·8	7·66	*165·7*
0·25 g. of *Redoxon* .. Vit.	1·1	0·9	1·03	1·06	1·9	2·5	2·7	2·5	**1·71**	2·3	2·5	1·46	1·0	0·8	0·78	0·55	0·43	0·5	0·54	**0·76**
Average quantity urine excreted..	145·4	53·6	93·4	151·6	147·2	109·7	22·3	21·6	*93*			143·6	37·5	106	124·4	119·6	64·2	37·8	15·6	*129·8*
Average quantity Vit. C., mg. ..	1·37	0·92	0·93	1·00	1·70	2·04	2·39	2·41	**1·59**	2·24	2·26	1·22	0·90	0·62	0·63	0·49	0·48	0·48	0·57	**0·65**

Table III

PREVENTIVE TREATMENT OF PUPILS BY ADMINISTRATION OF A 0·05 g. DOSE OF "REDOXON" from FEBRUARY 25TH to MARCH 24TH, 1941

Date	GROUP I 35 boys without *Redoxon*		GROUP II 36 boys receiving *Redoxon* from February 24th		GROUP III 42 girls without *Redoxon*		GROUP IV 41 girls receiving *Redoxon* from February 24th	
Time	8 a.m.	2 p.m.	8 a.m.	2 p.m.	8 a.m.	2 p.m.	8 a.m.	2 p.m.
Thursday, February 20th ..	1·15	1·28	1·15	1·18	1·04	1·00	1·02	0·94
Friday, February 21st ..	1·30	1·12	1·18	1·13	1·21	0·91	1·29	0·88
Monday, February 24th ..	1·26	1·17	1·13	1·09	0·92	0·97	0·98	0·98
Weekly average	1·21		1·16		1·01		1·02	
Tuesday, February 25th ..	1·12	1·03	1·19	1·26	0·95	0·96	1·24	1·31
Wednesday, February 26th ..	1·06	1·21	1·20	1·47	1·09	1·04	1·26	1·43
Thursday, February 27th ..	1·08	1·10	1·26	1·46	0·99	1·08	1·43	1·41
Friday, February 28th ..	1·10	1·05	1·18	1·39	1·02	1·08	1·34	1·49
Weekly average	1·13		1·30		1·03		1·38	
Monday, March 3rd ..	0·95	1·06	0·91	1·32	0·95	1·01	1·00	1·23
Wednesday, March 5th ..	1·04	0·99	1·23	1·16	0·92	0·95	1·04	1·16
Friday, March 7th	0·90	1·19	1·05	1·93	1·21	1·20	1·36	1·83
Weekly average	1·02		1·27		1·04		1·27	
Monday, March 10th ..	1·49	1·24	2·07	1·86	1·10	1·01	1·96	1·71
Wednesday, March 12th ..	1·06	1·34	1·40	2·62	1·56	1·28	2·26	3·13
Friday, March 14th	1·57	1·46	2·36	1·84	1·38	1·34	2·17	2·37
Weekly average	1·36		2·03		1·28		2·26	
Monday, March 17th ..	1·35	1·73	1·45	2·25	1·56	1·32	1·94	1·76
Wednesday, March 19th ..	1·72	1·68	2·39	2·55	1·84	1·62	2·42	2·13
Friday, March 21st	1·46	—	2·15	—	1·69	1·40	2·39	2·56
Weekly average	1·59		2·16		1·57		2·21	
Monday, March 24th ..	1·47	1·60	1·57	2·17	1·36	1·48	2·19	2·45
Average	1·53		1·87		1·42		2·32	
Average	**1·16**		**1·73**		**1·22**		**1·88**	

Among the girls of the control group, the mean quantity of vitamin C excreted during the observation month was 1·22; among the others the quantity was 1·02 before taking *Redoxon*, and it rose during treatment to 1·38, 1·27, 2·26, 2·21 and 2·32, with a general mean for the period of treatment of 1·88. During March, there were 13 absences of half a day on account of illness among the girls who took *Redoxon*, as against 29 amongst the others.

These data may be tabulated as follows:

Groups	*Redoxon*	Excretion of Vitamin C		Half-days of school attendance in March	
		Before the experiment	During the experiment	Possible	Missed on account of illness
I 35 boys	0	1·21	1·16*	1,470	27 = 1·84%
II 36 boys	+	1·16	1·73†	1,512	16 = 1·06%
III 42 girls	0	1·01	1·22*	1,764	29 = 1·64%
IV 41 girls	+	1·02	1·88†	1,722	13 = 0·76%
I + III 77 children ..	0	1·11	1·19*	3,234	56 = 1·73%
II + IV 77 children ..	+	1·09	1·81†	3,234	29 = 0·90%

* Average from February 20th to March 24th.
† Average from February 25th to March 24th.

Thus, among children who for a month, from February 25th to March 24th, received *Redoxon* at a dose of 0·05 g. daily for five days a week (a total of 21 tablets of 0·05 g. or 1·05 g. of *Redoxon*) school absenteeism on account of illness was in March only half of what it was in the case of the others (29 absences against 56).

The teachers themselves noticed improvement in the health of the children who had received *Redoxon*; they had purposely selected for *Redoxon* treatment the weaker children—i.e. those who were most often absent from school on account of illness. This selection enhances the value of the results obtained.

These observations, though they cannot be considered as final, are important. They confirm our previous findings regarding the relation between school absenteeism due to illness and vitamin C insufficiency.

We intend to resume our studies next winter by giving *Redoxon* or a similar product to a number of children for several months, if possible from December to March, and comparing them, from the health point of view, with a similar group of children not receiving ascorbic acid.

6. Ingestion of Ascorbic Acid and Albuminuria

In the course of all the saturation tests described above, while we were giving daily 1 to 5 tablets of 0·05 g. of *Redoxon* over shorter or longer periods, we attempted to ascertain whether this ingestion had produced albuminuria or increased that condition where present. The answer on both scores was negative.

Conclusions

The following conclusions may be drawn from these observations:

1. In conformity with our previous findings, the rate of expulsion of vitamin C in the urine of Lausanne children was, on an average, about 1·4 to 1·5 mg. in 1940–1941. This amount varied according to feeding and to the social conditions of the children.

2. The body position, rest or exercise and exposure to sun, seemed to have no influence on this excretion.

3. In saturation tests, in cases of vitamin balance, ingestion of ascorbic acid is followed very quickly—within from half-an-hour to two hours—by a marked increase of excretion of this vitamin in the urine; this increase is not observed immediately in cases of vitamin insufficiency. This test might be used for the control of vitamin C sufficiency. For this purpose, the urine should be titrated three or four times, every half-hour after the ingestion of 0·05 g. of ascorbic acid. A rise in vitamin excretion would signify vitamin sufficiency; the absence of rise, vitamin insufficiency.

4. Children who for a month received a daily preventive dose of 0·05 g. of ascorbic acid, and whose vitamin excretion in the urine had greatly increased in consequence, were absent from school on account of illness only half as frequently as their schoolfellows who did not receive vitamin C.

5. Vitamin C saturation tests did not reveal any increase in albuminuria.

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INTERNATIONAL BIOLOGICAL STANDARDS

SOME RECENT CHANGES RELATING TO THE INTERNATIONAL STANDARDS FOR CERTAIN OF THE SEX HORMONES AND FOR PITUITARY (POSTERIOR LOBE)

(From the Department of Biological Standards, The National Institute for Medical Research, Hampstead, London.)

ON account of the stocks of the original preparations which had served as International Standards for some of the Sex Hormones and for Pituitary (posterior lobe) having become exhausted, it has been necessary to replace these by other samples in order that the supply of these standards, undertaken by the Medical Research Council on behalf of the Health Organisation of the League of Nations, to laboratories, institutes and research workers throughout the world, shall be maintained.

The changes which have been introduced, the circumstances which led to them, the sources of the materials used for these Second, or replacement, International Standards, the chemical and physical properties and constants of those which are pure chemical substances, the results of the examination of their biological properties and, in the case of some of them, their biological assay are described in the Memoranda which are printed below.

In the case of each replacement every care has been taken and all practicable tests and examinations have been carried out at Hampstead, and when practicable in collaboration with experts in other countries, to ensure that these Second International Standards conform to the requirements of the original standards as defined and recommended for adoption by the Permanent Commission on Biological Standardisation.

In the case of the International Standards for the Oestrus-producing Hormone, the Male Hormone (Androsterone) and the Progestational Hormone of the corpus luteum (Progesterone) the standard preparation is a pure chemical substance; in the case of each of these the physical and chemical constants have been determined and have been found to conform to the requirements of the original definition of the standard material, and are not significantly different in any particular from those determined for the material which constituted the First International Standard. In the case of these three

hormones, the original standards for which are now being replaced by new preparations, there is every reason to believe that the value of the International Unit is being maintained.

The replacement of the original standard material by new preparations has necessitated certain changes in the Memoranda originally prepared for the information of recipients of the First International Standards. In the case of the three Memoranda for the Sex Hormones the changes are small; they comprise a statement as to the source of the material and their physical and chemical properties and constants. Some small additions have been made to the section relating to the conduct of biological tests; these are based upon information gained as a result of further experience of the comparative tests applied for the determination of potency, obtained since the First International Standard was established and adopted, and may be of interest or service. Since the original Memoranda relating to these three standards were not published in the *Bulletin*, it is considered that the best procedure is to publish the revised Memoranda for the Second International Standards for the Oestrus-producing Hormone, the Male Hormone, and the Progestational Hormone of the corpus luteum, as these now supersede the original documents.

It has also been necessary to make a small addition to the Memorandum relating to the International Standard for the Lactogenic Substance of the Anterior Lobe of the Pituitary Gland (Prolactin). It is suggested that this addition, printed below, be added as Appendix I to the original Memorandum which was published as Extract 20, pp. 909–912, Vol. VIII of the *Bulletin of the Health Organisation of the League of Nations* (1939).

Following the recommendation of the Third International Conference on the Standardisation of Sex Hormones, held at Geneva in August, 1938, the Department of Biological Standards at the National Institute for Medical Research, Hampstead, has acquired 4·5 kilograms of dried anterior pituitary lobe substance of oxen, prepared in accordance with the recommendation of the Conference, and this is now available, suitably dispensed, for the use of those requiring it for the investigation of those hormones of the anterior lobe of the pituitary gland for which international standard preparations have not yet been adopted. This international preparation is now available and will be supplied on application. A Memorandum relating to it has been prepared and is printed below.

The replacement of the First International Standard Preparation for Pituitary (posterior lobe), the stocks of which were approaching exhaustion in 1939, has been a more difficult and complicated problem. The steps which have been taken and the decisions which have been reached, the reasons for these decisions and the circumstances under which they had to be taken, are explained in the Memorandum on the Second International Standard Preparation for Pituitary (Posterior Lobe) which is printed below. It will be seen that a difficult situation was created when it became necessary to replace the First International Standard by a new preparation, and as it was impracticable to convene a meeting of the Permanent Commission on Biological Standardisation for the discussion of the problems involved and for a decision

to be made as to the action to be taken, the National Institute for Medical Research, acting in agreement with all the opinions recorded by those who co-operated in the relevant international investigations outlined in the Memorandum, has taken the steps indicated in order that the essential service of supply of this important international standard shall be maintained.

M.45

MEMORANDUM ON THE REPLACEMENT OF THE SUBSTANCE OF THE INTERNATIONAL STANDARD FOR THE OESTRUS-PRODUCING-HORMONE

Prepared by

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At an International Conference held in London in July, 1932, in connection with the Permanent Commission on Biological Standardisation of the Health Organisation of the League of Nations, it was decided to adopt as the International Standard for the Oestrus-producing Hormone the hydroxy-ketonic form of the hormone normally obtained from the urine of pregnancy. The National Institute for Medical Research was to act as the Central Laboratory on behalf of the Health Organisation of the League of Nations, to keep the standard, and to distribute it in suitable quantities to other national centres.

The standard was duly established in 1933 according to the directions of the Conference, a total of 20·9 g. being contributed by four research workers. During 1938 the supply of material began to run low, and it was decided that the standard had been so useful that arrangements should be made for its renewal when the original material became exhausted.

Preparation of the New International Standard in 1939

An approach to manufacturers known to be producing oestrone was met most generously and a contribution of 5 g. of crystalline oestrone was made towards the renewal of the standard by each of the following firms:

Bayer	Elberfeld, Germany
Boots Pure Drug Co., Ltd.	Nottingham, England
British Drug Houses	London, England
Ciba	Basle, Switzerland
Løvens Kemiske Fabrik	Copenhagen, Denmark
N. V. Organon	Oss, Holland
Parke, Davis & Co.	Detroit, Michigan, U.S.A.
Laboratoires du Dr. Roussel	Paris, France
Schering-Kahlbaum	Berlin, Germany

whose generous assistance is hereby gratefully acknowledged.

The total weight of the material removed from the nine containers was 45·07 g. and the separate contributions were mixed by recrystallisation as follows:

> The whole material was placed in a Soxhlet apparatus, and the containers washed out with alcohol. Extraction was carried out with a total of about 150 c.c. of aldehyde-free alcohol. When extraction was complete the oestrone separated out in large crystals with a slight bluish tinge, which weighed, after drying, 43·9 g. The crystals were ground to a fine powder, weight 43·6 g., and samples were removed for physical examination, leaving a weight of 43·57 g. of the air-dry preparation.

Comparison of the Materials Constituting the New and Original Standards

The following are the results of the determination of the physical constants indicated, of the new and original standard preparations, carried out by the same method.

	First (original) International Standard	Second (Replacement) International Standard
Melting Point (Kofler micro M.P. apparatus)	258°–259°	257°–260°
Optical Rotation $[\alpha]_D^{25}$	+ 169°	+ 168°
0·432 per cent in absolute alcohol $[\alpha]_{5,461}^{25}$ ..	+ 206°	+ 206°
Ultra-violet absorption $E_{1cm.}^{1\%}$—4 dm. tube ..	77·3 at 2,820 Å	76·8 at 2,820 Å

The two preparations therefore resembled each other so closely in physical properties that the new standard may be considered as identical with the original one.

Distribution of the Standard into Small Ampoules

The whole of the material provided for the Second International Standard (43 g.) was distributed, in approximately 30 mg. quantities, into small dry tubes. The tubes were placed *in vacuo* over phosphorus pentoxide for some weeks, sample tubes being weighed at intervals until an assurance was obtained that the weight was constant and accordingly the standard material was completely dry. The tubes were then filled with pure dry nitrogen gas and sealed.

The sealed tubes containing the standard were then transferred to cold storage and are there maintained constantly at a temperature of − 2° C. to − 4° C.

Definition of the Unit

At the London Conference it was agreed that the unit accepted for International use shall be the specific oestrus-producing activity contained in 0·1 gamma (= 0·0001 mg.) of the standard preparation, this quantity being approximately one-third of the original rat unit of activity as defined by Professors Allen and Doisy.

Suggestions for Use

A small desiccator, containing phosphorus pentoxide, should be reserved for the storing of the tube containing the International Standard. A small quantity of the standard should be removed from the tube and accurately weighed on a microbalance, the ampoule being returned to the desiccator. The small weighed quantity should be dissolved in oil, or alternatively in a small amount of alcohol, and suitably diluted for injection.

Precautions to be Observed in the Conduct of Biological Tests

The following notes and recommendations are based on the Report of the London Conference (C.H. 1091 dated Geneva, September 9th, 1932).

(i) The method regarded by the Conference as providing a suitable basis for the quantitative determination of activity, in comparison with the standard preparation, was that based on the series of changes which occur after the injection of oestrogen, in the cellular contents of the vaginal secretion of the spayed rat or mouse. The increase in the weight of the uterus of the immature intact female rat which follows the injection of a single dose of oestrogen and reaches a maximum in about 6 hours is also a suitable method.

(ii) The comparison of an unknown preparation with the standard for such specific activity can only be accurately made if the conditions of administration of both, and of observing the results produced in each case, are completely identical, particularly in the following respects:

(*a*) The Standard and the preparation under test must be administered in the same volume of identical solvents.

(*b*) Administration should be by truly subcutaneous and not intramuscular injection.

(*c*) The Standard preparation and the preparation under test should, in tests employing vaginal smears, both be administered by a method ensuring slow absorption at as near as possible identical rates. This can best be attained by administering both dissolved in the same oily solvent or by preparing both in watery solution and administering in an equal number of fractions, not less than three, the injections being spaced in an identical manner in both cases over a total time of not less than nine hours.

(*d*) The vaginal smears should be taken at the same intervals after the injection, from the animals injected with the Standard, and from those injected with the preparation under test, but should be continued at least once daily until the animals have returned to the dioestrous condition. On the day on which the maximum response is expected, at least two smears should be taken. If the maximum occurs in the animals receiving the Standard, and in those receiving the preparation under test, at intervals after the injection differing by more than twenty-four hours, the comparison cannot be regarded as valid.

(*e*) In comparing the activity of a preparation with the Standard, the procedure should aim at finding a dose of the unknown preparation producing the chosen reaction in the same proportion of animals, or to the same average degree in a group of animals, as that in which it is produced by a known dose of the Standard, as determined either directly or by reference to a standard curve. In the latter instance, the *position* of the standard curve must be determined afresh at each assay, its slope, if previously determined with accuracy, may be assumed to be constant. It is assumed that probit/log-dose co-ordinates will be used in computing potencies. A method should be chosen which has proved, in the hands of the particular investigator, to be capable of determining such equality of activity with a standard error of not more than $\pm$ 20 per cent. According to the experience of Members of the Conference, an accuracy of this degree is not attainable by the vaginal smear method, unless at least 20 animals each are used for the standard and test preparations. If a standard error of $\pm$ 10 per cent is demanded at least 80 animals per preparation would be needed.

October 1942

M.47

MEMORANDUM ON THE REPLACEMENT OF THE SUBSTANCE OF THE INTERNATIONAL STANDARD FOR MALE HORMONES: ANDROSTERONE

Prepared by

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At the Second International Conference on the Standardisation of Sex Hormones, held in London in July 1935, under the auspices of the Permanent Commission on Biological Standardisation of the Health Organisation of the League of Nations, it was decided to adopt an international standard of male hormone activity. Although the Conference recognised that androsterone was probably not the primary male hormone as elaborated by the testis itself, it was considered that this substance would be a suitable standard of reference, and it was decided that the proposed international standard for male hormone activity should consist of a pure crystalline preparation of artificially prepared androsterone with the following characters:

M.P. 183·5° to 184·5° (corrected)

$[\alpha]_D^{20}$ + 97·3° in absolute alcohol

The National Institute for Medical Research was to act on behalf of the Health Organisation of the League of Nations to keep the standard and to distribute it to national control centres.

A total of 10·17 g. of androsterone satisfying the requirements was obtained by pooling material from four contributors. This material was dispensed into ampoules each containing about 50 mg. and prepared for distribution. During 1939 the standard became exhausted and a canvass among members of the 1935 Conference showed a general opinion that the androsterone standard was useful and should be maintained. Arrangements were accordingly made for the renewal of the standard. Much delay was occasioned by the outbreak of war, but a generous gift of 30 g. of androsterone by Dr. Miescher on behalf of Ciba of Basle, has enabled the standard to be renewed on an adequate scale.

Preparation of the Second International Standard

The melting point and specific rotation of the androsterone provided for the establishment of the Second International Standard was determined and found to be:

M.P. 182° to 183° (corrected)

$[\alpha]_D^{20}$ + 96·5 (c = 1·06 in absolute ethyl alcohol)

The preparation, therefore, conforms to the requirements laid down at the Second International Conference on the Standardisation of Sex Hormones and may be accepted as suitable for adoption as the Second International Standard for Male Hormone.

The whole of the material provided for the Second International Standard (30 g.) was distributed, in approximately 50 mg. quantities, into small dry tubes. The tubes were placed *in vacuo* over phosphorus pentoxide for some weeks, sample tubes being weighed at intervals until an assurance was obtained that the weight was constant and accordingly the standard material was completely dry. The tubes were then filled with pure dry nitrogen gas and sealed.

The sealed tubes containing the standard were then transferred to cold storage and are there maintained constantly at a temperature of − 2° C. to − 4° C.

Definition of the Unit

At the Second Conference on the Standardisation of Sex Hormones, held in London in July 1935, it was agreed that *the International Unit of male hormone activity* should be defined as *the activity of* 0·1 *mg. of the International Standard Preparation of Androsterone, as measured by a specific biological reaction.* This weight is, approximately, the daily dose required to give an easily measurable response in the capon comb after five days. The Conference agreed that, on the available information, the test based on the induction of growth in the comb of the capon is at present the only one sufficiently specific for the quantitative determination of activity in comparison with the standard preparation.

Suggestions for Use

A small desiccator containing phosphorus pentoxide should be reserved for the storing of the tube containing the International Standard after the tube has been opened and part of the contents used. A small quantity of the standard should be removed from the tube and accurately weighed on a micro-balance the ampoule being then returned to the desiccator. The small weighed quantity should then be dissolved in oil or other solvent and suitably diluted for injection.

Precautions to be Observed in the Conduct of Biological Tests

The following notes and recommendations are based on the Report on the Second Conference on the Standardisation of Sex Hormones, London, 1935 (see *Quarterly Bulletin of the Health Organisation of the League of Nations*, Vol. IV, pp. 618–630).

The comparison of an unknown preparation with the standard for specific activity as defined above can only be accurately made if the conditions of administration of both, and of measuring the results produced in each case are completely identical, particularly in the following respects:

(*a*) The standard and the preparation under test must be administered in equal volumes of identical solvents. The method of administration must be identical for the standard and for the substance under test.

(*b*) The method of administration originally recommended by the Conference was injection into the pectoral muscles. Superficial application to the comb has also proved satisfactory, and although the response may be rather more variable than that to intramuscular injection, the method is extremely sensitive. It is suggested that the accuracy of methods based on the response of the comb of the baby chick should be further investigated.

(*c*) If, as is customary, the total dose is divided into portions for injection on several days, the division and the spacing of the doses must be identical for the standard and for the substance under test.

(*d*) In comparing the activity of a preparation with the standard, the procedure should aim at finding a dose of the unknown preparation producing a reaction quantitatively identical with that produced by a known dose of the standard, as determined either directly in the test or by reference to a standard curve drawn from results obtained on the same stock of birds. The *position* of the standard curve, if used, should be determined at each assay.

No sufficient information is yet available concerning the biological activity of artificially esterified preparations of male hormones to justify any detailed recommendation with regard to their standardisation. The androsterone standard now adopted, does not afford a suitable basis for their comparative assay in units, by simultaneous observation of their effects.

Recommendations for Labelling

The following notes and recommendations are extracted from the Report of the Second Conference on the Standardisation of Sex Hormones, London, 1935.

For the information of the practical user it is necessary that the activity of incompletely purified preparations, or extracts, containing forms of the hormone other than that constituting the standard, should be indicated in international units on the label of the bottle or other container of the preparation. These units, however, may have a varying value in accordance with details of application of the test. It is accordingly recommended that, with a view to greater uniformity of dosage in practice and to the more accurate interpretation of effects recorded in the literature, the details of the method used in determining the indicated unitage should be given in some document accompanying each package of such preparations.

In the case of preparations of pure substances of this group, the exact weight and chemical nature of the substance present should be indicated, in addition to a statement of the activity in international units; and the activity of a preparation or an extract consisting of incompletely purified substances should be indicated in the equivalent weight of the standard, as well as in units of activity. In the case of such incompletely purified preparations or extracts, it should be stated whether they are prepared from urine or from testes, or from both, and in the last case in what proportion.

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M.46

MEMORANDUM ON THE REPLACEMENT OF THE SUBSTANCE OF THE INTERNATIONAL STANDARD FOR THE PROGESTATIONAL HORMONE OF THE CORPUS LUTEUM: PROGESTERONE

Prepared by

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At the Second International Conference on the Standardisation of Sex Hormones, held in London in July 1935, under the auspices of the Permanent Commission on Biological Standardisation of the Health Organisation of the League of Nations, it was decided to establish a preparation as an international standard for the progestational hormone of the corpus luteum for which the name Progesterone was to be adopted. The Conference further agreed that the material for the standard should consist of pure crystalline progesterone, and that the National Institute for Medical Research, Hampstead, was to act as the Central Laboratory on behalf of the Health Organisation of the League of Nations, to keep the standard and to distribute it to national control centres.

Considerable difficulty was experienced in obtaining sufficient material for the establishment of this standard, but after considerable delay a total quantity of 3 g. was obtained from three laboratories; the three contributions were mixed, recrystallised, and finally 2·954 g. of a crystalline product, the physical properties of which are set out below (Table 2), was obtained. This material was dispensed in 20 mg. quantities in small glass tubes, and after being completely dried, the tubes were filled with pure dry nitrogen gas, sealed and placed in cold storage pending distribution to the National Control Centres of different countries.

The small quantity of material available for the first International Standard Progesterone yielded only 118 ampoules and it was obvious that early replenishment would be necessary. Accordingly, steps were taken to obtain further quantities of pure crystalline progesterone with a view to the establishment of a Second International Standard Preparation to take the place of the First Standard when that should be exhausted.

Thanks to the generosity of Messrs. Ciba Limited, N. V. Organon, Laboratoires due Dr. Roussel, and Schering-Kahlbaum, each of whom contributed a quantity of approximately 12·5 g. of Progesterone, a total quantity of 50 g. of material has been placed at the disposal of the Health Organisation of the League of Nations for the purpose of establishing a Second International Standard for Progesterone. Grateful acknowledgment is made for these generous contributions of valuable material.

Preparations of the Second International Standard

In each case the weight of material removed from the container was determined and the melting point, specific rotation in absolute alcohol and absorption coefficients in the ultraviolet in alcoholic solution were also determined. The results of these examinations are collected in Table I.

The four samples were mixed and extracted in a Soxhlet apparatus with about 200 c.c. of ether. The progesterone crystallised out from the ethereal solution in the prismatic

Table I

No. of sample	Weight (gm.)	M.P. (Kofler: uncorr.)	$[\alpha]_D^{25}$ c = 0·51	$[\alpha]_{5,461}^{25}$ c = 0·55	1% E(max.) 1 cm.	λ max
1	12·52	131°–131·5°	+ 186°	+ 232°	590	2,410
2	12·60	129·5°–130·5°	+ 185°	+ 223°	520	2,410
3	12·66	130°–131·5°	+ 188°	+ 225°	590	2,410
4	12·55	129·5°–131°	+ 188°	+ 229°	550	2,410

form: it was separated, dried, and the physical constants determined. These are given in Table II and, for comparison, the corresponding constants for the sample of progesterone which served as the First International Standard are also given in the table.

Table II

Preparation	M.P. (Kofler: uncorr.)	$[\alpha]_D^{25}$ c = 0·257	$[\alpha]_{5,461}^{25}$	1% E (max.) 1 cm.	λ max.
Second International Standard	130°–132°	+ 191°	+ 226°	530	2,410
First International Standard	128·5°–129°	+ 192°	+ 232°	510	2,430

The two preparations therefore resembled each other so closely in physical properties that the new standard may be regarded as identical with the original one.

Distribution of the Standard into Small Ampoules

The whole of the material provided for the Second International Standard (46·23 g.) was distributed, in approximately 65 mg. quantities, into small dry tubes. The tubes were placed *in vacuo* over phosphorus pentoxide for some weeks, sample tubes being weighed at intervals until an assurance was obtained that the weight was constant and, accordingly, the standard material was absolutely dry. The tubes were then filled with pure dry nitrogen gas, and sealed.

The sealed tubes containing the standard were then transferred to cold storage and are there maintained constantly at a temperature of − 2° C. to − 4° C. pending their despatch to the National Control Centres of different countries.

Definition of the Unit

The International Unit for the progestational hormone is defined as the specific progestational activity of 1 mg. of the international standard preparation.

Suggestions for Use

A small desiccator, containing phosphorus pentoxide, should be reserved for the storing of the tube containing the International Standard. A small quantity of the standard should be removed from the tube and accurately weighed on a microbalance, the ampoule being returned to the desiccator. The small weighed quantity should be dissolved in oil at a dilution suitable for injection.

Notice: To open the tube, a deep file mark is made entirely round the tube. The tubes are made of an exceptionally hard glass and do not "crack" easily when a hot point is applied to the file mark so made. If the end of the tube cannot be removed by the application of a hot point to the file mark, the tube should be held in a horizontal position and the break effected in the usual way. Since the tubes are filled with pure, dry, nitrogen gas, at ordinary pressure, glass splinters, should these be formed, should fall away and not into the tube.

Precautions to be Observed in the Conduct of Biological Tests

The following notes and recommendations are based on the Report of the Second Conference on the Standardisation of Sex Hormones, London, 1935 (see *Quarterly Bulletin of the Health Organisation of the League of Nations*, Vol. IV, pp. 618–630).

(*a*) The test for activity in International Units must, according to present information, be based on the production of a progestational reaction.

(*b*) The only method of applying this progestational reaction in practical testing, concerning which sufficient information is available to justify a recommendation, is that based on the degree of proliferation produced in the endometrium of the rabbit. This can be applied to the adult female rabbit deprived of the ovaries after coitus (Corner and Allen), or to the immature female rabbit after treatment with the oestrus-producing hormone (Clauberg).

(*c*) The comparison of an unknown preparation with the standard for such specific activity can only be accurately made if the conditions of administration of both, and of observing the results produced in each case, are completely identical; in particular the standard and preparation under test must be administered in the same volume of identical solvents.

Recommendations for Labelling

In the case of preparations of pure substances of this group, the exact weight and chemical nature of the substance present should be indicated, in addition to a statement of the activity in International Units; and the activity of a preparation or an extract consisting of incompletely purified substances should be indicated in the equivalent weight of the standard, as well as in units of activity.

October 1942

M.43

MEMORANDUM ON THE REPLACEMENT OF THE SUBSTANCE OF THE INTERNATIONAL STANDARD PREPARATION FOR PITUITARY POSTERIOR LOBE

Prepared by

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Introduction

The International Standard hitherto in use for preparations of the Pituitary posterior lobe was adopted in October, 1935, by the Permanent Commission on Biological Standardisation of the League of Nations Health Organisation.[1] This Standard material thus internationally adopted had been in use, since its preparation in 1926, as the British Standard for extracts of the Pituitary posterior lobe. The National Institute for Medical Research, Hampstead, London, was asked to act as the Central Control Laboratory for the preservation of the Standard, and for its distribution to the National Control Centres of other countries, requiring it for the comparative evaluation of their own locally prepared national standards.

By 1939 the greater part of this Standard had been issued to the various national centres, and it was necessary to take steps to replenish it. Arrangements were accordingly made for the preparation of a further quantity of the dried posterior lobe substance, in exact accordance with the methods used in preparing the original Standard, and now official in the U.S. Pharmacopoeia and in the Appendix to the British Pharmacopoeia. The material, as completely prepared in its final dry form, amounted to about 50 g. The Armour Laboratories of Chicago had generously provided the raw material and undertaken its preparation. The material was received at the National Institute for Medical Research in 5 sealed glass ampoules, each containing about 10 g. of the dry powder. From these, after careful mixing, it was distributed into over 1,500 small glass ampoules, a little more than 30 mg. being delivered into each. According to the routine now applied at the Institute for the preparation of such standards, the material was then further dried in the ampoules in evacuated desiccators over phosphorus pentoxide, until check weighings showed that constant weight had been attained. All the ampoules were then filled with pure, dry nitrogen and sealed. The same procedure should be followed in the preparation of National Standard samples in comparison with it. (See Memorandum M. 36, p. 7.)

Preliminary tests at the National Institute for Medical Research indicated that the new material, in respect of its three major activities—oxytocic, pressor and antidiuretic—was at least as potent as the existing Standard. Evidence was, in fact, obtained that the new material was slightly, but definitely, more active than the old, its margin of superiority being, for each type of activity, in the neighbourhood of 15 per cent. The distribution of the three activities appeared, therefore, not to be perceptibly different in the two preparations.

Since the preliminary tests thus indicated that the new material would be suitable as an International Standard, arrangements were made to have its potency in relation to the existing Standard independently determined, as exactly as possible, by workers in a number

[1] An account of the earlier happenings and experience on which this decision was based, and of the stages by which decisions were reached to define units of the three important activities in terms of the one Standard preparation, is given in Memorandum M. 36 of this series, to which the present Memorandum is supplementary.

of other laboratories. Under normal conditions, it would have been desirable to organise a fully international and co-operative comparison of the two preparations, the results of which would have been submitted to an International Conference. As this was impossible, a number of laboratories in Great Britain, the United States of America, France, Holland and Hungary were invited to collaborate: samples of both materials were sent to them, and they were asked to determine the relative activities of these by the methods which were familiar to them.

In the case of Standards for substances having no fixed or limiting activity, such as those for the different antitoxins, the units have a purely arbitrary value, fixed in terms of the particular sample originally adopted as a Standard, and maintained at successive renewals by exact determinations of the value of each new Standard sample in comparison with its predecessor. The redetermination of units in terms of a new Pituitary Posterior Lobe Standard is on a somewhat different basis. When this Standard was originally adopted by the League of Nations, Professor Voegtlin suggested that its preparation in accordance with prescription, at any time or place, would always give material of the same activity. There is, in fact, no reason to doubt that, if the material could always be obtained with the same minimum delay after slaughter, and immediately subjected to an invariable routine of dissection, extraction and drying, the resulting powder would show a constant and maximal activity. Experience showed, however, that those optimal conditions could not always be expected or attained in every country, so that the provision of an International Standard preparation was desirable and necessary. The need for it was increased when, in due course, separate units were adopted for the pressor and anti-diuretic activities of the Posterior Lobe extract, since new possibilities of variation arose from these decisions. Hitherto comparisons of the different samples of Posterior Lobe powder had been made by measurements of the oxytocic action. Account had now to be taken of the further possibility, that even such samples as were indistinguishable in oxytocic activity might be found to differ significantly from one another with respect to either or both of the two other activities, for which units had now been defined.

These considerations suggested certain conclusions with regard to the redefinition of the units in terms of the new Standard.

(1) If, as the preliminary comparisons suggested, the new Standard proved to be perceptibly, though only slightly, more active than the old, this fact would not warrant any change in the weight of the Standard to be adopted as containing 1 unit of activity. The slight difference, whatever its cause, might properly be regarded as indicating a closer approximation of the new Standard, at the date of comparison, to the ideal which was aimed at in the original definition of the unit, as the specific activity contained in 0·5 mg. of the dry powder, obtained by the prescribed procedure from the perfectly fresh and cleanly dissected posterior lobe material. There was no possibility, through a cumulative effect of such slight increases in the value of the unit at successive renewals, of increasing its value beyond that natural optimum.

(2) It would be important to put to further test the indication obtained in the preliminary comparisons, that the three different activities of this Standard, for which units had been defined, were represented in the new Standard in proportions to one another not significantly different from those in which they were found in the old. If this were confirmed—if, in other words, the three different activities of the new Standard all exceeded the corresponding activities of the old Standard in the same definite but small proportion—then it would be proper to suggest that each of the units should be redefined as the corresponding activity of 0·5 mg. of the new Standard material.

These considerations were recalled to those responsible for the comparative tests, at the time when the samples of the two preparations were distributed. It was suggested that, if the preliminary findings were generally confirmed, the proper policy would be simply to adopt the new Standard, without any modification of the unit indication; so that 0·5 mg. of the new Standard would still be indicated as the quantity containing

1 oxytocic, 1 pressor, or 1 antidiuretic unit. The results of the preliminary tests were in fact, as will be seen, substantially confirmed; and, of the participating experts who have reported, a number have specifically approved and none have disapproved the recommendation that the definition of each of the three units should remain without change of weight, as the specific activity of 0·5 mg. of the new Standard.

Results of the Co-operative Comparison

The laboratories listed below have co-operated in the determination of the relative potency of the two preparations. In the entry relating to each laboratory the name first mentioned is that of the contributor reporting the results.

Laboratory	Investigators	Activities compared
Laboratoire Nationale de Controle des Medicaments, Paris, France	Dr. Ch. Lormand	Oxytocic
Rijks Instituut v.d.Volksgezondheid, Utrecht, Holland	Dr. W. A. Timmerman	Oxytocic
	Dr. L. W. v. Esveld	Pressor
	Dr. M. Eekelen	Antidiuretic
State Hygienic Institute of Hungary, Budapest	Dr. J. Tomcsik	Oxytocic
	Dr. A. Stasiak	Pressor
Division of Pharmacology, National Institute of Health, Washington, U.S.A.	Dr. M. I. Smith	Oxytocic Pressor
Laboratories of Parke, Davis & Co., Ltd., Detroit, Mich., U.S.A.	Dr. L. T. Clark	Oxytocic Pressor Antidiuretic
The Lilly Research Laboratories, Indianapolis, U.S.A.	Dr. G. H. A. Clowes	Oxytocic
	Dr. E. E. Swanson	Pressor
	Dr. K. K. Chen	Antidiuretic
Armour Laboratories, Chicago, U.S.A.	Dr. F. Fenger	Oxytocic
Department of Pharmacology, University of Pennsylvania, Philadelphia, U.S.A.	Prof. A. N. Richards Dr. A. M. Walker	Antidiuretic (rabbit)
College of the Pharmaceutical Society, London, England	Dr. F. J. Dyer	Oxytocic
	Dr. F. Schutz	Pressor
	Dr. R. Wien	Antidiuretic
	Mr. P. B. Marshall	
Department of Pharmacology, Oxford University, Oxford, England	Prof. J. H. Burn	Oxytocic
	Dr. C. S. Jang	Pressor Antidiuretic
Laboratories of the British Drug Houses Ltd., London, N.1, England	Dr. S. W. F. Underhill	Oxytocic Pressor Antidiuretic
The Wellcome Physiological Research Laboratories, Beckenham, England	Dr. J. W. Trevan	Oxytocic Pressor
National Institute for Medical Research, Hampstead, London, N.W.3, England	Dr. F. C. MacIntosh	Oxytocic Pressor Antidiuretic

The methods used for the comparison were, in nearly all the laboratories, essentially the same; oxytocic activity being determined on the isolated guinea-pig uterus, pressor activity on the spinal cat, and antidiuretic activity on rats after oral administration of

water. In two laboratories pressor activity was determined on anaesthetised dogs, and in one laboratory the comparison of antidiuretic activity was made by a semi-quantitative method on rabbits.

The values obtained in the different centres are listed below: the order is not the same as that given above for the laboratories. In every case, the first figure is the potency of the proposed new Standard, expressed as a percentage of that of the old Standard. The mean of these values, and the standard deviation of the mean $\varepsilon = \sqrt{\left(\frac{\Sigma d^2}{n(n-1)}\right)}$ is given for each type of activity. One or two investigators have given for the ratio of activity of the two preparations, not an exact figure, but only limits between which this lies; in this case, the average of the limits has been used in calculating the mean. It would, no doubt, have been preferable to calculate the standard error of the results of the individual tests; but this was impossible, since in several cases the investigator had reported only a final value based on his tests as a whole, and had not given the results of the individual tests. For the same reason, no figure is given for the standard deviation of the weighted mean. The figure in parentheses is, for the oxytocic and pressor assays, the number of assays on which the first figure is based, and for the antidiuretic assays, the product of the number of rats used and the number of doses of pituitary extract received by each rat. This figure has been used, in each case, as weighting factor for the calculation of the weighted mean. One or two investigators, also, have reported carrying out "several" assays: for calculation of the weighted mean, "several" has been assumed to mean four. It should be said here that the value of such a weighted mean is questionable, at least for the oxytocic and pressor assays, in which accuracy depends more on the experience and judgment of the worker, and on the suitability of the animal preparation, than on the multiplication of experimental data. Such an objection applies with much less force to the antidiuretic assays; but here too it is impracticable, in a brief co-operative study of this sort, to allow for a number of factors, e.g. the slope of the dosage-response curve, which may vary from one laboratory to another and affect the weight of a result. In any case, the difference between weighted and unweighted means is, in this study, insignificant.

The following are the results obtained in the several laboratories.

Oxytocic activity	*Pressor*	*Antidiuretic*
110 (4)	120 (3)	111 (64)
111·5 (8)	115 (2)	128 (64)
119 (12)	130 (3)	122 (72)
125 (3)	100 (9)	112·5 (160)
105 (several)	121 (3)	107·5 (36)
100 (several)	112·5 (5)	118 (144)
106·5 (23)	117 (3)	73[1] (70)
112 (several)	107·5 (5)	
100 (4)	120 (5)	
115 (4)		
113 (11)		
115 (9)		
Mean 111·0 ± 2·1	*Mean* 115·5 ± 2·6	*Mean* 116·5 ± 3·1
Weighted Mean 111·0	*Weighted Mean* 113·0	*Weighted Mean* 116·5

By the semi-quantitative rabbit antidiuretic method the new standard was found to be somewhat stronger than the old.

Remarks

It may be remarked that the values obtained by all methods agree very satisfactorily, there being no significant difference between the distribution of the three sorts of activity

[1] Not included in calculating means. See under "Remarks."

in the two preparations. With one exception, all the investigators find the new powder equal or superior to the old in potency. In one laboratory only, the new standard has been found, after an obviously careful examination, to contain 27 per cent *less* antidiuretic activity per unit weight than the old. This finding is so completely at variance with all the others, that it is impossible to avoid the suspicion that it was the result of some unaccountable deterioration or contamination affecting one sample of the new standard material, or of some other accidental circumstance, and it has, accordingly, been excluded in the calculation of the mean.

From the combined results it may be concluded with some certainty that the new material is more potent than the old in respect of its three important activities, its margin of superiority being probably between 10 and 20 per cent, and that the distribution of the three activities is not significantly different in the two preparations. For reasons already discussed, however, it had been suggested that a difference of this minor degree in favour of the new Standard, if confirmed, would not make it advisable to redefine the unit as contained in a smaller weight of the new Standard than of the old Standard. This suggestion has now been reinforced by the fact that two contributors to the assay failed to detect any difference between the old and the new Standards in oxytocic activity, and one contributor similarly failed to observe any difference in pressor activity.

This Institute, therefore, as the Central Control Laboratory for the distribution of the International Standard Pituitary (posterior lobe) Powder, and in agreement with all the opinions recorded by those who have co-operated in the international trials, decides that the new material here under report shall be henceforth regarded as the International Standard for extracts of the Pituitary Posterior Lobe, and that 0·5 *mg. shall be accepted as the quantity of the new Standard containing* 1 *oxytocic*, 1 *pressor, or* 1 *antidiuretic unit.*

July 22, 1942

M.48

MEMORANDUM ON THE INTERNATIONAL PREPARATION OF DESICCATED OX ANTERIOR PITUITARY GLAND

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At the Third International Conference on the Standardisation of Hormones, held in Geneva in August, 1938, under the auspices of the Health Organisation of the League of Nations, considerable discussion took place concerning certain active principles of the anterior pituitary gland for which the establishment of standards was not recommended. With a view to facilitating research, and particularly the comparative assay of these principles, the following resolution was adopted by the Conference.

"Consideration of a Standard Preparation for the Whole Anterior Lobe of the Pituitary Gland

"The Conference expressed the hope that the Standards Department of the National Institute for Medical Research, Hampstead, London, England, would be able to facilitate comparative research on the hormones of the anterior lobe of the pituitary gland for which international standard preparations had not yet been adopted, by preparing or acquiring a suitable quantity (not less than 2 kg. or more than 5 kg.) of anterior pituitary lobe substance of oxen, dried without fractionation, so that samples might be furnished to participants, in different countries, in the co-operative investigations planned by the Conference.

"The Chairman gave an undertaking that the National Institute for Medical Research would do what was possible in the direction desired."[1]

After considerable delay due to a number of different causes it has now been possible to make available for distribution material of the kind referred to in the recommendation of the Conference.

Preparation of the Material

4·5 kg. of dessicated ox anterior pituitary gland was provided by Armour and Company, Chicago, through the courtesy of Mr. J. B. Bradshaw whose generous assistance is hereby acknowledged.

The following description of the method of preparation was furnished by the Armour Laboratories: "The glands were collected in acetone, being minced as they were placed in the acetone. Four extractions were made with anhydrous alcohol, followed by two extractions with petroleum ether. The material was then dried in a vacuum oven without heat."

The material, which was received at the National Institute for Medical Research, Hampstead, England, in the form of a finely divided powder, was subjected to various biological assays (see below) and was then filled out into wide test tubes, each containing

[1] Report of the Third International Conference on the Standardisation of Hormones (Geneva, August 11th to 12th, 1938): see *Bulletin of the Health Organisation, League of Nations*, 1938, Vol. VII, p. 887.

approximately 5 g. The tubes were placed, in an upright position, in large desiccators containing phosphorus pentoxide and the desiccators evacuated. The tubes were left, exposed to the dehydrating action of phosphorus pentoxide *in vacuo*, for several months; they were then filled with pure dry nitrogen gas and sealed. The sealed containers were then transferred to cold storage (— 2° C. to — 4° C.) where they have been continuously maintained since their preparation was completed.

Biological Assay of the Material

The biological assays carried out on the preparation were as follows:

Gonadotrophic hormone: Tested at the National Institute for Medical Research on immature female rats, five daily doses of 25 mg. of the powder caused an increase in ovarian weight to 37 mg. from the normal weight of 10 mg.; tested on oestrous rabbits, 1 mg. given intravenously caused ovulation in 4 out of 11 animals, 1·5 mg. in 6 out of 9 and 2·0 mg. in 5 out of 5.

Prolactin: Armour Laboratories reported that assays carried out in their laboratories by Dr. Kutz showed that the material contained about 600 I.U. of prolactin per g.

To obtain additional information specimens of the material were sent to two interested members of the 1938 Conference, Dr. Riddle (Baltimore) and Dr. Hamburger (Copenhagen). These workers reported, respectively, that the material contained 450 I.U. and 550 I.U. of prolactin per g.

Thyrotrophic hormone: Tested at the National Institute for Medical Research on groups of ten immature female guinea-pigs, having an average weight of 230 g., five daily doses of 10 mg. raised the thyroid weight from the normal of 30 mg. to 44 mg. Twice this dose brought the thyroid weight to 57 mg.

Suggestions for Use

A small desiccator containing phosphorus pentoxide should be reserved for the storing of the tube containing the international preparation, after the tube has been opened and part of the contents used. The quantity required for a series of tests should be removed and accurately weighed, the tube being then returned to the desiccator. The weighed quantity should then be suspended in saline for injection. Suspension of the material is facilitated by grinding it with saline in a mortar and bringing the volume to the required amount.

Supply of the International Preparation

The International Preparation of Desiccated Ox Anterior Pituitary Gland is maintained on behalf of the Health Organisation of the League of Nations, at the National Institute for Medical Research, Hampstead, London, N.W.3, and supplies will be forwarded on request to the Directors of National Control Centres for local distribution in their respective countries, or to individual laboratories, institutes or research workers in those countries in which national control centres have not yet been established.

October 1942

MEMORANDUM ON THE INTERNATIONAL STANDARD FOR THE LACTOGENIC (CROP GLAND-STIMULATING) SUBSTANCE OF THE ANTERIOR LOBE OF THE PITUITARY GLAND (PROLACTIN, GALACTIN, MAMMOTROPHIN)

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APPENDIX

Method of Making Solution

Grind one tablet (10 mg.) of the standard preparation in a mortar with the minimum amount of N/1,000 sodium hydroxide solution required to make a stiff paste (about 0·02 c.c.). Continue the grinding for about 2 minutes, and then slowly add, grinding all the time, sufficient N/1,000 sodium hydroxide solution to make the total volume up to 5 c.c. The pH of the solution should then be approximately 7·8 (glass electrode). The solution will be slightly cloudy owing to the presence of about 0·7 mg. of the powder in suspension, but is quite suitable for injection. The addition of 45 mg. of sodium chloride to make a 0·9 per cent solution can be made if necessary.

STANDARD PREPARATIONS FOR THE ASSAY OF THE THREE GAS-GANGRENE ANTITOXINS, *CL. PERFRINGENS*, *CL. VIBRION SEPTIQUE* AND *CL. OEDEMATIENS*

by P. HARTLEY and D. G. EVANS

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In April 1940 a request was received from the Health Organisation of the League of Nations enquiring whether the Medical Research Council would be prepared to supply the International Standards for the antitoxins and antisera to those laboratories which had hitherto received these materials from the State Serum Institute, Copenhagen. Since considerable stocks of these international standards are maintained at Hampstead, for supply to laboratories in Great Britain and the Overseas Dominions in fulfilment of the obligations undertaken in respect of the British Therapeutic Substances Act (1925) and the British Pharmacopoeia, 1932, the Medical Research Council readily acceded to this request and was glad to make this contribution in order to ensure the continuance of this international service.

Of the twelve standards concerned and maintained at Hampstead, nine are part of the identical material adopted as the international standard and supplied, in normal times, from Copenhagen to laboratories other than those situated in Great Britain and the Overseas Dominions. The British standards for diphtheria and tetanus antitoxin have been repeatedly and regularly tested in the Laboratories of Hampstead and Copenhagen, and the identity of the unit, as defined in terms of the British standards, with the international unit, has been established, and assurance that this identity has been constantly maintained, has been secured. The Hampstead Institute also maintains and provides for issue a standard solution of diphtheria antitoxin for use in the flocculation test. It was possible, therefore, for the Department of Biological Standards at Hampstead to undertake the service requested by the League and accepted by the Medical Research Council.

At the request of the Health Organisation of the League of Nations, a notification of the arrangements which had been made to ensure the continuance of the supply of these standards for antitoxins and antisera was widely circulated, and a notice to the same effect was published in the medical and scientific journals in England in order that this alternative source of supply of the antitoxin and antiserum standards should be made known as widely as possible.

It was soon evident that, while the stocks of the International Standards for the Gas-Gangrene Antitoxins for *Cl. perfringens*, *Cl. Vibrion Septique* and *Cl. oedematiens* were adequate to serve the needs of Great Britain and the Overseas Dominions for some years, they were not sufficiently large to

meet the increased demands which might be made on them in consequence of these new arrangements. Their replacement, therefore, became a matter of some urgency, and steps were at once taken to meet the possible emergency. In view of the fact that the preparation of an antitoxin standard, and the determination of the unit value in terms thereof, takes a considerable time, it is fortunate that this possible shortage was foreseen, and action taken well in advance of its actual occurrence, so that it has been possible to maintain the supply of these three standards without interruption.

The purpose of this note is twofold. Firstly, since it has not been possible, because of the exhaustion of the stocks of material, to continue the issue of standard solutions prepared from material identical with that maintained at Copenhagen, laboratories in those countries which are temporarily using the standards made available to them from Hampstead will need to be assured that, in providing standard solutions prepared from material other than the actual international standard, every care has been taken to ensure that the replacement standards are as nearly identical with the original international standards as is possible and practicable, and, in particular, that the values of the international units are being maintained. Secondly, while these three new dry stable antitoxin preparations take their place as the British Standards for perfringens, Vibrion Septique and oedematiens antitoxin, respectively, it is neither intended nor suggested that they should replace the corresponding international standards deposited at Copenhagen. They are in the same category and enjoy the same status as the British Standards for diphtheria and tetanus antitoxins and, like these, they have been assayed as exactly as possible in terms of the existing international standards.

It is well known that antitoxins may exhibit abnormal properties, displayed in the manner of their combination with the corresponding toxins, the firmness of such combination and so on, and it is important that a sample differing markedly from the normal should not be chosen to serve as a standard preparation. Under present circumstances, whereby laboratories which have been accustomed to use the international standards and will resume that use in the future, it was considered of especial importance to ensure that the standard preparations supplied for the use of such laboratories in the interim period should resemble, as closely as possible, the international standards. The material for the preparation of the three British standards, for perfringens, Vibrion Septique and oedematiens antitoxins respectively, has been generously provided by the Wellcome Physiological Research Laboratories, Beckenham, and we are indebted to Mr. A. T. Glenny for his help and co-operation in selecting samples of each of the three antitoxins which resemble very closely the original international standards.

Of equal importance to the character and properties of the standard preparations is the determination of their potency in terms of the corresponding international standard, for on this depends the maintenance of the international unit as defined and accepted by international agreement. Accordingly, each of the three finally prepared standard preparations has

been assayed in this laboratory, in strict comparison with the corresponding international standard, using several toxins and various methods; and, moreover, we have had the advantage of the co-operation of Mr. A. T. Glenny of the Wellcome Laboratories and Dr. G. F. Petrie of the Lister Institute of Preventive Medicine, who have kindly carried out similar comparative tests in their own laboratories. The protocols of all these tests are recorded in the sections which follow, from which it will be seen that the determination of the international units in terms of these three new standard preparations has been effected with an accuracy within the limits of error of the assay itself. It is submitted that the value of the international unit has, in each case, been maintained, and that the standard preparations for perfringens, Vibrion Septique and oedematiens antitoxins supplied from this Institute may be accepted with confidence by workers in other countries who may require them, in this temporary war emergency, for purposes of standardisation and assay.

The Production of Dry Stable Preparations from the Three Selected Samples of *Cl. Perfringens*, *Cl. Vibrion Septique* and *Cl. Oedematiens* Gas-Gangrene Antitoxins

The conversion of the chosen samples of antitoxin into the dry stable condition was carried out by the usual method employed at this Institute.[1] Approximately 2·5 litres of each of the antitoxins were available. By means of a precision distribution apparatus and with aseptic precautions exactly 5 c.c. quantities of serum were distributed into ampoules; for each antitoxin a total of about 400 such filled ampoules was obtained. Samples taken at different stages during the distribution of each antitoxin were all found to be sterile. The contents of the ampoules were reduced to the dry condition by exposure to the dehydrating action of phosphorus pentoxide in high *vacuo*. The drying was continued until no further loss of weight, as shown by the periodic weighing of six selected ampoules from each sample of antitoxin, occurred. The ampoules were then filled with pure dry nitrogen gas, sealed, and transferred to cold storage at — 2° C. to — 4° C.

The Assay of the Dry Preparations in Terms of the Corresponding International Standard

In the case of each dry standard preparation, preliminary tests on the dissolved products indicated that:

1 ampoule of the perfringens dry preparation contained 3,080 international units.
1 ampoule of the Vibrion Septique preparation contained 4,200 international units.
1 ampoule of the oedematiens preparation contained 3,800 international units.

[1] **P. Hartley**: "A Simple Laboratory Method for the Desiccation of Serum and other Protein Solutions," *Quart. Bull. Hlth. Org. League of Nations*, Special Number, November, 1936, page 735.

It was desired to issue the perfringens and oedematiens preparations in the form of standard solutions each of which contained 20 international units per c.c., and the Vibrion Septique preparation as a standard solution of which 1 c.c. contained 50 international units. Accordingly, the contents of 1 ampoule of the perfringens preparation were dissolved in 154 c.c., 1 ampoule of the Vibrion Septique preparation in 84 c.c., and 1 ampoule of the oedematiens preparation in 190 c.c. of 66 per cent glycerol-saline.

The potency of these solutions in terms of the corresponding international standard was first determined at Hampstead, and subsequently by Mr. A. T. Glenny at the Wellcome Physiological Research Laboratories at Beckenham, and by Dr. G. F. Petrie at the Lister Institute, Elstree. The results of these comparative tests are summarised below.

A. Assay of the New Perfringens Antitoxin Standard Preparation

1. *By the Mouse Intravenous Method*

Laboratory	Toxin and dose	New standard solution		International standard solution	
		?International units	Proportion of mice surviving	International units	Proportion of mice surviving
Hampstead	Welchpool γ 2·75 mg.	0·22	11/12	0·22	10/12
		0·20	9/12	0·20	9/12
		0·18	3/12	0·18	2/12
		0·16	0/12	0·16	0/12
	N.X. 119 0·4 mg.	0·22	10/10	0·22	10/10
		0·20	10/10	0·20	9/10
		0·18	6/10	0·18	5/10
		0·16	0/10	0·16	0/10
	K. 178–180 0·025 c.c.	0·22	4/4	0·22	4/4
		0·20	3/4	0·20	3/4
		0·18	1/4	0·18	0/4
		0·16	0/4	0·16	0/4
Beckenham	N.X. 119 2·0 mg.	1·1	8/8	1·1	7/8
		1·0	4/8	1·0	4/8
		0·9	0/8	0·9	1/8
Elstree	K. 178–180 0·025 c.c.	0·22	21/21	0·22	17/21
		0·20	19/21	0·20	11/21
		0·18	6/21	0·18	0/21

2. *By the Guinea-Pig Intracutaneous Method*

Laboratory	Toxin and dose	New standard solution ?International units	Reaction	International standard solution International units	Reaction
Hampstead	Welchpool γ 0·15 mg.	0·12	No reaction	0·12	No reaction
		0·11	No reaction	0·11	No reaction
		0·10	No reaction	0·10	No reaction
		0·09	Marked reaction; necrosis	0·09	Marked reaction; necrosis
		0·08	Marked reaction; necrosis	0·08	Marked reaction; necrosis

B. Assay of the New Vibrion Septique Antitoxin Standard Preparation

1. *By the Mouse Intravenous Method*

Laboratory	Toxin and dose	New standard solution		International standard solution	
		?International units	Proportion of mice surviving	International units	Proportion of mice surviving
Hampstead	Coronation 3·3 mg.	1·1	16/21	1·1	16/21
		1·0	6/21	1·0	4/21
		0·9	2/21	0·9	1/21
	V.S.E. II 2·2 mg.	0·55	5/6	0·55	5/6
		0·50	1/6	0·50	2/6
		0·45	0/6	0·45	0/6
	A.V. 204 2·6 mg.	0·55	10/10	0·55	10/10
		0·50	10/10	0·50	10/10
		0·45	4/10	0·45	4/10
		0·40	0/4	0·40	0/4
Beckenham	A.V. 241 3·44 mg.	1·1	15/15	1·1	15/15
		1·0	3/15	1·0	14/15
		0·9	1/15	0·9	3/15
		0·8	0/15	0·8	0/15
Elstree	V.S.E. II 2·1 mg.	0·55	12/12	0·55	12/12
		0·50	9/22	0·50	8/22
		0·45	0/12	0·45	0/12

2. *By the Guinea-Pig Intracutaneous Method*

Laboratory	Toxin and dose	New standard solution ?International units	Reaction	International standard solution International Units	Reaction	Guinea-pig
Hampstead	Coronation 0·8 mg.	0·24	No reaction	0·24	No reaction	A
		0·22	No reaction	0·22	No reaction	
		0·20	Necrosis: 6 × 3 mm.	0·20	Necrosis: 10 × 3 mm.	
		0·18	Necrosis: 10 × 8 mm.	0·18	Necrosis: 12 × 7 mm.	
		0·16	Necrosis: 15 × 10 mm.	0·16	Necrosis: 20 × 7 mm.	
		0·24	No reaction	0·24	No reaction	B
		0·22	No reaction	0·22	No reaction	
		0·20	Necrosis: 3 × 5 mm.	0·20	Necrosis: 7 × 6 mm.	
		0·18	Necrosis: 10 × 10 mm.	0·18	Necrosis: 12 × 10 mm.	
		0·16	Necrosis: 17 × 8 mm.	0·16	Necrosis: 18 × 9 mm.	
Elstree	V.S.E. II 2·1 mg.	0·55	0	0·55	0	2
		0·50	Trace	0·50	Trace	
		0·45	5 × 5 mm.	0·45	5 × 5 mm.	
		0·55	<5 × 5 mm.	0·55	Trace mm.	4
		0·50	5 × 5 mm.	0·50	5 × 5 mm.	
		0·45	6 × 6 mm.	0·45	6 × 6 mm.	

C. Assay of the New Oedematiens Antitoxin Standard Preparation

By the Mouse Intramuscular Method

Laboratory	Toxin and dose	New standard solution		International standard solution	
		? International units	Proportion of mice surviving	International units	Proportion of mice surviving
Hampstead	S 0·38 mg.	0·024	6/6	0·024	6/6
		0·022	6/6	0·022	5/6
		0·020	3/6	0·020	5/6
		0·018	3/6	0·018	4/6
		0·016	1/6	0·016	0/6
	K. II 0·0045 c.c.	0·024	4/4	0·024	3/4
		0·022	3/4	0·022	2/4
		0·020	1/4	0·020	1/4
		0·018	0/4	0·018	0/4
		0·016	0/4	0·016	0/4
	A.E. 200 T. 1 mg.	0·22	4/4	0·22	4/4
		0·20	4/4	0·20	4/4
		0·18	2/4	0·18	3/4
		0·16	1/4	0·16	0/4
Beckenham	A.E. 200 2·67 mg.	1·1	8/10	1·1	10/10
		1·0	1/10	1·0	4/10
		0·9	0/10	0·9	0/10
Elstree	K. II 0·0045 c.c.	0·022	4/12	0·022	8/12
		0·020	0/12	0·020	1/12
		0·018	0/12	0·018	0/12
	K. II 0·0044 c.c. (intravenous)	0·022	9/12	0·022	12/12
		0·020	4/12	0·020	11/12
		0·018	2/12	0·018	5/12

Determination of the Weight of each New Standard Preparation that is Exactly Equal to One International Unit of Antitoxin

A. Perfringens Antitoxin

The weight of the total solids contained in each of nine ampoules was determined and found to be:

0·3510 g.; 0·3505 g.; 0·3504 g.; 0·3468 g.;
0·3492 g.; 0·3471 g.; 0·3477 g.; 0·3468 g.; 0·3486 g.

Mean = 0·34868 g.
σ = 0·00168 g. Coefficient of variation 0·480%
σm = 0·00056 g.

Thus, since each ampoule contains 3,080 international units, 1 *international unit is contained in* $\frac{348 \cdot 68}{3{,}080} = 0 \cdot 1132$ *mg. of the new standard preparation.*

B. Vibrion Septique Antitoxin

The weight of the total solids contained in each of nine ampoules was determined and found to be:

0·4129 g.; 0·4082 g.; 0·4097 g.; 0·4095 g.;
0·4107 g.; 0·4068 g.; 0·4055 g.; 0·4094 g.; 0·4096 g.

Mean = 0·40914 g.
σ = 0·00215 g. Coefficient of variation 0·525%
σm = 0·00072 g.

Thus, since each ampoule contains 4,200 international units, 1 *international unit is contained in* $\frac{409 \cdot 14}{4{,}200} = 0 \cdot 0974$ *mg. of the new standard preparation.*

C. Oedematiens Antitoxin

The weight of the total solids contained in each of nine ampoules was determined and found to be:

0·4324 g.; 0·4336 g.; 0·4313 g.; 0·4330 g.;
0·4306 g.; 0·4320 g.; 0·4304 g.; 0·4301 g.; 0·4296 g.

Mean = 0·43144 g.
σ = 0·00139 g. Coefficient of variation 0·321%
σm = 0·00046 g.

Thus, since each ampoule contains 3,800 international units, 1 *international unit is contained in* $\frac{431 \cdot 44}{3{,}800} = 0 \cdot 1135$ *mg. of the new standard preparation.*

We desire to express our thanks to Mr. A. T. Glenny and Dr. G. F. Petrie for their help and co-operation in this work.

NOTE ON THE COMPLEXITY OF TETANUS TOXIN[1]

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INTRODUCTION

It has recently been shown that estimates of the antitoxic potency of samples of tetanus antitoxin are dependent on the sample of toxin used for the assay (LLEWELLYN SMITH, 1938; SORDELLI, 1938). It has long been known that samples of tetanus toxin may differ from one another qualitatively as well as quantitatively (GLENNY, BARR, ROSS and STEVENS, 1932); but there is little knowledge at present as to the precise nature of the differences in the samples of toxin responsible for the different values assigned to antitoxic sera, when assayed by different toxins. These differences have been ascribed to varying contents of toxoid among different toxin samples, to different slopes of the neutralisation curves, and to the varying firmness (or speed) with which the samples of toxin combine with antitoxin.

There is also, of course, the possibility of the existence in tetanus culture-filtrates of two or more toxic substances, physiologically similar but immunologically distinct. As far as is known no evidence has hitherto been brought for the existence of more than one such tetanus toxin. Though TULLOCH (1919) showed that tetanus antitoxin prepared from type I toxin was more efficacious against type I infections than against those caused by other types, he did not attribute this to differences in the nature of the toxins produced by the different types, but rather to a type-specific reaction between antiserum and (*a*) a leucotoxic substance, (*b*) an anti-phagocytic substance produced by the organisms.

If samples of tetanus toxin contain only a single lethal factor, the toxicities of samples of toxin for mice should be proportional to their toxicities for rabbits or guinea-pigs. FILDES (1929) quotes the relative amounts of toxin required to kill equal weights of a variety of different species of animal. According to his table, the guinea-pig is twice as sensitive as, and the rabbit six times less sensitive than, the mouse to injections of tetanus toxin. There is a footnote to the table, stating that most German authors describe the rabbit as one hundred and fifty times less sensitive than the mouse. More recently FRIEDEMANN (1939) gives, without comment, figures for the ratio of the lethal dose for mice to that for guinea-pigs of tetanus toxin injected

[1] This investigation was completed in August 1939. For various reasons publication of the results has had to be deferred. In the meantime, an important paper by Johs. Ipsen, who has approached some of the questions dealt with in this paper in a somewhat different way, has been published in this *Bulletin* (1940/41, Vol. IX, No. 4, 447 and 452). In so far as the two investigations deal with the same problems, the results now recorded are in general agreement with those reported independently by Ipsen. It is considered that the publication of these results is desirable in view of the practical importance of the problems arising from the complex nature of tetanus toxin.

intracerebrally. Using four toxins made from different strains, he finds that to one toxin rabbits were ten times more resistant than guinea-pigs (weight for weight), whereas to another toxin rabbits were nearly twice as sensitive as guinea-pigs.

It is the purpose of this paper to show that the ratio of the lethal dose for rabbits to that for mice (weight for weight) may vary dramatically and significantly from one sample of toxin to another.

Experimental

Toxin samples. Five samples of toxin were used and these were chosen so as to be of as varied a character as possible. Thus the samples were derived from three different manufacturing laboratories, four of them were produced from different strains, and, of these, at least three strains were of different serological types (one strain had not been typed). Similarly, in three instances the culture medium was different, in all five the growth period differed, four of the toxins were precipitated with ammonium sulphate while the fifth was preserved in the liquid condition by dilution with an equal volume of glycerol. Of the four precipitated toxins, three were preserved dry and the other was subsequently redissolved in 50 per cent glycerol-saline. These differences in the preparation of the five toxin samples are summarised in Table 1 (columns 2–6).

The samples of toxin also differed in their biological reactions, e.g. in their combining properties with antitoxins of different quality, in the slopes of the reaction curves with antitoxin, in the ease with which the antitoxin complexes dissociated on dilution, in their content of toxoid and in their immunising efficiency. The differences in combining properties are illustrated by differences in the potency ratio of two samples of antitoxin (4101 and the 1st British standard antitoxin) when the different toxins are used for the comparative assay (Table 1, columns 11 and 12). The qualitative differences between these two antitoxin samples have already been discussed (Llewellyn Smith, 1938). The slopes of the reaction curves were calculated, by the method of Gaddum, from the proportion of mice dying in groups injected with mixtures consisting of varying doses of serum 4101 plus a fixed dose of the toxin under test, under comparable conditions (Table 1, column 9). Some of the figures are taken from Table 6 of the paper already cited, by Llewellyn Smith. The ease of dissociation was estimated by observing the shift in the L+ or Lo dose when mixtures of antitoxin and the toxin under test were diluted 5, 10 or 100 fold. No quantitative figure has been assigned to the dissociability as the tests were not all performed under strictly comparable conditions (Table 1, column 10). The number of lethal doses of a toxin which are neutralised by a fixed dose of antitoxin is inversely proportional to its content of toxoid. Figures for the ratio of the L+/5 (*v.* International Standard Antitoxin) to the L.D. 50 for mice injected subcutaneously are given in Table 1, column 7. The immunising efficiency was estimated by injecting groups of six rabbits with a series of injections of toxin-antitoxin mixture (see later). After seven injections

Table 1 PROPERTIES OF THE FIVE SAMPLES OF TETANUS TOXIN

1	2	3	4	5	6	7	8	9	10	11	12
	Differences in Preparation					Differences in biological properties					
								Combining properties with antitoxin			
No. of toxin	Source of toxin	Strain and type	Culture medium	Growth period (days)	How preserved	No. of mouse lethal doses in L+/5 dose	Antigenic efficiency (geo. mean Antitoxin produced)	Slope of reaction curve (*b*)	Dissociation on dilution	Potency ratio 4101/1st British Standard	
										mice	guinea-pig
509	Elstree	Strain 279, Tulloch type 1	Horse muscle, Witte peptone 1% glucose	11	Liquid toxin diluted with equal volume glycerol	750	—	143	Very slight dissociation	52	79·5
C. 35	Copenhagen	Strain 3, Tulloch type 2	Beef muscle, Riedel peptone	8	Ppted. with $(NH_4)_2SO_4$ and dried	2,100	—	73·3	Very slight dissociation	77	100
A.W. 226 B.P.	Beckenham	Strain 12, Tulloch type 3	Horse muscle, Difco peptone	3	Ppted. with $(NH_4)_2SO_4$ and dried	1,900	Bad antigen 0·08 Intl. units per c.c.	183	Slight dissociation	75	—
A.W. 226 H.P.	Beckenham	Strain 12, Tulloch type 3	Horse muscle, Difco peptone	12	Ppted. with $(NH_4)_2SO_4$ and dried	320	Good antigen 0·75 Intl. units per c.c.	43	No dissociation	58	—
A.W. 179	Beckenham	Strain 34, type unknown	Horse muscle, Witte peptone 1% glucose	9	Ppted. with $(NH_4)SO_4$ and dry toxin dissolved in 50% glycerol-saline	256	—	65	Considerable dissociation	64	74

the animals were bled and their sera tested for antitoxin. The geometric means of the antitoxin produced by the two groups are given in Table 1, column 8.

Determination of the Average Lethal Dose for Rabbits, Mice and Guinea-Pigs

Animals. The mice used were of uniform stock and weighed 18–20 g.; the guinea-pigs, also of uniform stock, weighed as nearly 350 g. as possible; the rabbits used were of mixed breeds and of various weights, but they were randomised with regard to weight and breed among the five toxins, and the results of the experiments gave no indication that the lethal dose per 100 g. body weight was influenced by the breed or weight.

Table 2

Determination of L.D. 50 of Tetanus Toxin 509 in Rabbits, Mice and Guinea-Pigs

Toxin and test animal	Date	No. of animals in group	Dose injected c.c per 100 grams.	Day of death
Toxin 509; rabbits i.v.	11.5	1	0·01	6
	11.5	1	0·001	S
	16.5	4	0·015	3, 6, S, S
	16.5	4	0·0075	S, S, S, S
	22.5	4	0·02	3, 3, 4, 4
Toxin 509; mice s.c.	17.12	3	0·0005	3, 3, 3
	17.12	3	0·00025	4, 4, 6
	29.12	3	0·00025	3, 4, 5
	29.12	3	0·000125	S, S, S
	29.12	3	0·000063	S, S, S
Toxin 509; guinea-pigs s.c.	31.12	1	0·000114	3
	31.12	1	0·000057	3½
	31.12	1	0·000028	S
	10.1	5	0·000071	3, 4, 4½, 4½, 5
	10.1	5	0·000048	6, 7, 8, 8½, S
	10.1	5	0·000031	S, S, S, S, S

S denotes survival for 9 days or more.

Method of estimating the L.D. 50. Preliminary determinations of the lethal dose were carried out using one to three animals per dose and doses spaced at two-fold (or sometimes even wider) intervals. Having thus obtained the approximate lethal dose, groups of three to five animals were injected with each of two or more doses spaced at 1·2, 1·5 or 2 fold intervals. The animals were observed for nine days, and the L.D. 50 calculated by the method of Behrens (1929). The mice and guinea-pigs received the toxin subcutaneously, the rabbits intravenously; approximate determinations of

the lethal dose for mice by the intravenous route were made, however, to make certain that the subcutaneous and intravenous lethal doses were parallel, and this was found to be so, the intravenous lethal dose being about double that by the subcutaneous route.

Two typical determinations of the L.D. 50 of tetanus toxins for mice, guinea-pigs and rabbits are given in Tables 2 and 3 and the results are summarised in Table 4.

Table 3

DETERMINATION OF L.D. 50 OF TETANUS TOXIN A.W. 179 IN RABBITS, MICE AND GUINEA-PIGS

Toxin and test animal	Date	No. of animals in group	Dose injected (c.c. per 100 g. body weight)	Time to die (days)
Toxin A.W. 179; rabbits i.v.	11.5	1	0·03	1
	11.5	1	0·003	1½
	16.5	1	0·0015	3
	16.5	1	0·00075	5.
	22.5	1	0·0003	5
	30.5	1	0·00015	S
	30.5	1	0·000075	S
	8.6	4	0·0003	S, S, S, S
	8.6	4	0·00015	S, S, S, S
	14.6	5	0·0005	4, 4, 4, 4, 4
Toxin A.W. 179; mice s.c.	17.12	3	0·002	3, 3, 3
	17.12	3	0·001	4, 4, 4
	17.12	3	0·0005	S, S, S
	17.12	3	0·00025	S, S, S
	29.12	3	0·00083	4, 5, 5
	29.12	3	0·0007	5, 5½, 6½
	29.12	3	0·00057	6, S, S
	29.12	3	0·00048	S, S, S
Toxin A.W. 179; guinea-pigs s.c.	6.1	1	0·00069	4
	6.1	1	0·00034	6½
	6.1	1	0·00017	S
	24.1	5	0·00017	4½, 4½, 5½, 5½, 6
	24.1	5	0·000114	6, 6, 8, 8, 8
	24.1	5	0·000077	S, S, S, S, S

From Table 4 it will be seen that while the toxicities of the five samples of toxin for mice were approximately parallel to those for guinea-pigs, as shown by the approximate constancy of the ratio in column 9, the toxicities for rabbits were by no means parallel to the toxicities for mice and guinea-pigs (see columns 6 and 7). Thus, while it requires eighty-five times as much of toxin 509 to kill 1 g. of rabbit as to kill 1 g. of mouse, with toxin A.W. 179 the lethal dose per gram of rabbit is only just one half the value for mice; there is a 150 fold difference in the relative sensitivity of the two species to these two samples of toxin. Where the sensitivities of rabbits

and guinea-pigs were compared, the extreme divergence observed in the relative sensitivity was 108 fold.

This is a strong indication that two (or more) toxic factors occur in samples of tetanus toxin and that mice and guinea-pigs are relatively more sensitive to one of these, rabbits being relatively more sensitive to the other. On this hypothesis, toxin 509 would contain a larger proportion of the factor to which mice are relatively more sensitive, and toxin A.W. 179 a larger proportion of the factor to which rabbits are relatively more sensitive.

Table 4

LETHAL DOSES FOR MICE, RABBITS AND GUINEA-PIGS OF FIVE SAMPLES OF TETANUS TOXIN.

No. of Toxin	L.D. 50 (mice) per 100 gm. body weight		L.D. 50 (rabbits) per 100 gm. body weight i.v.	L.D. 50 (guinea-pigs) per 100 gm. body weight s.c.	Relative resistance of animals (weight for weight)		
	i.v. (approximate values only)	s.c.			rabbit: mouse	rabbit: guinea-pig	mouse: guinea-pig
509	0·00025 c.c.	0·0002 c.c. (15)	0·017 c.c. (14)	0·000048 c.c. (18)	85	354	4·2
C. 35	0·004 mg.	0·0025 mg. (27)	0·20 mg. (16)	0·00077 mg. (19)	80	260	3·25
A.W. 226 B.P.	0·003 mg.	0·002 mg. (35)	0·05 mg. (13)	0·00072 mg. (13)	25	70	2·8
A.W. 226 H.P.	0·015 mg.	0·005 mg. (25)	0·02 mg. (16)	0·0008 mg. (27)	4	25	6·25
A.W. 179	0·00075 c.c.	0·000625 c.c. (24)	0·00035 c.c. (20)	0·00012 c.c. (18)	0·56	2·9	5·2

NOTE.—The figures in brackets denote the number of animals used for the determination.

From an examination of Tables 1 and 4, an attempt was made to correlate changes in the mouse-rabbit lethal dose ratio with other properties of the toxin. There was no observable connection between the toxicity ratio for rabbits and mice and the culture medium or growth period, the strain or type, the content of toxoid, the slope of the reaction curve with antitoxin or any other property of the toxin.

ATTEMPT TO DETECT ANTIGENIC DIFFERENCES IN THE TWO LETHAL FACTORS

The two toxins A.W. 226 B.P. and A.W. 226 H.P. were chosen as antigens, partly because they were the only ones available in sufficient quantity, and partly because the similarity in the method of preparation of these two toxins eliminated many possible causes of variation in behaviour.

Groups of six rabbits were immunised with a series of doses of the toxin under test combined with antitoxin No. 4101. This was done in order to provide an antigen that was not too toxic and at the same time to avoid the possible alteration in the antigenic nature of the toxin that is stated to follow detoxification by formaldehyde (SORDELLI; personal communication).

The animals were given two courses of immunisation with an intervening rest period of three months. The doses of the two toxins given were always proportional to their L+ doses, so that each group of rabbits received the

Table 5

IMMUNISATION OF RABBITS, SIX IN EACH GROUP, WITH TETANUS TOXIN-ANTITOXIN MIXTURES

Toxins used: A.W. 226 B.P. and A.W. 226 H.P. *Antitoxin used*: 4101.

Date	Mixture injected (AW. 226. B.P. Series)		A.T. produced, Geo. Mean I.U./c.c.	Mixture injected (A.W. 226. H.P. Series)		A.T. produced, Geo. Mean I.U./c.c.
	Weight of toxin, mg.	Vol. of A.T. 4101 c.c.		Weight of toxin, mg.	Vol. of A.T. 4101 c.c.	
2.2.39	3·5	1/60		1·25	1/60	
9.2.39	3·8	1/60		1·35	1/60	
16.2.39	4·2	1/60		1·5	1/60	
23.2.39	8·4	1/30		3·0	1/30	
2.3.39	16·8	1/15		6·0	1/15	
9.3.39	Animals bled		0·08	Animals bled		0·75
9.3.39	33·6	2/15		12·0	2/15	
13.6.39	33·6	1/2		12·0	2/15	
17.6.39	33·6	1/4		12·0	1/15	
20.6.39	33·6	1/8		12·0	1/30	
23.6.39	33·6	1/16		12·0	1/60	
27.6.39	33·6	1/32		12·0	1/120	
7.7.39	Animals bled out		1·2	Animals bled out		3·8

same number of "combining units" of antigen, although this was contained in different weights of the two dry preparations. (The doses combining with 0·2 international unit of antitoxin were 0·85 mg. for toxin B.P. and 0·31 mg. for toxin H.P. so that, measured in grams of dry material injected, the group immunised with toxin B.P. received two and a half times as much as that immunised with toxin H.P.)

After the first course of immunisation sample bleedings were taken, and it was immediately apparent that the group that had received toxin B.P. had not responded nearly as well as that which had received toxin H.P. The geometric mean of the first group was less than one tenth of that produced by the second group (Table 1, column 8). Moreover, at the second course of immunisation the rabbits immunised with H.P. toxin could withstand greater numbers of lethal doses without showing symptoms of tetanus than those immunised with B.P. It is thus clear that the immunising

efficiencies of the two toxins are neither proportional to the weight of dry toxin administered nor to the number of "combining units" of antigen.

Table 6

ANTITOXIC TITRES OF SERA OF RABBITS IMMUNISED WITH TETANUS TOXINS A.W. 226 B.P. AND A.W. 226 H.P., WHEN ASSAYED BY MEANS OF THE HOMOLOGOUS AND HETEROLOGOUS TOXINS

Assays Performed in Mice

Serum No.	Toxin used for Immunisation	Antitoxin titre when assayed with—		
		toxin C35	AW 226 BP	AW 226 HP
343	A.W. 226 B.P.	0·6 ± 100%	0·62 ± 20%	0·68 ± 20%
348		1·2 ± 100%	1·5 ± 20%	1·5 ± 20%
349		2·2 ± 100%	2·9 ± 20%	2·6 ± 20%
350		1·2 ± 100%	1·1 ± 20%	1·3 ± 20%
351		1·4 ± 100%	1·2 ± 20%	1·5 ± 20%
Geo. mean		1·2	1·29	1·39
344	A.W. 226 H.P.	4·0 ± 100%	4·3 ± 20%	4·0 ± 20%
345		4·0 ± 100%	6·2 ± 20%	5·5 ± 20%
352		3·2 ± 100%	5·5 ± 20%	5·7 ± 20%
353		2·8 ± 100%	2·7 ± 20%	2·9 ± 20%
354		2·8 ± 100%	5·0 ± 20%	4·6 ± 20%
355		8·0 ± 100%	6·6 ± 20%	6·0 ± 20%
Geo. mean		3·8	4·85	4·64

Table 7

ANTITOXIC TITRE OF SERA OF RABBITS IMMUNISED WITH TETANUS TOXINS A.W. 226 B.P. AND A.W. 226 H.P. WHEN ASSAYED BY MEANS OF THE HOMOLOGOUS AND HETEROLOGOUS TOXINS

Assays Performed in Rabbits

Serum No.	Toxin used for immunisation	Antitoxic titre when assayed with—	
		AW 226 BP	AW 226 HP.
348	A.W. 226 B.P.	1·6 ± 50%	1·6 ± 50%
349	A.W. 226 B.P.	3·0 ± 50%	3·0 ± 50%
345	A.W. 226 H.P.	6·0 ± 50%	6·0 ± 50%
355	A.W. 226 H.P.	7·0 ± 50%	7·0 ± 50%

At the end of the second course of immunisation, the rabbits were chloroformed and bled out and the sera were titrated individually for antitoxin content in comparison with the international standard antitoxin, using three toxins; viz. C. 35, A.W. 226 B.P. and A.W. 226 H.P. The titrations were performed in mice, using three animals per serum dose, and doses spaced at 20 per cent intervals. The mixtures were allowed to combine for one hour at room temperature and injected subcutaneously. Assays at 1·5 fold intervals and using only one animal per dose were also performed in rabbits. The results are summarised in Tables 6 and 7.

It will be seen from the tables that there is no evidence of greater neutralisation by the homologous than by the heterologous toxin, when the assay is performed in either mice or rabbits. There is thus no evidence that the hypothetical multiple lethal factors differ antigenically.

Summary

1. The ratio of the lethal dose of tetanus toxin for rabbits to that for mice may vary as much as 150 fold from one sample of toxin to another, suggesting the presence of multiple lethal factors in the toxic filtrates.

2. The presence of different proportions of the multiple toxins in different filtrates could not be correlated with other differences in the filtrates.

3. No evidence was found of antigenic differences in the multiple toxins

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OBSERVATIONS ON THE VARIABLE INTERACTIONS OF TETANUS TOXINS AND ANTITOXINS

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Introduction

Comparative observations on the titration of samples of tetanus antitoxin have brought to light the following paradox. Serologists are agreed that the assay of tetanus antitoxin can be carried out with a high degree of precision when only a few test-animals, whether guinea-pigs or mice, are used: in other words, the slope of the neutralisation curve is a steep one, and yet, if a sample of antitoxin is distributed to a number of laboratories for assay, the values assigned to it may vary widely. The cause of the discrepancies is as yet unknown. Hartley (unpublished: *B.H.O.L.N.*,[1] p. 740) showed as early as 1927 that there are qualitative differences between the American and the British Standards, and that tetanus toxin-antitoxin mixtures dissociate on dilution, the degree varying with the sample of antitoxin. In 1934 the Permanent Commission on Biological Standardisation adopted a plan of co-operative investigation which was extended in 1936 (Jensen, 1936) and 1937, and in 1938 a full analysis of the data obtained in these extensive studies was published (*B.H.O.L.N.*, 1938, pp. 684–688 and 713–784). This publication also contains an account of studies on the problem by M. Llewellyn Smith, Sordelli, and Ipsen and Rostock, and deals also with certain administrative aspects which arise, such as the range of variation in the unitage of serum samples intended for human use that can be regarded as acceptable by the control authorities. A Committee presided over by Dr. P. Hartley and working for the British Pharmacopoeia Commission carried out some additional potency determinations and showed that in 99 per cent of the assays the range of variation lies between 73·5 per cent and 136 per cent of the true value (*B.H.O.L.N.*, p. 776), a conclusion which agrees with the estimate of the Copenhagen workers (*B.H.O.L.N.*, p. 729).

For some years past the present writer had met with discrepancies when determining the L+/5 dose of freshly prepared toxins by tests in guinea-pigs against 1/5 I.U. of the British and American standards. An occasion for making detailed observations on the subject presented itself when he was invited by Dr. P. Hartley, in March 1937, to take part in comparative tests of the values of the five sera that had been chosen by the Permanent Commission as the basis of their International inquiry. The following account of the work subsequently done by the writer describes the lines on which an effort was made to throw light on the problem.

Mode of Preparation and General Characters of the Glycerinated Test-Toxins Used in the Work

Mode of preparation

Glycerinated test-toxins have been used during the past nineteen years for titrating routine batches of tetanus antitoxin produced in the Serum Department of the Lister Institute (MacConkey, 1924).

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[1] These initials, when used in this paper, signify *The Bulletin of the Health Organization of the League of Nations* Vol. 7, No. 5, October, 1938.

The broth medium for the cultures from which the test-toxins used in the work were prepared was the same for all. The medium consisted of a horse-meat infusion made with tap-water to which was added 1 per cent Witte peptone and 1 per cent dextrose; sodium chloride was not added and there was no layer of coagulated muscle at the bottom of the culture flask. The initial pH was 7·4–7·6 and the final pH was 6·8–7·0. The temperature of incubation was about 35° C., and the period of incubation varied from 4 to 21 days. The strain used was in every instance No. 279, a Type I (Tulloch) strain, received from the National Collection of Type Cultures, the Lister Institute, London.

The freshly filtered broth culture was mixed with an equal volume of glycerol.

A note on the individual test-toxins

Details of the seven test-toxins exclusively used in the work will be found in Table 1. Of these No. 509 is a routine test-toxin which is stored in the cold room, and No. 509C is a more recent sample from the stock bottle which had been stored in the cold room from the date of its preparation in May 1935 until December 1935, since when it has been kept constantly in a refrigerator at — 10° C. Test-toxin No. 540 was prepared in April 1937 by the method of VANÍČEK (1930) as follows: the original crude toxin was concentrated about three times, and to some extent purified, by dialysis in a cellophane bag against one or two lots of glycerol until its glycerol content was found to be 50 per cent. Test-toxin 550 was prepared in two parts: 550*a* from a culture grown for 4 days before filtration, and 550*b* from the same batch of culture but grown for 12 days. Test-toxin 559 was treated similarly but the growth in some of the flasks was terminated at 4, 12 and 21 days respectively in order to obtain the test-toxins 559*a*, 559*b* and 559*c*. The constants of the dry toxin C35, which was prepared at the State Serum Institute, Copenhagen, by precipitating a toxic filtrate with ammonium sulphate, have been added for comparison.

The constitution of the test-toxins

The number of L.D.50's in the L+/5 dose of the nine toxins included in Table 1 varies from 120 to 305 when estimated in the guinea-pig as compared with 865 to 2420 when estimated in the mouse. Partial conversion into toxoid had taken place during incubation of the cultures from which 550*b* and 559*c* were prepared, the one a 12-day and the other a 21-day test-toxin.

The suitability of the test-toxins for titration tests

(*a*) *The stability of glycerinated test-toxins.* These preparations were treated with scrupulous care by storing them in the cold room, except for an hour or two at room temperature when in use. Exposure to even moderate daylight was avoided as much as possible. Glycerinated tetanus test-toxins, when properly stored and handled in this way, are reasonably

Table 1

Summary of the Data Concerning the Seven Glycerinated Test-Toxins Used in the Work

Identification No.	Test-toxin				Guinea-pig (s.c.)(1)			Mouse (i.p.)(2)		
	Date of preparation	Strain from which derived	Source	Days' growth of corresponding culture	L.D. 50 (c.c.)	L+/5 dose (c.c.)	No. of L.D.50 in L+/5 dose	L.D.50 (c.c.)	L+/5 dose	No. of L.D. 50 in L+/5 dose
509	22/5/1935	No. 279 (Tulloch, type 1) N.C.T.C., London	Lister Institute, Elstree	11	1/11000	1/50	220	1/42500	1/35	1,210
509C	22/5/1935	No. 279 (Tulloch, type 1) N.C.T.C., London	Lister Institute, Elstree	11	1/20000	1/65·5	305	1/50000	1/41	1,220
540	27/4/1937	No. 279 (Tulloch, type 1) N.C.T.C., London	Lister Institute, Elstree	5	1/42500	1/255	170	1/140000	1/97·5	1,440
550*a* (3)	24/1/1938	No. 279 (Tulloch, type 1) N.C.T.C., London	Lister Institute, Elstree	4	1/20000	1/75	270	1/75000	1/31	2,420
550*b* (3)	1/2/1938	No. 279 (Tulloch, type 1) N.C.T.C., London	Lister Institute, Elstree	12	1/6000	1/50	120	1/27500	1/28	980
559*a* (3)	19/9/1938	No. 279 (Tulloch, type 1) N.C.T.C., London	Lister Institute, Elstree	4	1/15000	1/64	230	1/44000	1/28	1,570
559*b* (3)	27/9/1938	No. 279 (Tulloch, type 1) N.C.T.C., London	Lister Institute, Elstree	12	1/15000	1/68	220	1/48000	1/31	1,550
559*c* (3)	7/10/1938	No. 279 (Tulloch, type 1) N.C.T.C., London	Lister Institute, Elstree	21	1/9000	1/52	170	1/22500	1/26	865
C35	—	T67 (Tulloch, type 2) N.C.T.C., London	State Serum Institute, Copenhagen	8	0·003 mg.	0·7 mg.	230	0·00077 mg.	1·1 mg.	1,430

Note: (1) s.c. = toxin injected subcutaneously.
(2) i.p. = toxin injected intraperitoneally.
(3) The brackets in the first column indicate that the enclosed test-toxins were derived from the same batch of culture.

stable. Thus, when this work was begun in March 1937, the L+/5 dose of T509 was 1/50 c.c. for the guinea-pig; in March 1938 it had changed to 1/48 c.c., and in October 1938 to 1/46 c.c.[1]

(*b*) *The character of the neutralisation curve.* The experience of the writer is that by the use of the glycerinated test-toxin 509 the titre of samples of tetanus antitoxin can be estimated in the mouse and the guinea-pig within 4 per cent, when a group of five animals is used for each serum dose. Toxin 458, which was prepared in July 1930, and T509 prepared in May 1935, were examined by M. LLEWELLYN SMITH from the point of view of the steepness of slope of the neutralisation curve of mixtures containing them (*B.H.O.L.N.*, pp. 748, 760). Her experiments indicated that T458 had a real tendency to produce steeper slopes than the Copenhagen test-toxin C35, and that in mouse tests T509 gave slopes which were as steep as, or even steeper than, those produced by T458. She noted that in both guinea-pigs and mice the dose-response curve of the assay when T509 was used was exceedingly steep; dose-intervals of 2 per cent gave appreciable changes in the mortality-rate.

THE METHODS EMPLOYED THROUGHOUT THE WORK

The L+/5 dose (= 1/5 I.U. or 1/10 U.S.A. unit) of each toxin was separately determined in the guinea-pig and in the mouse by means of the British Standard Serum. The time of contact of the mixtures, and of all toxin-antitoxin mixtures tested, was 3/4 hour at 20°–21° C. For guinea-pigs as the test-animal the reaction took place in a volume of 4 c.c., and for mice in a volume of 1 c.c. The dose was injected subcutaneously into guinea-pigs weighing 340–380 g., and intraperitoneally into mice weighing 17–22 g. The rate of absorption from the latter route of injection may be considered to be intermediate between that from the intravenous and the subcutaneous routes; it has the advantage over these of simplicity and the absence of leakage.

The mice were kept singly in sets of ten boxes arranged for ready observation, and the final record for both mice and guinea-pigs was made on the morning of the 6th day. The test-animals were observed at regular intervals, 4-hourly when possible, and the degree of the toxic symptoms and the time of death were noted by means of conventional signs. This procedure, though at times laborious, defined with precision the stage and progress of the illness and the death-time, and thus checked the accuracy of the tests. Irregularities within the limits of a test, such as lack of correlation between the serum dose administered and the degree of severity of the symptoms, were most unusual. The method of record-keeping adopted made it possible for an accurate estimate of the titre of a serum to be made when only a few test-animals were used for each dose of serum—an important consideration in view of the large number of tests that were undertaken.

[1] Later, the requisite amount of toxin was measured in the cold room, and at the present time (January, 1943) the test-toxin and other standard reagents are kept in a refrigerator in the testing laboratory, with the result that the test-dose has not perceptibly altered during the past year.

Variations in the Potency Ratio, Derived from the Titres as Estimated by the Glycerinated Test-Toxin 509 in the Mouse and the Guinea-Pig, of (1) Routine and Standard Sera from a Variety of Sources; (2) Routine and Experimental Sera Prepared in Horses at Elstree; and (3) Experimental Sera Prepared in Guinea-Pigs

Variations in the potency ratios of a number of miscellaneous sera

Table 2 gives the results of assaying in the mouse and the guinea-pig eighteen preparations of tetanus antitoxin from widely scattered sources. The test-toxin used was 509 and the test-dose used was the L+/5 dose as estimated in both the mouse and the guinea-pig in terms of the British Standard Tetanus Antitoxin. The mouse units of each serum preparation have been expressed as a percentage of the guinea-pig units; and the data in the table are arranged in accordance with this potency ratio.[1]

The serum samples include the five sera chosen for the International inquiry and standard sera from three countries, namely, from Washington, Copenhagen and London. Most of the other samples are from the United Kingdom; two are from the United States, and there is one sample from France. Of the eighteen preparations thirteen are natural sera, most of them taken from a single horse, and five are concentrated products.

With one exception, the percentage potency ratios fall below 100; they range from 151 for a non-avid serum, to 58 for Serum No. 922, a serum was which obtained from a "well-prepared" horse, and which is characterised by its ability to combine rapidly with tetanus toxin (Ramon, Lemétayer and Pirosky, 1937).[2] A very large pool, namely, the mixed serum from three hundred bleedings taken from twenty-eight horses gave a value of 71/100, and this doubtless represents an average result. The titres of the sera examined range from 9 to 9,600 mouse units and from 10 to 14,000 guinea-pig units per c.c. There is no correlation between antitoxin content and the mouse/guinea-pig potency ratio. This is apparent when the three sera which give a 66 per cent ratio are compared. Again, the Elstree laboratory standard No. 2 and Serum No. 1 of the International test give similar ratios, and yet these sera show a 130-fold difference in potency.

Variations in the potency ratio of serum samples from horses immunised at Elstree

A high proportion of the serum preparations detailed in Table 2 were presumably derived from "well-prepared" horses. An attempt was made to obtain samples of serum with a fairly high unitage from a horse which had not received preparatory doses of toxoid followed by a rest interval before the first immunising course. This animal proved to be refractory when immunised with an efficient antigen, and even after two courses of toxoid it produced less than 50 I.U. per c.c.; tests in mice of the samples

[1] In this paper the phrase "mouse units" or "mouse titre" is meant to indicate the potency obtained when the assays were carried out in mice; and "guinea-pig units" or "guinea-pig titre" when the assays were carried out in guinea-pigs.

[2] I am obliged to Mr. Glenny for the sample of non-avid serum and to Prof. Ramon for a sample of his flocculating serum, No. 922.

Table 2

SHOWING THE RESULTS OF TITRATING EIGHTEEN SERUM PREPARATIONS FROM VARIOUS SOURCES IN THE MOUSE AND THE GUINEA-PIG. THE TEST-TOXIN USED WAS T509, AND THE L+/5 DOSE OF THIS WAS ESTIMATED IN BOTH MOUSE AND GUINEA-PIG IN TERMS OF THE BRITISH STANDARD SERUM. THE PERCENTAGE POTENCY RATIOS HAVE BEEN ARRANGED IN ORDER

Serum preparation	Source	Natural or concentrated and whether from single horse or pool	Mouse units per c.c.	G.-pig units per c.c.	$\frac{\text{Mouse titre}}{\text{G.-pig titre}}$ %
Non-avid serum	Received from Mr. Glenny: FC3010K	N	590	390	**151**
British Standard Serum	Nat. Inst. Med. Research, London	N	10	10	**100**
International Standard	State Serum Institute, Copenhagen	N	9·52	10·26	**93**
No. IV	Permanent Commission Biological Standardisation	N: 2 sera mixed	900	1,040	**87**
Elstree Lab. Stand. No. 2 (glycerinated)	Single horse serum	Concentrated	9	11	**82**
No. I	As No. IV	N: single	1,140	1,430	**80**
Elstree Pool	Pool 14 horses, bled 24/4/37	Concentrated	700	900	**78**
		Glycerinated			
"P"	Prepared in U.S.A.	Concentrated	5,800	7,500	**77**
Elstree Pool	Pool 14 horses, bled 24/4/37	N	910	1,260	**72**
Elstree Pool A_1–D_3	Pool 300 bleedings from 28 horses	N	850	1,200	**71**
Pool "R.G.W."	Pool of 4 horses 2/12/1938	N	2,500	3,500	**71**
Avid serum	Received from Mr. Glenny: F3234 B	N: single	1,120	1,570	**71**
No. II	As No. IV	N: single	1,330	1,930	**69**
L 23787	Pepsin-digested antitoxin: U.S.A.	Concentrated	9,600	14,000	**69**
No. III	As No. IV	N: single	4,290	6,470	**66**
U.S.A. Standard Serum	Nat. Institute of Health, Washington	N	9·6	13·4	**66**
No. V	As No. IV	Concentrated pool	8,780	13,260	**66**
"Hampstead" No. 922	Received from Dr. P. Hartley	N	56	89	**63**
	Received from Prof. Ramon	N: single	290	500	**58**

taken at this time indicated a considerably higher potency than that derived from guinea-pig tests; the percentage ratio was >340/100, the most discrepant inverse ratio met with in the whole of the work.

Two horses were given a single dose of 50 c.c. of tetanus toxoid with the addition of 2 per cent alum; a sample taken 36 days later was not assayed with precision but the titre estimated in mice was found to be higher than that which was derived from tests in guinea-pigs.

The results obtained from two routine horses "S" and "D" are as follows. The mouse titre of the first sample of "S" was higher than the guinea-pig titre, whereas samples from "D", a horse which responded better than "S", yielded ratios below 100; it is possible that an earlier sample would have exhibited the reverse ratio.

These results point to the following conclusion. The serum of horses which receive a single dose of the immunising antigen, or which are immunised without arranging for a preparatory dose and interval of rest, or which are sampled after a short course of immunisation, tend to give the reverse potency ratio, that is, the mouse titre is higher than the guinea-pig titre.

Variations in the potency ratio of antitoxic sera obtained by immunising guinea-pigs

The conclusions to be drawn from the observations under this heading are the same as those which are based on the results obtained in horses. Thus, at an early stage of the immunisation the mouse titres of the samples were higher than those given by guinea-pig tests; as the immunisation proceeded the titres reached equality, and finally the mouse titres of the samples progressively decreased relatively to the corresponding guinea-pig titres.

Discrepancies in the Titres of a Number of Routine and Standard Sera from a Variety of Sources, when Assayed in the Mouse and the Guinea-Pig by Means of Four Glycerinated Test-Toxins, Including 509

The variations in the mouse/guinea-pig potency ratio of which an account has been given in the last Section were derived from tests in which the routine glycerinated test-toxin 509 was used. An obvious extension of this work was to compare the results with those obtained by means of other test-toxins stabilised by glycerol but dissimilar in respect of the time of growth of the toxin culture. Such a comparison would give, it was hoped, some indication whether the period of growth is an important factor in determining the discrepant results. Test-toxin 509 originated from a culture of 11 days' growth. A concentrated glycerinated toxin No. 540 was also available; it had been obtained by filtering a culture of 5 days' growth. These toxins were prepared from the same tetanus strain and by essentially the same methods. Nevertheless, steps were taken to secure strictly comparable data by preparing toxins that were similar to 509 and 540 but were derived from the same batch of culture; this was effected by filtering part

of a batch after 4 days' growth (550*a*) and the remainder after 12 days' growth (550*b*). After the addition of glycerol the test-dose (L+/5) of each test-toxin was found by reference to the British standard serum.

Discrepancies in the titres and in the potency ratios when the assays were made by two "short-growth" and two "long-growth" test-toxins

Thirteen serum preparations were examined and all of them are included in the list which is given in Table 2. The results are assembled in Table 3, and the sera are arranged in order of the 509 potency ratios. The features of this Table to which attention is directed are briefly as follows:—

(1) There is a tendency for the potency ratios to decrease or, in other words, for the difference between the mouse and the guinea-pig titre to increase from left to right of the table, that is, towards the columns which contain the results given by the short-growth toxins.

(2) The discrepancy between the maximum and the minimum titres, irrespective of whether these were estimated in mice or guinea-pigs, is rather greater in the lower half of the table. To cite an example, Serum No. IV of the International test is the second on the list and has 4 out of 8 titres equal; the extreme values are 850 and 1,040 units. In contrast with this result the potency values of the last serum sample in the table, No. 922, range from 290 to 1,000 units.

(3) The mouse titres, as estimated by the short-growth test-toxins, are higher than those estimated by the long-growth test-toxins. Thus twelve out of thirteen mouse titres given by 550*a* are higher than those given by 550*b*. Eight out of thirteen mouse titres given by 540 are higher than those given by 509. Combining the two groups we obtain 20 out of 26 cases (77 per cent) in which the mouse titres are higher when estimated by the short-growth toxins than by the long-growth toxins. Again, all of the thirteen 550*a* guinea-pig titres are higher than the 550*b* titres; and twelve out of the thirteen 540 guinea-pig titres are higher than the 509 guinea-pig titres, that is, 25 out of 26 (96 per cent) of the titres estimated in the guinea-pig by the short-growth test-toxins are higher than the titres estimated by the long-growth toxins.

(4) Scrutiny of the results reveals a tendency for the sera to separate into groups; these are indicated in the last column of the table, and the best examples are the groups B and C. The potency ratios derived from the mouse and the guinea-pig titres of the four sera in B show a marked similarity and so also do the two sera in C. Group B includes two natural sera from individual horses and two concentrated pools. There is no correlation between unitage of the sera in each group and the potency ratios. Thus the Elstree laboratory standard in Group A shows a fair agreement with No. 1 serum of the International test and yet there is a 100-fold difference in titre between them; the mean units are 13 and 1315. The significance of the similarities in the potency ratios which have the effect of grouping the samples will be discussed later, but it would seem reasonable to suggest that they indicate an identity in neutralising action, in respect of the four test-toxins, of the antitoxin which each contains.

Table 3

COMPARES THE TITRES OF THIRTEEN SERUM PREPARATIONS FROM VARIOUS SOURCES WHICH WERE ESTIMATED IN THE MOUSE AND THE GUINEA-PIG BY FOUR GLYCERINATED TEST-TOXINS. THE SERA HAVE BEEN ARRANGED IN ORDER OF THE PERCENTAGE POTENCY RATIOS DERIVED FROM THE MOUSE AND GUINEA-PIG TITRES AS ESTIMATED BY THE GLYCERINATED TEST-TOXIN 509

Serum preparation	TEST-TOXIN												Serum Group
	509 (11-day)			550*b* (12-day)			550*a* (4-day)			540 (5-day)			
	M. units per c.c.	G.P. units per c.c.	M. titre[2] / G.P. titre %	M. units per c.c.	G.P. units per c.c.	M. titre / G.P. titre %	M. units per c.c.	G.P. units per c.c.	M. titre / G.P. titre %	M. units per c.c.	G.P. units per c.c.	M. titre / G.P. titre %	
British Standard Serum ..	10	10	**100**	10	10	**100**	10	10	**100**	10	10	**100**	
International Standard (Copenhagen)	9·5	10·3	**93**	8·4	11	**76**	9·4	14	**67**	9·4	16·8	**56**	
No. IV (P.C.B.S.[1])	900	1,040	**87**	850	850	**100**	850	950	**89**	850	1,000	**85**	
Elstree Lab. Standard (glycerinated)	9	11	**82**	10	15	**67**	12	20	**60**	10	16	**63**	
No. I (P.C.B.S.)	1,140	1,430	**80**	1,000	1,400	**71**	1,200	1,600	**75**	1,100	1,650	**67**	A
Elstree Pool, concentrated glycerinated ..	700	900	**78**	780	1,250	**62**	950	1,650	**58**	800	1,650	**48**	
"P"	5,800	7,500	**77**	5,000	6,500	**77**	5,500	8,400	**65**	5,000	9,000	**56**	
No. II (P.C.B.S.)	1,330	1,930	**69**	1,400	2,500	**56**	1,550	3,200	**48**	1,550	3,600	**43**	
L 23787	9,600	14,000	**69**	9,500	18,000	**53**	12,000	26,000	**46**	10,000	24,500	**41**	B
No. III (P.C.B.S.)	4,290	6,470	**66**	4,700	8,500	**55**	5,800	12,000	**48**	5,500	12,000	**46**	
No. V (P.C.B.S.)	8,780	13,260	**66**	8,800	16,000	**55**	10,000	22,000	**45**	9,500	20,000	**48**	
U.S. Standard Serum ..	9·6	13·4	**66**	7	11	**64**	8·5	12·5	**68**	7	13·5	**52**	
"Hampstead"	56	89	**63**	68	111	**61**	88	160	**55**	78	175	**45**	C
No. 922 (G. Ramon) ..	290	500	**58**	400	650	**61**	480	850	**56**	460	1,000	**46**	

[1] The initials signify "Permanent Commission on Biological Standardisation."

[2] M = mouse; G.P. = guinea-pig.

Correspondence in proportional values between two of the sera examined

Table 4 has been abstracted from an unpublished table which dealt in a similar manner with the whole of the data in Table 3, and it is admittedly the best illustration of correspondence in value between any pair of the sera. The mouse titre, as estimated by 509, of each serum listed in the complete table was chosen as the basal value and was assessed at 100, and the remaining seven titres were converted into a proportional value. Table 4 gives the results for two of the three sera in Group A, namely the Elstree laboratory standard and the concentrated pool of 24/4/1937. The close similarity of the mouse values can hardly be explained by chance; the correspondence between the guinea-pig values is less striking. The significance of the correspondence of the mouse values is perhaps indicated by comparison with those yielded by an experimental horse ''4,'' which was immunised with a 4-day toxin (559*a*) and which was sampled at an interval of one month. The two samples from this horse were titrated in mice and guinea-pigs by 509 and by the three homologous test-toxins 559*a* (4-day), 559*b* (12-day), and 559*c* (21-day); the mouse titre given by 509 was chosen as the base line. The various mouse titres of the two samples of ''4'' exhibit a remarkable approximation. It seems permissible to assume that the virtual identity of the mouse values of the Elstree laboratory standard and the concentrated pool are due to the same cause, namely, identity of the neutralising properties of the antitoxins due, perhaps, to a chance similarity of the immunising antigens. The point may again be stressed that there is no correlation between the antitoxin content of the sera and the potency ratios. Thus the mean units of the sera compared in Table 4 are 13 and 1085 (1 : 83) and the mean units of the samples of ''4'' are 944 and 2350 (1 : 2·5).

The range of variation of the mouse and the guinea-pig titres respectively

An analysis has been made of the available data in respect of the range of variation of the mouse and the guinea-pig titres. The results are briefly as follows. In nine out of the thirteen sera the percentage difference between the mouse titres, as estimated by means of the short-growth test-toxins 550*a* and 540, is smaller than the percentage difference between the mouse titres, as estimated by means of the long-growth test-toxins 509 and 550*b*. A similar analysis of the guinea-pig titres points in the same direction. Thus in eleven out of thirteen serum preparations the range of variation is smaller when short-growth test-toxins are used. If we combine the two sets of data we find 20 out of 26 cases (77 per cent) in which the mouse and guinea-pig titres show less deviation when estimated by short-growth test-toxins than by long-growth test-toxins. Again, in eleven out of thirteen serum preparations which were titrated by 550*a* (4-day) and 550*b* (12-day) test-toxins *derived from the same culture-growth* the mouse titres show more restricted deviations than the guinea-pig titres. An attempt to interpret the facts and conclusions in the whole of this section will be made when the available evidence has been completed.

Table 4

COMPARES THE PROPORTIONAL TITRATION VALUES DERIVED FROM THE THREE GLYCERINATED TEST-TOXINS, 550*a*, 550*b* AND 540 OF (1) THE BRITISH STANDARD SERUM, (2) THE ELSTREE LABORATORY STANDARD, AND (3) THE CONCENTRATED AND GLYCERINATED POOL OF 24/4/1937, IN TERMS OF THE TITRES ESTIMATED IN MICE BY T509 AND ASSESSED AS 100 UNITS

The corresponding proportional values derived from the three glycerinated test-toxins, 559*a*, 559*b* and 559*c* for two samples of the horse "4" are added for comparison.

	Proportional mouse-titre values				Proportional guinea-pig-titre values			
	Test-toxin				Test-toxin			
No. of Test-Toxin	509	550*b*	550*a*	540	509	550*b*	550*a*	540
Days' growth of corresponding culture	11	12	4	5	11	12	4	5
SERUM SAMPLE								
British Standard Serum	100	100	100	100	100	100	100	100
Elstree Lab. Standard Serum	100	111	133	111	122	167	222	178
Concentrated glycerinated Pool 24/4/1937	100	111	135	114	129	179	236	236
	Test-toxin				Test-toxin			
No. of Test-Toxin	509	559*a*	559*b*	559*c*	509	559*a*	559*b*	559*c*
Days' growth of corresponding culture	11	4	12	21	11	4	12	21
SERUM SAMPLE								
Horse "4," 10/3/1939	100	173	164	155	91	209	236	246
Horse "4," 11/4/1939	100	170	163	155	104	226	255	218

Discrepancies in the Titres of Samples of Tetanus Antitoxin when Estimated by Homologous and Heterologous Test-Toxins

The work described in the foregoing sections has dealt with: (1) the potency ratios derived from the mouse and guinea-pig titres of eighteen serum preparations collected from widely different sources and tested against the glycerinated test-toxin 509; and (2) discrepancies in the titres of a number of routine and standard sera from a variety of sources when assayed in the mouse and the guinea-pig by means of four glycerinated test-toxins prepared at Elstree, including 509. All of these titrations may be regarded as having been carried out in accordance with the normal routine procedure, that is, the test-toxins employed for all of them were "heterologous," in the sense that the batch of culture from which they were prepared was different from that which yielded the toxins or toxoids that were used for the production of the sera in question. The next line of investigation clearly lay in comparing the titres of serum samples when estimated by homologous and heterologous test-toxins; the terms, homologous and heterologous antitoxins, bear the same implication as they do when applied to toxins.

Accordingly, two horses named "Four" and "Twelve" were immunised with a 4-day *toxoid* (550*a*) and a 12-day *toxoid* (550*b*) respectively; these toxoids were prepared from the same batch of culture. Three consecutive samples of serum at 3 or 4 weeks' interval were taken and assayed by means of the two homologous glycerinated test-toxins 550*a* and 550*b* and the two heterologous test-toxins, namely, 540, a 5-day glycerinated test-toxin and the routine 509, an 11-day test-toxin. The crucial point of this set of observations is that *the test-toxins* 550*a and* 550*b were homologous, that is, they originated from the same batch of culture as the toxoids used for the immunisation of the horses.* It will be noted that the four test-toxins just mentioned are precisely those which were used for titrating the thirteen serum preparations from various sources of which the last section gives an account.

Later, arrangements were made on a similar plan for immunising three horses, "4," "12," and "21," with a specially prepared set of *toxins*, namely, 559*a* (4-day), 559*b* (12-day) and 559*c* (21-day), and all of them from the same batch of culture: a glycerinated test-toxin was made at each of the three stages of growth. The three horses received two preparatory doses of the corresponding toxoid to obviate the risk of immunising them with toxin before an adequate antitoxin immunity was reached. Samples of serum were taken at a month's interval and were titrated against the three homologous glycerinated test-toxins and the heterologous test-toxin 509; the L+/5 dose of the 550 and the 559 test-toxins was determined in mice and guinea-pigs by reference to the British standard serum (Table 1).

The immunising doses and dose-intervals and the time of taking the blood-samples were exactly the same for "Four" and "Twelve"; and likewise for "4," "12" and "21," although there was no correspondence between the two groups of horses; for example, the interval between the first immunising dose and the first sample of serum was 26 days for the two horses and 60 days for the group of three horses.

Table 5

RECORDS THE RESULTS OF TITRATING SERUM SAMPLES FROM HORSES IMMUNISED WITH A 4-DAY (550*a*) AND A 12-DAY (550*b*) TOXOID DERIVED FROM THE SAME BATCH OF CULTURE; THE SAMPLES WERE TITRATED BY THE TWO HOMOLOGOUS AND BY TWO HETEROLOGOUS TEST-TOXINS, ALL OF WHICH WERE STABILISED BY THE ADDITION OF GLYCEROL

Serum sample	Test-toxin							
	540 (5-day)		550*a* (4-day)		550*b* (12-day)		509 (11-day)	
	M. titre / G.P. titre	% ratio	M. titre / G.P. titre	% ratio	M. titre / G.P. titre	% ratio	M. titre / G.P. titre	% ratio
Horse "FOUR": 30/5/1938	650 / 550	*118* / *100*	750 / 650	*115* / *100*	700 / 650	*108* / *100*	420 / 450	93 / 100
Horse "FOUR": 20/6/1938	750 / 800	94 / 100	1,100 / 950	*116* / *100*	900 / 1,100	82 / 100	700 / 450	*155* / *100*
Horse "FOUR": 25/7/1938	650 / 800	81 / 100	850 / 950	89 / 100	750 / 800	94 / 100	450 / 450	100 / 100
Horse "TWELVE": 29/5/1938	600 / 550	*109* / *100*	850 / 650	*131* / *100*	750 / 650	*115* / *100*	450 / 375	*120* / *100*
Horse "TWELVE": 21/6/1938	1,000 / 1,300	77 / 100	1,150 / 1,300	88 / 100	1,100 / 1,300	85 / 100	650 / 650	100 / 100
Horse "TWELVE": 26/7/1938	900 / 1,200	75 / 100	1,000 / 1,600	63 / 100	1,050 / 1,300	81 / 100	650 / 700	93 / 100

NOTE: (1) The results enclosed within the vertical dotted lines are those estimated by the homologous test-toxins 550*a* and 550*b*; the results within the double dotted lines are those estimated by the strictly homologous test-toxin.
(2) M = mouse; G.P. = guinea-pig.
(3) Where the mouse titre is higher than the guinea-pig titre, the corresponding percentage potency ratio is shown in italics.

The results of assays of serum samples from the five experimental horses

The results have been brought together in Table 5, in which the mouse and the guinea-pig titres of samples from the horses "FOUR" and "TWELVE" are recorded; Table 6 contains similarly arranged data concerning the horses "4," "12" and "21." Both tables confirm the conclusion already reached that samples of serum taken in the early stage of the immunisation tend to show the reverse potency ratio, a result which signifies that the mouse titre is higher than the guinea-pig titre. Table 5 is a good example of this relation: the ratios derived from each of the four test-toxins used, whether homologous or heterologous, illustrate it. The potency ratios tend to decrease the further the immunisation is carried, that is, the difference between the mouse and the guinea-pig titre increases.

Proportional potency values of the samples from these horses in terms of the mouse titres estimated by the test-toxin 509

Tables 7 and 8 show at a glance for each of the various samples that are detailed in Tables 5 and 6 the proportional value, relative to the 509 mouse titre, of the antitoxin content of each sample as estimated by the homologous

Table 6

RECORDS THE RESULTS OF TITRATING SERUM SAMPLES FROM HORSES IMMUNISED WITH A 4-DAY, A 12-DAY AND A 21-DAY TOXIN (559*a*, 559*b* AND 559*c*), DERIVED FROM THE SAME BATCH OF CULTURE; THE SAMPLES WERE ASSAYED BY THE THREE HOMOLOGOUS TEST-TOXINS AND BY THE HETEROLOGOUS TEST-TOXIN 509, ALL OF WHICH WERE STABILISED BY THE ADDITION OF GLYCEROL

HORSE "4"

[Immunised with 4-day toxin (559*a*)]

No. of Test-toxin	559a		559b		559c		509	
Days' growth of corresponding culture	(4)		(12)		(21)		(11)	
Date of serum sample	M. titre / G.P. titre	% ratio	M. titre / G.P. titre	% ratio	M. titre / G.P. titre	% ratio	M. titre / G.P. titre	% ratio
3/1/1939	—	—	—	—	—	—	16 / 6	*267* / *100*
10/3/1939	950 / 1,150	83 / 100	900 / 1,300	69 / 100	850 / 1,350	63 / 100	550 / 500	*110* / *100*
11/4/1939	2,300 / 3,050	75 / 100	2,200 / 3,450	64 / 100	2,100 / 2,950	71 / 100	1,350 / 1,400	96 / 100

See Notes to Table on next page.

Table 6—*continued*

HORSE "12"

[Immunised with 12-day toxin (559*b*)]

No. of test-toxin	**559a**		**559b**		**559c**		509	
Days' growth of corresponding culture	(4)		(12)		(21)		(11)	
Date of serum sample	M. titre / G.P. titre	% ratio	M. titre / G.P. titre	% ratio	M. titre / G.P. titre	% ratio	M. titre / G.P. titre	% ratio
3/1/1939	—	—	—	—	—	—	32 / 20	*160* / *100*
10/3/1939	1,050 / 1,250	84 / 100	1,100 / 1,350	81 / 100	1,000 / 1,450	69 / 100	700 / 700	100 / 100
11/4/1939	2,100 / 2,500	84 / 100	1,800 / 2,700	67 / 100	2,000 / 2,900	69 / 100	1,100 / 1,300	85 / 100

HORSE "21"

[Immunised with 21-day toxin (559*c*)]

No. of test-toxin	**559a**		**559b**		**559c**		509	
Days' growth of corresponding culture	(4)		(12)		(21)		(11)	
Date of serum sample	M. titre / G.P. titre	% ratio	M. titre / G.P. titre	% ratio	M. titre / G.P. titre	% ratio	M. titre / G.P. titre	% ratio
3/1/1939	—	—	—	—	—	—	20 / 12	*167* / *100*
10/3/1939	1,350 / 1,900	71 / 100	1,150 / 1,800	64 / 100	1,150 / 1,900	61 / 100	750 / 750	100 / 100
11/4/1939	2,200 / 3,100	71 / 100	1,900 / 3,100	61 / 100	1,900 / 3,100	61 / 100	1,050 / 1,200	88 / 100

NOTE: (1) The homologous test-toxins in the heading of the Table are indicated by heavy type.
(2) Where the mouse titre is higher than the guinea-pig titre, the corresponding percentage potency ratio is shown in italics.
(3) The results enclosed within the dotted lines are those estimated by the strictly homologous test-toxin.
(4) M = mouse; G.P. = guinea-pig.

Table 7

COMPARES THE PROPORTIONAL TITRATION VALUES DERIVED FROM THE THREE GLYCERINATED TEST-TOXINS, 550*a*, 550*b* AND 540 OF (1) THE BRITISH STANDARD SERUM, (2) THE SAMPLE OF 25/7/1938 OF HORSE "FOUR" AND (3) THE SAMPLE OF 26/7/1938 OF HORSE "TWELVE" IN TERMS OF THE TITRES ESTIMATED IN MICE BY T509 AND ASSESSED AS 100 UNITS

	Proportional mouse-titre values				Proportional guinea-pig-titre values			
	Test-toxin				Test-toxin			
No. of Test-Toxin	509	550*b*	550*a*	540	509	550*b*	550*a*	540
Days' growth of corresponding culture	11	12	4	5	11	12	4	5
SERUM SAMPLE								
British Standard Serum	100	100	100	100	100	100	100	100
Horse "FOUR," 25/7/1938	100	*167*	**189**	144	100	*178*	**211**	178
Horse "TWELVE," 26/7/1938	100	**162**	*154*	138	108	**200**	*246*	185

NOTE: The figures shown in heavy type represent the values obtained by using the strictly homologous test-toxin.
The figures shown in italics represent the values obtained by using the homologous test-toxin.
The remaining figures represent the values obtained by using the heterologous test-toxin.

Table 8

COMPARES THE PROPORTIONAL TITRATION VALUES DERIVED FROM THE THREE GLYCERINATED TEST-TOXINS, 559*a*, 559*b* AND 559*c* OF TWO SAMPLES FROM THE HORSES "4," "12" AND "21" IN TERMS OF THE TITRES ESTIMATED IN MICE BY T509 AND ASSESSED AS 100 UNITS

	Proportional mouse-titre values				Proportional guinea-pig-titre values			
	Test-toxin				Test-toxin			
No. of Test-Toxin	559*a*	559*b*	559*c*	509	559*a*	559*b*	559*c*	509
Days' growth of corresponding culture	4	12	21	11	4	12	21	11
SERUM SAMPLE								
British Standard Serum	**100**	100	100	100	**100**	100	100	100
Horse "4," 10/3/1939	**173**	*164*	*155*	100	**209**	*236*	*246*	91
Horse "4," 11/4/1939	**170**	*163*	*155*	100	**226**	*255*	*218*	104
Horse "12," 10/3/1939	*150*	**157**	*143*	100	*179*	**193**	*207*	100
Horse "12," 11/4/1939	*191*	**164**	*182*	100	*227*	**245**	*264*	118
Horse "21," 10/3/1939	*180*	*153*	**153**	100	*253*	*240*	**253**	100
Horse "21," 11/4/1939	*209*	*181*	**181**	100	*295*	*295*	**295**	114

NOTE: The figures shown in heavy type represent the values obtained by using the strictly homologous test-toxin.
The figures shown in italics represent the values obtained by using the homologous test-toxin.
The remaining figures represent the values obtained by using the heterologous test-toxin.

and the heterologous test-toxins; the guinea-pig titres are also included in the comparison so that the results of all the tests on each sample of serum are referred to the corresponding 509 mouse titre, to which a value of 100 has been assigned. This analysis justifies the following statements:—

(1) All of the homologous titres in Tables 5 and 6, as estimated in both the mouse and the guinea-pig, are higher than those given by the heterologous toxin 509.

(2) The samples from the horses "FOUR" and "4," which were immunised with a 4-day toxoid and toxin respectively, show that the highest mouse value corresponds to the strictly homologous test-toxin, that is, the 4-day one.

For both samples of "4" the mouse values diminish regularly in the sequence of the 4-day, 12-day and 21-day homologous test-toxin; and the proportional values of the two samples show a remarkable similarity and are indeed virtually identical, although the potency of the second is two and a half times that of the first sample. The guinea-pig values for "4" do not repeat this satisfactory correspondence.

(3) The strictly homologous mouse value of the horse "TWELVE" is higher than the value given by the homologous 4-day test-toxin, and both of these are higher than the two heterologous values. This statement is in agreement with the corresponding findings for the horse "FOUR."

The homologous mouse and guinea-pig values for the second sample of "12" contrast with the corresponding sample of "4," the horse immunised with the 4-day toxin, in being consistently higher than those of the first sample.

(4) All of the homologous values of the horse "21" agree with those of "12" in respect of the consistently higher values of the second sample as compared with the first sample.

The identity of the mouse values given by the homologous 12-day and 21-day test-toxins for the two samples of the horse "21," which was immunised with a 21-day toxin, is notable. Table 6 gives the actual titres of these samples and it will be seen that of four pairs of comparable titres as estimated in the mouse and the guinea-pig by the homologous 12-day and 21-day test-toxins, three pairs are identical and the other differs by not more than 5 per cent. Three of the mouse/guinea-pig potency ratios are identical and the fourth is not significantly different, although the titre of the second is higher than that of the first sample.

A comparison between the mouse and the guinea-pig titres of routine and experimental serum preparations when estimated by means of both homologous and heterologous test-toxins

The data in Tables 3, 5, 6, 7 and 8 were so arranged as to make possible a direct comparison, on the one hand, between the titres of the thirteen sera from varying sources (cf. Table 3) as estimated by the four heterologous test-toxins, 550*a* (4-day), 540 (5-day), 509 (11-day) and 550*b* (12-day); on the other hand, the titres of the samples "FOUR" and "TWELVE" as estimated by the same toxins but now regarded as the homologous 550*a* and 550*b* test-toxins and the heterologous 540 and 509 test-toxins (Tables 5

and 7). The results are summarised in Table 9 and they show that 96 per cent of the titres as estimated by the homologous test-toxins 550*a* and 550*b* are higher than the titres as estimated by the similar but heterologous test-toxins 540 and 509. From the data of Group B, which serves as a control, it is seen that, when all test-toxins are heterologous, only 56 per cent of the titres as estimated by 550*a* and 550*b* are higher than those estimated by 540 and 509; the difference is not significant.

Group C in Table 9 refers to the titres of samples from the experimental horses "4," "12" and "21" as detailed in Tables 6 and 8. When the samples were tested by the three homologous test-toxins the titres were,

Table 9

ANALYSIS OF THE RESULTS OBTAINED BY TITRATING SAMPLES OF SERUM FROM THE FIVE EXPERIMENTAL HORSES "FOUR" AND "TWELVE" AND "4," "12" AND "21" BY MEANS OF HOMOLOGOUS AND HETEROLOGOUS TEST-TOXINS; THE RESULTS OF GROUP B CONTROL THOSE OF GROUP A

Group of serum samples	Source	Result of analysis
A	Horses "FOUR" and "TWELVE" (Cf. Table 5)	23 out of 24 titres (96%) estimated by the homologous test-toxins 550*a* and 550*b* are greater than those estimated by the heterologous test-toxins 540 and 509
B (Control to Group A)	13 sera from various sources (Cf. Table 3)	29 out of 52 titres (56%) estimated by the heterologous test-toxins 550*a* and 550*b* are approximately equal to those estimated by the heterologous test-toxins 540 and 509
C	Horses "4," "12" and "21" (Cf. Table 6)	36 out of 36 titres (100%) estimated by the homologous test-toxins 559*a*, 559*b*, and 559*c* are greater than those estimated by the heterologous test-toxin 509

without exception, higher than the titres as estimated by the heterologous test-toxin 509.

These results carry considerable weight in support of the theory which it is proposed to formulate in explanation of the discrepant interactions between tetanus toxins and antitoxins.

CERTAIN VARIABLE FACTORS WHICH MAY ACCOUNT FOR THE DISCREPANT TITRES OF SAMPLES OF TETANUS ANTITOXIN WHEN DIFFERENT TEST-TOXINS ARE USED

The toxoid content of the test-toxins

The data have been examined from this point of view but have not yielded evidence of any correlation between toxoid content of the test-toxins and

the resulting titres of antitoxins as estimated in the mouse and the guinea-pig. Thus the mean titres of samples tested in both the mouse and the guinea-pig by the short-growth test-toxins 550*a* and 540 give good correspondence, although toxin 540 has a considerably higher content of toxoid than toxin 550*a* (Table 1). The same conclusion can be drawn from the titres given by the long-growth test-toxins 550*b* and 509.

Again, the mean titres of samples tested in both the mouse and the guinea-pig by the homologous test-toxins 559*b* and 559*c* are virtually identical, although 559*c* is much more toxoided than 559*b*. Lastly, the toxoid content of 559*b* and 509 is similar and yet the 559*b* titres of the homologous serum samples are much higher than those given by 509.

The varying avidity of tetanus antitoxins

The term "avidity" appears to have been defined in various ways. Non-avid sera are said to be characterised by (1) dissociation of the toxin-antitoxin complex on dilution *in vitro* and also *in vivo*, so that dissociation is more likely to occur in the guinea-pig, owing to its larger blood volume, than in the mouse, (2) slow rate of combination of the complex *in vitro*, and (3) the production of flat neutralisation curves (Llewellyn Smith, *B.H.O.L.N.*, p. 751).

Reference to Table 2 shows that the list of serum preparations in the table is headed by a sample of a non-avid serum for which the percentage potency ratio of the mouse and the guinea-pig titres is 151/100, a result which is consistent with the explanation that the mouse titre is higher than the guinea-pig titre because of dissociation of the complex in the larger animal. If this explanation is applied to a sample of serum at the other end of the list, for example, to the avid serum of Glenny and to the Hampstead serum and to Ramon's No. 922, we are forced to admit that these sera are more easily dissociable in the mouse than in the guinea-pig, so that a relatively larger dose must be used in order to neutralise the test-dose of toxin—a conclusion which seems paradoxical. The problem of avidity will be re-examined in the discussion of the data that have been brought together in this paper.

The mode of preparation of the test-toxins, that is, whether by ammonium sulphate precipitation or by the addition of glycerol

The most important data hitherto published on the discrepancies that occur in testing samples of tetanus antitoxin are those which were collected by the Permanent Commission on Biological Standardisation. The Commission distributed five serum samples for assay in nine Institutes (*B.H.O.L.N.*, p. 776), and the results of all the tests are summarised in a table (pp. 780, 781). The present author has compared the results of his own titrations with those of the other laboratories recorded in this table and finds that, on the average, the titres of the five sera as estimated by him in the mouse are 16 per cent lower than the figures derived from the assays of the other laboratories; and similarly his guinea-pig titres are about 10 per cent higher than those of the other Institutes (Table 10). The mouse titres of the other laboratories are on the whole lower than the corresponding guinea-pig titres.

Table 10

COMPARES THE TITRES AND PERCENTAGE POTENCY RATIOS OF THE FIVE TEST-SERA EXAMINED IN THE INTERNATIONAL INQUIRY AS ESTIMATED BY FOUR GLYCERINATED TEST-TOXINS AND BY TOXINS PRECIPITATED BY AMMONIUM SULPHATE

	A			B			C			D			E		
No. of Test-toxin	T509 (11-day, glycerinated)			T550*b* (12-day, glycerinated)			T550*a* (4-day, glycerinated)			T540 (5-day, glycerinated)			Sulphated test-toxins of other labs.		
	Mouse I.U. per c.c.	G.P. I.U. per c.c.	M. titre / G.P. titre %	Mouse I.U. per c.c.	G.P. I.U. per c.c.	M. titre / G.P. titre %	Mouse I.U. per c.c.	G.P. I.U. per c.c.	M. titre / G.P. titre %	Mouse I.U. per c.c.	G.P. I.U. per c.c.	M. titre / G.P. titre %	Mouse I.U.* per c.c.	G.P. I.U.* per c.c.	M. titre / G.P. titre %
SERUM SAMPLE															
I	1,140	1,430	**80**	1,000	1,400	**71**	1,200	1,600	**75**	1,100	1,650	**67**	1,230	1,224	**100**
II	1,330	1,930	**69**	1,400	2,500	**56**	1,550	3,200	**48**	1,550	3,600	**43**	1,570	1,771	**89**
III	4,290	6,470	**66**	4,700	8,500	**55**	5,800	12,000	**48**	5,500	12,000	**46**	5,040	6,214	**81**
IV	900	1,040	**87**	850	850	**100**	850	950	**89**	850	1,000	**85**	984	917	**107**
V	8,780	13,260	**66**	8,800	16,000	**55**	10,000	22,000	**45**	9,500	20,000	**48**	10,660	12,119	**88**

* The mouse values in this Section of the Table represent the mean of 10 mouse titres reported by the other laboratories.
The guinea-pig values in this Section of the Table represent the mean of 8 guinea-pig titres reported by the other laboratories.
M. = Mouse. G.P. = Guinea-pig.

The closer approximation to equality of the mouse and guinea-pig titres recorded by the other laboratories is clearly indicated by the percentage potency ratios in Section E of Table 10 as compared with the corresponding Elstree ratios.

The Elstree titres included in the Commission's Table were arrived at by means of the glycerinated test-toxin 509. Table 10 compares the titres of the five sera when tested by four glycerinated test-toxins (Sections A, B, C and D) and by eight test-toxins used by the other laboratories, the test-dose of which was based on one or other of the three standard sera (Section E). For each pair of estimations given in the table the mouse units are expressed as a percentage of the guinea-pig units. The resulting potency ratios are very similar for samples 2, 3 and 5, whereas samples 1 and 4 give a higher percentage figure throughout the Elstree tests (Sections A–D). This division of the five sera into two groups is confirmed by the data in Section E, which represent the means of the titres obtained in the other laboratories. Most of the estimates recorded by these laboratories were obtained by the use of dry test-toxins prepared by precipitating a broth toxin with ammonium sulphate (Ipsen, 1940/41), so that the comparison would seem to be essentially one which is drawn between the results given by glycerinated test-toxins and by dry sulphated test-toxins.

It would appear that the Copenhagen sulphated test-toxin C35 and similar test-toxins are more easily neutralised by samples of tetanus antitoxin in mice than the glycerinated test-toxins and at the same time less easily neutralised in guinea-pigs; and it may be asked whether the precipitation process brings about the loss of a toxic factor for the mouse which is not readily neutralised by antitoxin but which is preserved in the glycerinated test-toxins, or whether the precipitated toxin produces a toxin-antitoxin complex which is more easily dissociated in the tissues of the guinea-pig, so that relatively more antitoxin is needed to effect neutralisation. On the other hand, it is open to argue that the explanation of the difference between the results given by the glycerinated and the dry toxins is due to some alteration of the toxin molecules caused by the action of the glycerol. The stabilising action of glycerol on enzymes and toxins does not support this view and, moreover, the high neutralising activity of the homologous sera from the experimental horses for the appropriate glycerinated test-toxins shows that the specific molecules in these toxins combine well with antitoxin molecules produced by immunisation with a routine preparation of tetanus toxoid or toxin.

The circumstance that the Elstree results were based solely on the British standard serum whereas the results in Section E of Table 10 were derived from the use of the International standard, the U.S.A. standard and the British standard does not appear to give a clue to the differences in unitage of the various sera. Thus the Commission's Table shows that the guinea-pig titres of Laboratory C (C35: International standard) agree well with the Elstree guinea-pig titres, whereas the mouse titres of Laboratory C are about 20 per cent higher than those given by a test-dose of the glycerinated test-toxin 509 estimated by the British standard serum.

Summary of the Results

A summary of the results obtained in this work follows; it provides a basis for the attempt that is made in the subsequent discussion to formulate a theory by which they may be interpreted.

(1) The titres of samples of tetanus antitoxin, as estimated in the mouse or the guinea-pig by heterologous[1] test-toxins derived from short-growth cultures, are higher than the corresponding titres, as estimated by heterologous test-toxins derived from long-growth cultures.

(2) The titres of samples of tetanus antitoxin, when estimated by heterologous "short-growth" test-toxins, show less deviation than those estimated by heterologous "long-growth" test-toxins; this statement applies to tests in both the mouse and the guinea-pig.

(3) When the titres of samples of tetanus antitoxin were estimated in both the mouse and the guinea-pig by a heterologous 4-day and 12-day test-toxin *derived from the same batch of culture* the differences between the mouse titres were smaller than those between the guinea-pig titres.

(4) The varying toxoid content of the glycerinated test-toxins that were used for the assays does not furnish a clue to the discrepant results.

(5) The divergence between the mouse and the guinea-pig titres of samples of tetanus antitoxin, as estimated by four glycerinated test-toxins, is greater than that which is apparent in the results given by the test-toxin C35 and other dry sulphated test-toxins; thus the Elstree mouse titres for the five International sera assayed on behalf of the Permanent Commission on Biological Standardisation are, on the average, about 16 per cent lower and the guinea-pig titres about 10 per cent higher than those obtained in the other participating laboratories.

(6) When the test-toxin is homologous with respect to the antitoxins under test, the factor of differences in the time of growth of the culture from which the test-toxin was derived has much less influence on the resulting titres than when the test-toxin is heterologous: the homologous factor is the dominant one.

(7) Of the titres of samples of antitoxin estimated by the homologous test-toxins 550*a* and 550*b*, 96 per cent are higher than those given by the similar but heterologous test-toxins 540 and 509, whereas in tests where all four toxins are heterologous in respect of the sera under test only 56 per cent of the titres estimated by 550*a* and 550*b* are higher than those estimated by 540 and 509.

Similarly, for horses immunised with a 4-, 12- and 21-day toxin 100 per cent of the titres of the serum samples estimated by the homologous test-toxins are higher than those given by the heterologous test-toxin 509.

(8) The considerable range of variation in the titres of sera assayed by the glycerinated test-toxins prepared at Elstree cannot be ascribed to the use of different strains or serological types of *Cl. tetani* nor to variations in

[1] Note: The term "heterologous" means that the test-toxins were prepared from a culture batch which did not at the same time provide the immunising antigen for the horses that yielded the samples of antitoxin under test. The term "homologous" means that both the test-toxin and the immunising antigen for the horses that yielded the antitoxins under test were derived from the same batch of culture.

the mode of preparation of the culture medium, because the strain used throughout this work was the same, namely, No. 279, a Type 1 (Tulloch) culture, and the technique of preparing the broth cultures from which the toxins were made was the same throughout.

(9) When a number of serum preparations from well-immunised horses were assayed in both mice and guinea-pigs by means of the L+/5 dose of the heterologous glycerinated test-toxin 509 as determined for each test animal by the British standard serum, and when the mouse titre was expressed as a percentage of the corresponding guinea-pig titre, the resulting percentage potency ratios, instead of being invariably equal to 100 per cent as for the British standard, were found to vary from 93 per cent to 58 per cent. There is no correlation between the potency ratios of the sera examined and their antitoxin content.

(10) The mouse/guinea-pig potency ratios derived from assays by heterologous short-growth test-toxins tend to be smaller than those derived from assays by heterologous long-growth test-toxins, that is, the mouse titres are lower in relation to the guinea-pig titres.

(11) When a number of sera were assayed by two heterologous short-growth and two heterologous long-growth test-toxins and the resulting mouse/guinea-pig potency ratios were arranged in order, a number of groups were distinguishable in which the ratios showed a close correspondence; the antitoxin content of the sera composing each group varied widely.

(12) A comparison of the mouse/guinea-pig potency ratios of the five International sera tested on behalf of the Permanent Commission on Biological Standardisation shows that the mouse and guinea-pig titres as estimated by means of dry sulphated test-toxins approximate more closely than those derived from tests in which the titrations were effected by means of four glycerinated test-toxins. The explanation of this difference may be that the precipitation process brings about loss of a toxic factor for the mouse. All of the toxins in this series of tests were heterologous.

(13) The mouse/guinea-pig potency ratios of sera from prepared and well-immunised horses varies from about 40 per cent to 100 per cent: an expression of the fact that the mouse titre is lower than the corresponding guinea-pig titre. On the other hand, the reverse ratio—with the mouse titre higher than the guinea-pig titre—is characteristic of the serum samples from unprepared horses and from prepared horses taken at an early stage of the immunisation and tested by means of both homologous and heterologous test-toxins.

DISCUSSION AND INTERPRETATION OF THE RESULTS

The discrepant results of assaying samples of tetanus antitoxin.

Consideration of the foregoing statements leads to the conclusion that crude tetanus toxins are not homogeneous solutions of toxin and toxoid molecules. The argument in support of this view is based on the circumstance that the whole of the work presented in this paper rests on a com-

parison of the potency values that are assignable to a particular serum by the use of different test-toxins; and that the titre of a sample of antitoxin in relation to a particular test-toxin measures their mutual neutralising affinities in a reacting mixture, not only *in vitro* but perhaps also *in vivo*. The fact that the titres of samples of tetanus antitoxin, as estimated in the mouse or the guinea-pig by heterologous short-growth test-toxins, are higher than the corresponding titres, as estimated by heterologous long-growth test-toxins, indicates that the toxin and the toxoid molecules in the short-growth toxins possess greater chemical and antigenic uniformity than those in the long-growth toxins. It is reasonable to suppose that in the early stages of the culture-growth a high proportion of the toxin and toxoid molecules are of the "normal" or "primary" type, but that as a result of some biochemical action on the labile "primary" molecules during continued incubation of the culture a change is effected in the specific binding groups of a proportion of them that is of such a nature and degree as to modify them in an immunological sense. The altered specific character of the "secondary" molecules has an important consequence for, corresponding to the primary and secondary antigens, primary and secondary antitoxins will be formed. In the view of the present writer the logical sequence in analysing the problem of discrepancies in assays of tetanus antitoxins is to consider (1) the chemical and antigenic heterogeneity of the crude toxins or toxoids that are used for immunising horses, (2) the production, as a result, of varying mixtures of the corresponding antitoxins in samples of the serum, and (3) the degree of heterogeneity of the test-toxin that is used for titrating these samples: the antigenic composition of this toxin may be quite dissimilar to that of the immunising antigen.

The finding that the titres as estimated by heterologous short-growth test-toxins vary within narrower limits than those estimated by long-growth test-toxins is consistent with the theory of the complex nature of crude tetanus toxins when these are harvested late. Moreover, whatever interpretation may be put upon the origin of the groups that are distinguishable in a number of sera when assayed by four glycerinated test-toxins, two of them derived from short-growth cultures and two from long-growth cultures, the separation into groups of a miscellaneous collection of sera reinforces the suggestion that tetanus toxins and antitoxins are complex mixtures rather than homogeneous solutions of the specific molecules. And lastly, the relatively high titres of antitoxins when estimated by homologous test-toxins, in comparison with the corresponding titres when estimated by heterologous test-toxins, are highly significant and provide virtual proof of the theory.

With regard to the results yielded by the interactions of homologous reagents it is noteworthy that the mouse titres of samples from the experimental horses "4" and "Four" that were immunised with a 4-day antigen show a regular decrease, when estimated by the homologous test-toxins, in the order of the 4-day (strictly homologous), 12-day and 21-day toxin. In contrast to this result there is the striking agreement between the mouse and the guinea-pig potency values of the two samples taken from the horse "21," which was immunised with a 21-day toxin, when these were assayed

by means of the homologous 12-day and 21-day test-toxins. These results give support to the view that tetanus toxins increase in complexity during incubation of the culture and indicate that the antigenic modifications of the toxin and toxoid molecules do not proceed indefinitely but that there is a limit to the process. In general, the titres given by the three homologous test-toxins are not very different and are much higher than the titres given by a heterologous test-toxin. When the reagents are homologous, the antigens, whether 4-, 12- or 21-day, would seem to possess a common specificity which is characteristic of the particular batch, and thus the factor of time of growth of the cultures from which they were derived is of secondary importance. The fact that the same strain was used and the same methods of preparation were followed indicates that it will probably prove to be difficult to reproduce an immunising antigen or test-toxin with exactly the same antigenic composition.

If the view is accepted that the primary toxin and toxoid molecules in any crude short-growth toxin are susceptible of modification in the direction of a specific bias, two possibilities need to be considered. First, there may be only one antigenic variant, which would presumably increase up to a maximum so that filtered samples of a batch of culture during the period of growth and incubation would represent a range of mixtures of primary (P) and secondary (S) toxin and toxoid molecules with an increasing formation of secondary molecules at the expense of the primary molecules. Secondly, there may be more than one antigenic variant, so that the proportions depend upon the factor of time of incubation of the culture. For the present and in default of evidence, it would seem best to avoid complicating the issues and to assume that only one antigenic variant exists; "late" toxins will, therefore, consist of mixtures in varying proportions of the P and S molecules. One must suppose that the P molecules always form a larger proportion of the total than the S molecules. This relation is likely to be true also for the corresponding antitoxin molecules in samples of anti-toxic serum, although the proportion of the anti-P and anti-S molecules may not be the same as that of the P and S molecules in the immunising antigen, since the result will depend upon the response to each kind of toxin molecule in the immunised animal, not only in different horses, but also in the same horse at different stages of its immunisation.

A note may be added regarding the strain of *Cl. tetani* used throughout this work, namely, No. 279, a type 1 (Tulloch) strain. This has been tested for purity on several occasions by my former colleague, Dr. D. W. Henderson, who has found it free from contamination with other anaerobes and true to the serological type. The suggestion has been made that the discrepancies in the titration results of tetanus antitoxins when examined in different laboratories may be due to the use of different serological types for preparing the toxins. These types are based on flagellar agglutination and, so far as is known up to the present, tetanus strains possess a common "O" antigen with an additional heat-stable antigen in the strains of certain types; type 1 is included in the group which possesses only the common "O" antigen (Gunnison, 1937; MacLennan, 1939). That the flagellar antigens are

correlated with toxin production is even less likely than that such a correlation should exist in respect of the "O" antigen.

Variations in the potency ratio, derived from the titres as estimated in the mouse and the guinea-pig of antitoxic sera from different sources

The origin of the discrepancies in the titres of sera, which derive from the use of different test-toxins, is not the only problem presented by the data in this paper, for another emerges from a comparative study of the unitage of sera when these are assayed in the mouse and the guinea-pig by means of the same test-toxin, and when the mouse units are expressed as a percentage of the guinea-pig units. The last five statements of the preceding summary illustrate the relationships which exist between the mouse and the guinea-pig titres. If the mouse/guinea-pig potency ratio of the British standard serum is taken as 100 per cent, the corresponding ratios of the sera examined fluctuate between about 40 per cent and $>$ 340 per cent; most of them are below 100 per cent. Actually, the equivalence of the titres of the British standard serum, as estimated in the mouse and the guinea-pig by the adjustment of the L+/5 dose of all the test-toxins employed, represents a dividing line between two sets of conditions, which need separate consideration: (1) those which influence the titre of the serum samples taken from an unprepared horse or from a prepared horse at an early stage of the immunisation, when the ratio tends to be above 100, and (2) those which influence the titre of serum samples taken from a prepared and well-immunised horse, when the ratio tends to be below 100. It is best to discuss the last-mentioned class of titration values first. A good example is serum 922 which ends the list in Table 3, with mouse/guinea-pig ratios ranging from 61 per cent to 46 per cent for four glycerinated test-toxins; this serum was taken from a well-prepared horse (Ramon *et al.*, 1937).

When seeking for the cause of the fluctuations in the mouse/guinea-pig ratios, two questions may be asked: (1) May it be that the primary and secondary molecules have a quantitatively different toxic action in the mouse and the guinea-pig? and (2) Are the primary and secondary toxin-antitoxin complexes characterised by a difference in co-aptation which influences their firmness of union or, otherwise stated, their tendency to dissociate *in vitro* or *in vivo*? The ratios of serum No. 922 indicate that the toxin reagent in the test-dose is relatively more difficult to neutralise in the mouse than in the guinea-pig. There is no ground for supposing that the essential toxic action in the mouse or guinea-pig has been altered as a result of the modifying process, if one may judge by numerous careful observations of the symptoms in both mice and guinea-pigs; and the ratios are consistently low, apart from the time of growth of the cultures from which the test-toxins were derived. On the theory that the variable factor is the relative firmness or looseness of the complex *in vitro* or *in vivo* we may take into account four complexes, namely, P.T.–P.AT, P.T.–S.AT, S.T.–S.AT and S.T.–P.AT, the first and the third of which are homologous and the second and fourth are composed of heterologous reagents; it may be supposed that

the homologous reagents form a firmer union than the heterologous reagents, *in vitro* at least. But this train of ideas should be applicable equally to both of the test-animals and so does not explain the relative dissociability of the complex or mixture of complexes in the mouse. We are thus driven to conclude that the particular complexes formed by No. 922 and similar sera are, for some unknown reason, more easily dissociated in the tissues of the mouse than in those of the guinea-pig.

It was thought that a comparison between No. 922, which ends the list in Table 2, and the non-avid serum which heads it, might provide a clue. The non-avid serum has a mouse/guinea-pig potency ratio of 151/100, and presumably was taken from an unprepared horse or from a prepared horse at an early stage of immunisation; the relatively high titre favours the first supposition. The high mouse titre of this serum relatively to the guinea-pig titre can be explained on the theory that the complex is easily dissociable in the guinea-pig as being the larger test-animal, the effect being similar to that which follows dilution of the reacting substances *in vitro*. This explanation cannot, however, be applied with any show of reason to the ratios given by the serum No. 922, because it would lead to the conclusion that dissociation takes place more readily in the smaller test-animal.

There remains for consideration the origin of the reverse ratio, where the mouse titre of a serum sample is higher than the guinea-pig titre. Here the idea of dissociation resulting from dilution of the complex in the larger animal fits, but leaves unanswered the reason for the successive lowering of the mouse titre in relation to the guinea-pig titre as the immunisation proceeds. One may suppose that in early samples from a prepared horse the antitoxin molecules are immature in some biophysical sense and form a loose, easily dissociable complex. Ramon, Lemétayer and Pirosky (1937) compared the titres obtained by the flocculation technique and by tests in guinea-pigs for the two types of serum and found that the antitoxin from a well-prepared horse—actually No. 922—combined rapidly with the toxin and gave the same titre by both methods, whereas antitoxin from an unprepared horse combined so slowly with the toxin that the *in vivo* titre was much lower than the flocculation titre except when the reacting mixture was kept for 6 hours at 40° C. before injection into the guinea-pig; the authors ascribe these results to a difference in "quality" of the antitoxins. It is of interest that even homologous toxins and antitoxins give the reverse ratio; this provides evidence in favour of the "quality" of the antitoxin as being the preponderant factor in the reaction rather than the toxin component.

It would be of interest to immunise horses with and without a preparatory dose, to use the same tetanus antigen throughout, and to test the serum samples by means of the homologous as well as heterologous test-toxins. An examination of such samples from the viewpoint of criteria that are adopted as a measure of "avidity" might throw light on this aspect of the subject, because the work on avidity of tetanus sera has been carried out with heterologous reagents.

The third statement of the foregoing summary notes that, when the titres of a number of sera were estimated in both the mouse and the guinea-pig

by a heterologous 4-day and 12-day test-toxin derived from the same batch of culture, the differences shown by the mouse titres were smaller than those shown by the guinea-pig titres. This result is also seen when the reagents are homologous, and the reason may be the tendency of the complex to dissociate in the larger test-animal.

The preceding discussion—to revert to the origin of the groups in Table 3—makes it appear probable that the similarity of the potency ratios derived from the mouse and the guinea-pig titres, as estimated by two short-growth and two long-growth test-toxins, can be attributed to identity or close similarity in the proportion of the primary and secondary antitoxins in the sera which form the group.

Lastly, the question may be asked whether there is any approach, other than the serological one, to the solution of the problems with which this paper deals. A biochemical or biophysical analysis of crude tetanus toxins is unlikely to prove successful owing to the lability of the toxin molecules, especially when crude toxins are submitted to processes of purification (Eaton and Gronau, 1938). On the other hand, the present author is strongly of the opinion that the newer biophysical methods should be applied to testing the theory of the complexity of tetanus antitoxins and to investigating the physical characters of avid and non-avid tetanus sera and of sera that are widely different in respect of the potency ratios they yield when assayed in the mouse and the guinea-pig.

General Conclusions

The following conclusions may be drawn from the work described in this paper.

(1) Crude tetanus toxins, whether employed as immunising antigens or as test-toxins, are to be regarded as mixtures in varying proportions of the primary toxin molecule and of an antigenic variant of this; preparations of tetanus serum likewise consist of mixtures in varying proportions of the corresponding antitoxins. Differences in mutual neutralising affinities of these components in a reacting system account for the discrepancies in titre of tetanus sera when different test-toxins are used for the assays.

(2) The variable potency ratios of tetanus sera derived from assays in the mouse and the guinea-pig, when the same test-toxin is used, are probably referable to physical or chemical differences in the antitoxin component of the reacting system.

Acknowledgement. My best thanks are due to Dr. C. Russell Amies for carrying out some of the assays.

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The opportunity has been taken while revising this paper for publication to add a brief note amplifying the conclusions as stated above. The

suggestion has been made (p. 138) that in the early stages of the culture-growth a high proportion of the toxin and toxoid molecules are of the normal or primary type but that, as the result of some biochemical action on the labile primary molecules during continued incubation of the culture, a change is effected in the specific binding groups of some of them that is of such a nature and degree as to modify them in an immunological sense. The present writer is now of the opinion that the biochemical action referred to may be such as to cause denaturation of the toxin and toxoid molecules, a process which is known to alter to a greater or less extent the serological specificity of antigenic substances submitted to it. This possibility can readily be tested experimentally.

Again, it is stated (p. 139) that the experiences recorded in this paper indicate that it will probably be difficult to reproduce an immunising antigen or a test-toxin with exactly the same antigenic composition, even when the same strain and the same method of preparation of the culture-medium are used. This statement does not accord with the recent work of Ipsen (1940–41), who found that toxins produced with the same strain are alike, not only in the qualities revealed by an antitoxin assay, but also in other respects, for example, in their toxicity to rabbits and mice. In the view of the present writer, as already expressed, the mode of preparation of the test-toxins, that is, whether glycerinated or precipitated by ammonium sulphate, may account for the contradictory results; and it may be that the precipitation method brings about some denaturation of the toxin leading to a loss of certain toxophoric groupings which are conserved when glycerol is added to the filtered broth toxin. The possibility of some such change was touched upon when discussing the differences in the potency ratios derived from assays of tetanus antitoxins in the mouse and the guinea-pig in which glycerinated and precipitated test-toxins were used.

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THE BIOLOGICAL STANDARDISATION OF HEPARIN

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In this paper an attempt is made to outline some of the facts which must be kept in mind, and the difficulties which may arise, when it is required to compare the strength of preparations of heparin. The properties of a satisfactory standard heparin are described: they correspond with those of the new Provisional International Standard. Finally, the procedures now available for the assay of heparin are listed and briefly discussed.

It is necessary first to consider what will be the probable composition of the heparin preparations whose potency is to be determined; for the choice of a suitable standard, and of a suitable method for comparing other preparations with it, must depend on the expected composition of such other preparations.

The Composition of Heparin Preparations

Although our knowledge of the chemistry of heparin has been much extended during the past few years, in many respects it is still imperfect: on a number of points, indeed, the leading authorities are in sharp disagreement. These uncertainties are due, in the first place, to the size and complexity of the molecule of heparin, which makes characterisation difficult, and in the second place, to the adoption by different investigators of different methods for the isolation and assay of active material. It is unnecessary here to enumerate the points at issue; but it is proper to mention a number of facts which show that even highly purified heparin preparations may differ considerably in biological activity. Heparin, as it occurs naturally, is, in fact, probably not a single chemical compound, but a family of closely related principles, which nevertheless differ in activity both quantitatively and qualitatively. These several natural heparins may, in their turn, be further modified by the chemical procedures directed towards their isolation.

1. Heparin can be obtained from a variety of tissues as a crystalline barium salt of very high potency (Charles and Scott, 1936; Charles and Todd, 1940). The barium salts prepared from the various tissues of a given mammalian species have been found to be identical in potency, whereas preparations obtained by the same technique from other species may be of quite different potency (Jaques and Waters, 1940; Jaques, Waters and Charles, 1942). Furthermore, the *relative* potency of the heparins obtained from different species is not constant, but depends on the particular form of test used to compare their anticoagulant activity. These differences in the biological effects of crystalline material from different species are not asso-

ciated with any detectable difference in the crystalline form or sulphur content of the barium salts, and their chemical basis is, as yet, uncertain.

2. It is possible by fractionating crude heparin preparations with brucine to obtain purified materials containing different proportions of esterified sulphuric acid (JORPES, 1935, 1942), the most active preparations being those with the highest sulphur content. JORPES suggests that several heparins of different sulphur content occur *in vivo*, the strongest (and most highly esterified) corresponding to the crystalline barium salt of CHARLES and SCOTT. It is clear, however, from the results of JAQUES, WATERS and CHARLES (see also below) that variations in sulphur content are not the sole cause of variations in the potency of purified heparin preparations.

3. Even preparations, apparently crystalline, of the barium salt of ox heparin, and preparations of the sodium salt prepared from these, have been found to vary in potency by 30 per cent or more, when tested on mixtures of recalcified plasma and tissue extract (thrombo kinase) (MACINTOSH, 1941*a*). These variations could not be explained by differences in ash or water content. When the same preparations were compared by means of a test on the fresh whole blood of the rabbit, these differences in potency were found to be significantly smaller, although they did not disappear.[1] On the other hand, the Toronto workers have assayed many samples of the crystalline barium salt of ox heparin, prepared by them, on freshly drawn cat's blood, and have never been able to detect any variation in activity. Crystallinity of ox heparin preparations is thus, apparently, not an infallible indication of uniform and maximal activity, although specially designed tests may be necessary to detect with certainty the differences between crystalline preparations. It should be noted, however, that a number of such preparations, when tested in this laboratory by MACINTOSH's (1941*a*) method, which appears to accentuate the deficiencies of sub-maximally active material, were found to be of uniform, maximal activity.

These facts indicate that heparin preparations, even when they have been purified by procedures involving crystallisation and contain no inert material, are not necessarily samples of a single pure substance. The high and uniform potency of certain samples of the crystalline barium salt of ox heparin does suggest, however, that this material may represent a definite chemical individual. At any rate, it has not so far been found possible further to concentrate its activity, or to prepare more active material from bovine tissue by other methods.

It is an extraordinary fact that methods of biological assay similar in principle, and all measuring, in one way or another, the most characteristic biological property of heparin, should give different results. The most probable explanation is that the various members of the heparin family vary in their affinity for the several components of the complex system involved in the clotting of blood. It is to be remembered that heparin exerts its anticoagulant effect at several stages of the clotting process.

[1] These tests were carried out by Messrs. W. A. BROOM and E. M. BAVIN, Boots Pure Drug Co. Ltd., Nottingham. I am indebted to them for their co-operation, and to Mr. E. C. FIEILLER of the same firm for a statistical treatment of the results.

Choice of a Standard Heparin

It follows from what has been said that whatever preparation of heparin may be selected to serve as standard, it will not be possible to compare every commercial preparation with it with reproducible results, unless the conditions of assay are in all cases identical. At the present time (see p. 149) it seems unwise to prescribe such rigid conditions for the assay of heparin. The more serious sources of discrepancy can, however, be eliminated by proper selection of the standard heparin.

(*a*) *Species of origin.* All the heparin now available for human therapy comes from ox tissue. Although dog heparin is more potent (Jaques and Waters, 1940; Jaques, Waters and Charles, 1942) there is no reason to suppose that it, or any other form of heparin, will supersede the bovine variety. The standard should, therefore, be a sample of ox heparin.

(*b*) *Purity and Activity.* The standard should, furthermore, be composed of the purest and most active ox heparin available. The reason for this is obvious. Such a standard could be validly compared, by any method of measuring anticoagulant activity, with any other heparin preparations whose activity is due entirely to the same active principle. It seems very probable that the single, most active bovine heparin has been prepared in approximately pure form, and is represented by the best samples of the crystalline barium salt, and by the "chondroitin trisulphuric acid" of Jorpes (1942). It may be predicted that, as methods of manufacture improve, commercial heparin preparations will tend to consist wholly of this substance, and that, in consequence, the potency values assigned to such preparations will depend less and less on the particular method chosen to measure them. On the contrary, if a submaximally active material, containing less potent forms of heparin, is chosen as standard, different results will be obtained by different methods of assay, and the discrepancies among the results will become greater as the products to be assayed become purer.

Mode of salt formation. Heparin is a strong acid and forms a variety of salts. The barium salt is the only form which has been crystallised: it contains some 12 per cent of water of crystallisation, and forms acid solutions. The form used clinically is the sodium salt, which is approximately neutral in reaction. As heparin tends to be unstable in acid solution, it seems possible that, over a long period of storage, the sodium salt might maintain its potency better than the crystalline (hydrated) barium salt; and in fact, there is reason to believe that the latter is liable to deteriorate when kept at ordinary temperatures. (There is, however, no conclusive evidence that the barium salt would be unstable when stored at a low temperature.) A stronger argument for selecting the sodium salt is that it is the form clinically used, and that its biological activity is due for all practical purposes entirely to the heparin anion. It might in the future be desired to compare the standard with an unknown preparation in respect of some activity which would be affected by the barium ion. Any evidence for the purity and homogeneity of a sample of crystalline barium salt should apply equally to a sample of sodium salt prepared from it. There is no reason to suppose

that the specific anticoagulant activity of heparin is in any way modified by its mode of salt formation, apart from a possible direct influence of the cation itself on the clotting process.

Finally, it is desirable that the standard should be *dry*. Dryness should promote stability, and would facilitate replacement of the standard, when it becomes exhausted, by material of equal potency. The difficulties introduced by the necessity for weighing small quantities of hygroscopic material are not likely to be serious.

In summary, it has been considered that a suitable standard for heparin should consist of the dry neutral sodium salt of ox heparin, prepared from the crystalline barium salt of CHARLES and SCOTT (1936), and representing in unaltered condition the most active form of heparin obtainable from ox tissues.

METHODS FOR THE STANDARDISATION OF HEPARIN

The potency of the earliest, crude preparations of heparin, prepared by the method of HOWELL and HOLT (1918), was stated in terms of a purely biological unit, without reference to a standard preparation. The unit was generally taken to be that quantity of heparin which, added to 1 c.c. of cat's blood, kept at 0° C., would just suffice to prevent its clotting. The unreliability of a unit so defined is now well understood. Most recent workers have, accordingly, selected some one sample of heparin as reference standard, and assessed the potency of other samples in terms of this. The assay of the unknown sample then consists in determining the weight of this sample, which exerts an anticoagulant effect equal to that exerted by a given weight of the standard.

Although it has been generally agreed that heparin preparations should be assayed by comparison with a standard, no single method of comparison has, so far, been generally adopted. The methods which have been suggested fall into two classes: (*a*) methods which measure, in one way or another, the specific anticoagulant activity of heparin in systems containing blood or its components; and (*b*) methods which measure some physical or chemical property of heparin which appears to be related to its anticoagulant activity. It is clear that methods of class (*b*) are only generally applicable, if they can be shown to give results agreeing with those given by the methods of class (*a*).

The methods of the first class differ among themselves with regard to the clotting reagents used. The simplest procedure is to measure the effect of heparin in delaying the spontaneous clotting of mammalian blood shed into a glass vessel. Generally a series of test-tubes is taken, which contain graded dilutions of the standard and the unknown preparation, and the blood is run into these tubes directly from an artery of the animal. After a suitable time, the tubes are examined, and the dose of each preparation which has just sufficed to prevent clotting is noted. Alternatively, and preferably, the tubes are examined at intervals, or the degree of clotting in the different tubes is estimated: thus the comparison between standard and unknown is made, in effect, at a number of dose-levels, instead of at one

dose-level only. Some skill is required for the drawing of successive uniform samples of blood from the living animal, and in the judgment of the end-point, but with practice, remarkably accurate results may be obtained.

Some of the difficulties of the whole blood assay may be avoided by the use of oxalated or citrated blood or plasma. This is likewise mixed, in a series of tubes, with suitable dilutions of the standard and test samples of heparin, and the clotting process is initiated by the addition of either calcium salts or thrombin. In the former case, a suitable tissue extract may be added to accelerate clotting and so provide a sharper end-point. Alternatively, bird plasma, which does not clot spontaneously *in vitro*, may be used, and tissue extract added to initiate coagulation. Such methods have the considerable practical advantage that variations in the reactivity of the clotting reagents are less serious than in the simpler methods mentioned above, in which the only reagents are blood and glass. It is not possible, unfortunately, to use purified thrombin and fibrinogen as the clotting reagents, for heparin does not retard clotting in this system unless another factor, present in plasma but not yet characterised, is added.

In Table 1 are listed the procedures for the standardisation of heparin which have been described in detail, and which measure, in one way or another, the specific anticoagulant activity of heparin.

Table 1

METHODS FOR THE BIOLOGICAL STANDARDISATION OF HEPARIN

Authors	Reagents	Remarks
FISCHER and SCHMITZ (1932)	Bird plasma, tissue extract	Arbitrary biological unit proposed: no permanent standard
CHARGAFF, BANCROFT and STANLEY-BROWN (1932)	Bird plasma, tissue extract	As above
ASTRUP (1938)	Bird plasma, tissue extract	
WILANDER (1939)	Fresh ox blood	Method ascribed to E. JORPES
REINERT and WINTERSTEIN (1939)	Oxalated ox plasma, Ca salt	
DAM and GLAVIND (1939)	Human blood, tissue extract	
JAQUES and CHARLES (1941)	Fresh cat blood	Method of CHARLES and SCOTT, based on that of HOWELL
JAQUES and CHARLES (1941)	Oxalated ox blood, thrombin	Unsuitable for barium salt of heparin
JAQUES and CHARLES (1941)	Bird plasma, tissue extract	Modified from the method of FISCHER and SCHMITZ (1932)
SCHÜTZ (1941)	Fresh rabbit blood	
MACINTOSH (1941*a*)	Oxalated horse plasma, Ca salt, tissue extract	
FOSTER (1942)	Oxalated ox plasma, Ca salt	Modified from the method of REINERT and WINTERSTEIN (1939)

All these procedures are, no doubt, capable of giving reproducible results of good precision, particularly with proper statistical treatment of the data of individual assays. The selection of one would be merely a matter of convenience, if it could be shown that each of them gave the same result, when used to compare the potency of any given pair of preparations. Unfortunately this is not always the case, as has already been pointed out. It would be desirable for most purposes, indeed, to choose that method which most accurately assessed the therapeutic efficiency, in man, of the sample under test. There are, however, at present no grounds for supposing that any of the methods listed may be accepted as best in this respect. The simple, whole-blood methods, for example, appear to measure the effect of heparin in preventing the disintegration of blood platelets which have come in contact with a glass surface; and the other methods are likewise all, to some extent, artificial, and fail to reproduce the conditions under which heparin acts *in vivo*. It is possible, but not proved, that the species from which the blood or plasma for the test is taken, may influence the result: human blood would, perhaps, be best. While these points remain to be decided, it is impossible to recommend one or another of the methods listed as clearly superior to the rest. Fortunately, it seems probable that the results of all methods will agree well enough for the practical purpose of standardising heparin for clinical use, provided that the material comes always from the same animal species, and consists largely or wholly of the unaltered, most active form of heparin present in the tissues of that species.

Brief mention should be made of those methods for standardising heparin which do not directly depend on the measurement of its anticoagulant effect. Two of these have been suggested. One (Fischer and Schmitz, 1932) depends on the effect of heparin on the solubility of proteins at a pH near their isoelectric point. The other (MacIntosh, 1941*b*) is based on the property, shared by heparin with other anticoagulants containing esterified sulphuric acid, of forming complexes with certain basic dyes. These methods are rapid and objective. They have not, however, been shown to give results agreeing so well with those of the biological methods as to permit the latter to be discarded. Thus the dye method, for example, fails to discriminate between the heparins of different mammalian species, which have been found to differ widely in potency when examined by a number of clotting tests (Jaques *et al.*, 1942). Such purely physical and chemical methods of assay are undoubtedly useful for many purposes, but they should not be accepted without reserve as giving a true measure of the therapeutic activity of heparin.

Finally, it must be pointed out that heparin is generally prepared in the form of one of its salts, and that the activity of the barium salt, for instance, may be modified by the presence of the barium ion in the clotting mixture (Jaques and Charles, 1941). Likewise, if the preparation to be assayed contains a preservative, or other added material, the possibility that its presence may compromise the assay must be excluded by suitable control experiments.

SUMMARY

The conditions necessary for a satisfactory biological assay of heparin, and the properties of a suitable heparin standard, are described.

The methods available for the assay of heparin preparations are listed and briefly discussed.

I am indebted to Sir Henry Dale, lately Director of this Institute, for the benefit of his advice on many of the points discussed.

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M.49

MEMORANDUM ON A PROVISIONAL INTERNATIONAL STANDARD FOR HEPARIN (1942)

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During recent years the use of heparin, due largely to improvements in the methods of its isolation from animal tissues, has greatly increased, and the question of its standardisation and assay has become of urgent practical importance. As in the case of other substances, the potency of which can only be determined by biological methods of assay, it is generally agreed that the preparations used in different countries should be assayed in relation to a common standard and their potency expressed in a commonly accepted, uniform system of notation.

Under normal conditions the task of selecting a suitable standard preparation, and the responsibility for making recommendations for its use on an international basis, would have been undertaken by the Permanent Commission on Biological Standardisation of the Health Organisation of the League of Nations. This procedure was impracticable owing to the war. Accordingly, the Department of Biological Standards of the National Institute for Medical Research, London, N.W.3, which is responsible to the Commission for the supply, storage and distribution of the International Standards for certain drugs and hormones and the vitamins, with the co-operation of such experts in this field as are readily accessible in present circumstances, has taken the initiative indicated below whereby a Provisional International Standard Preparation for heparin, in terms of which a Provisional International Unit of activity has been defined, has been made available for the use of all who may require it, either for purposes of research or for standardisation and assay. It is to be understood that the action now taken, and questions relating to the suitability of the material selected for use as the standard preparation and the definition of the unit, will be reviewed in due course by the Commission, when that body is able to resume its activities.

The Provisional Standard Preparation

The specimen of heparin which served as the basis for the Standard was prepared by the Connaught Laboratories of the University of Toronto, and made available for this purpose through the kindness of Professor C. H. Best. The material, 85 gm. in weight, was received at the National Institute for Medical Research, in the form of an air-dried powder. After testing, it was distributed into about 400 small ampoules, each containing about 50 mg., and 40 larger ampoules, each containing about 1000 mg. of the powder. According to the routine now applied at this Institute for the preparation of such standards, the material was further dried in the ampoules over phosphorus pentoxide, until check weighings showed that constant weight had been reached. All the ampoules were then filled with pure dry nitrogen and sealed. They have been stored in the dark at a temperature of − 2° C. to − 4° C.

Properties of the Standard Preparation

The International Standard preparation is a greyish-brown, moderately hygroscopic

powder, completely soluble in distilled water or 0·9 per cent NaCl solution. The pH of a 1 per cent aqueous solution is approximately 5·6.

Chemical analyses of the dry powder, which have been carried out by Dr. Weiler at Oxford, gave the following results.

Carbon: 24·2%; 24·0%
Hydrogen: 3·79%; 3·80%
Nitrogen (Dumas): 5·16%
Sulphur: 12·45%

A series of micro Kjeldahl estimations, carried out in this Institute, gave the following results: Nitrogen, 4·30, 4·30, 4·40, 4·40, 4·35, 4·42, 4·32, 4·17 per cent. Mean = 4·33 per cent.

The values for nitrogen are much higher than those which have been reported elsewhere for purified heparin preparations (CHARLES and SCOTT, 1936; CHARLES and TODD, 1940; REINERT and WINTERSTEIN, 1939; and JORPES and BERGSTROM, 1942). The discrepancy has not yet been explained: it is too great to be accounted for by variations in water content and in mode of salt formation. The analytical data for the other elements accord fairly well with those given in the literature.

Ampoules of the Standard have been stored at 37° C. for 6 months without detectable deterioration in the potency of the powder. Neutral sterile solutions in 0·9 per cent saline likewise appear to be quite stable for at least several months.

Before its final drying, the Standard Material contained about 12·9 per cent of water. On drying it showed a gain in activity per unit of weight closely corresponding to its loss of weight.

Before its final drying, the Standard Material was compared in two laboratories with the provisional British Standard for heparin, issued by this Department in March 1941. The two materials, which contained about the same proportion of water, were found by both laboratories to differ by not more than 3 per cent in potency.

Definition of the Provisional International Unit

It is proposed that the activity of heparin be expressed in units, the activity corresponding to that of 1/130 mg. of the standard dry powder being defined as one unit. The unit so defined is believed to be very nearly equal to that proposed by MURRAY and BEST (1938), and sometimes referred to as the Toronto unit: this unit represents the activity of 1/100 milligramme of a sample of the crystalline barium salt of heparin, prepared by the method of CHARLES and SCOTT (1936), and stored in the Connaught Laboratories, University of Toronto.

The theoretical ratio of the potencies of the dry sodium salt and the crystalline (hydrated) barium salt may be calculated from the empirical formulas given by REINERT and WINTERSTEIN (1939) and CHARLES and SCOTT (1940): this ratio is, in fact, 130 : 100.

The unit based on the British Provisional Standard (March 1941), which was described as containing 110 Toronto units per milligramme, is a practically identical quantity.

Use of the Provisional International Standard

The Provisional International Standard will be supplied to the Directors of National Control Centres in those countries in which these have been established, and, on application, directly from this Institute to individual laboratories, institutes and research workers who are unable to obtain their supplies from national centres.

As in the case of other International Standards which have been provided, it is suggested that, whenever practicable, other countries should establish their own national standard for heparin, assayed exactly in terms of the Provisional International Standard, and supply it for local use in their own country. It is recommended that such national standards should be specimens of the sodium salt of ox heparin, prepared by methods involving the crystallisation of the barium salt, or by such other methods as may yield a product not markedly inferior to the Provisional International Standard in representing the unaltered, and most active anticoagulant constituent of ox tissue. The powder so obtained should be dried and stored in the manner described above. A national standard should

not be more than 20 per cent less active, weight for weight, than the provisional International Standard Preparation.

Solutions of the International Standard or of a national standard are prepared by weighing the powder from a freshly opened ampoule in a stoppered weighing-bottle, avoiding all but the briefest exposure to the air, and dissolving it completely in a measured volume of 0·9 per cent NaCl solution prepared with distilled water, to make a solution of convenient strength. The standard powder dissolves rather slowly, and care must be taken that the last solid particle has disappeared before the solution is divided or diluted. If, for any reason, the whole of the contents of an ampoule is not used immediately, the remaining powder may be stored in a dry open ampoule over phosphorus pentoxide in an evacuated desiccator, which ought to be kept in the dark at a temperature below 0° C.

Solutions containing 0·5 per cent or more of the International Standard are stable for at least 6 months if they are protected from contamination by bacteria or moulds and stored in a cool place. They may be sterilised by passage through a bacterial filter or by heating in the autoclave at 110° C. for thirty minutes. Tricresol (0·3 per cent) or other preservative may be added, if it has first been shown that the quantity of preservative used is insufficient to affect the results of whatever assay method has been chosen.

The Assay of Heparin Preparations

It is essential that the assay of preparations of heparin shall be carried out in strict comparison with the Standard Preparation. In conformity with usual practice, no particular method or methods for the assay of heparin are prescribed in this Memorandum. It is expected that individual workers will employ the methods of which they have experience and in which they have confidence. It is suggested, however, that the method employed should measure the specific anticoagulant activity of heparin and not some chemical or physical property which may be considered to be associated with it. Provided the method employed is capable of distinguishing potency differences of 10 per cent, the questions as to whether fresh or oxalated whole blood or plasma should be used, whether extracts containing thrombin or thrombokinase (thromboplastin) should be added, or whether the actual clotting time should be measured or merely the presence or absence of a clot should be observed, may be left to the decision of the individual worker.

One source of uncertainty in the standardisation of heparin must, however, be pointed out, though it appears unlikely that it will often lead to serious practical difficulty. This lies in the fact that the potency of a given preparation of heparin, stated in terms of the potency of another preparation, is often not a fixed quantity, but depends to some extent on the particular biological test used for the comparison. Discrepancies thus arise, when the results of one kind of biological assay are compared with the results of another, although either method by itself may give results which are sufficiently precise and reproducible. Similar discrepancies have been met with in the standardisation of other drugs: they are due to the presence, in the standard or the preparation to be tested, or in both, of more than one substance which can affect the biological test. In the case of heparin, as elsewhere, it is unfortunately not yet clear which of the methods available measures most accurately the therapeutic efficacy of the material under test. These uncertainties are of little practical importance so long as the preparations being tested are, like the Standard, of good quality. Thus the activity of heparin purified by careful crystallisation of the barium salt appears to be constant to within 30 per cent, whatever method of assay be used, if allowance be made for differences in water content and mode of salt formation: most batches show practically the same maximal activity. On the other hand, material which has been incompletely purified, or denatured by drastic chemical treatment during its isolation, may vary by more than 50 per cent in potency, when compared with the standard by different methods. The same is true of heparin preparations extracted from other than bovine material. Uncertainties of this order of magnitude should be avoided, and it is desirable that the appropriate authority in each country should take what steps it may deem necessary to ensure that any heparin intended for clinical use is of satisfactory quality.

Summary

1. A Provisional International Standard for heparin has been established. The Standard material is a dried sample of the sodium salt of heparin prepared by methods involving the crystallisation of the barium salt, and believed to represent in practically pure and unaltered form the most active anticoagulent principle which can be extracted from bovine tissue.

2. The Standard material contains, by definition, 130 units of activity per milligramme.

3. Some particulars have been given of the manner in which the Standard is to be used.

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LEXIQUE POLYGLOTTE DES MALADIES CONTAGIEUSES

Contribution
à la Nomenclature nosologique internationale

POLYGLOT GLOSSARY OF COMMUNICABLE DISEASES

Contribution
to the International Nomenclature of Diseases

par le — by

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INTRODUCTION

Il y a quelques années, deux philologues publièrent, sous le nom de « Faux Amis ou les Trahisons du Vocabulaire anglais »,[1] une liste de mots anglais identiques — ou ressemblant fort — à des mots français, mais de sens différent, et susceptibles par conséquent de trahir la confiance du traducteur non averti.

Les noms de maladies dans les différentes langues sont bien souvent assez ressemblants pour faciliter leur traduction d'une langue à une autre, mais ils comportent un certain nombre de « faux amis » qui, trop fréquemment, sont causes de méprises et de confusions.

C'est ainsi que le mot « typhus », qui en France désigne exclusivement le « typhus exanthématique », désigne en Allemagne la fièvre typhoïde. Sans doute le nom officiel complet de cette affection est-il en Allemagne *Typhus abdominalis*, mais en fait, dans le langage courant, le premier nom seul s'emploie... et prête à confusion.

La confusion est plus grave encore quand des médecins suisses ou belges de langue française emploient le mot « typhus » pour désigner l'infection eberthienne.

En français, le mot « anthrax » désigne un conglomérat de furoncles, mais en anglais, en russe et en plusieurs autres langues, ce mot désigne le « charbon » ou infection par le *Bacillus anthracis*. Encore un faux ami.

Bien des gens croient que le latin comporte des vertus unificatrices qui éliminent de tels inconvénients. Or, nous voyons que non seulement la terminologie latine des maladies est bien moins uniforme que la terminologie botanique ou zoologique, et que

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[1] Kœssler, M., & Derocquigny, J. : *Les Faux Amis ou les Trahisons du Vocabulaire anglais*. Vuibert, édit., Paris, 1928.

INTRODUCTION

Some years ago, under a title which might be rendered as " False Friends, or Pitfalls of the English Vocabulary " [1], two philologists published a list of English words identical, or nearly identical, in form with French words, but with different meanings, and therefore likely to mislead the unwary translator.

In different languages, the names of diseases often present sufficient resemblance to facilitate their translation from one language to another, but amongst them there are a number of " false friends " which may only too often be the cause of mistakes and confusion.

Thus, the word " typhus ", which in France is reserved exclusively for " typhus fever " *(typhus exanthématique)*, in Germany designates " typhoid fever ". True, the complete official name in Germany is *Typhus abdominalis*, but, in fact, in common parlance the first name alone is used and may mislead the French reader.

Confusion is even more likely when French-speaking Swiss or Belgian physicians use the word " typhus " to designate " enteric fever ".

In French, the word " anthrax " corresponds to a conglomeration of boils, but in English, in Russian, and in several other languages, the word means " infection by *Bacillus anthracis* " — another false friend.

Many people suppose that the use of Latin acts as a unifying factor and does away with such difficulties, but unfortunately this is not the case. Latin nomenclature of diseases is far less uniform than Latin botanical and zoological nomenclatures. Not only does the name of a single disease have several Latin homo-

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[1] Kœssler, M., & Derocquigny, J. : *Les Faux Amis ou les Trahisons du Vocabulaire anglais*. Vuibert, édit., Paris, 1928.

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non seulement une même maladie comporte des séries de noms latins homonymes, mais que — et cela est plus grave — un même nom latin désigne parfois plusieurs maladies différentes. Nous l'avons vu pour le mot « typhus », nous le revoyons pour *rubeola* qui souvent désigne la rougeole *(morbilli)* et plus rarement la rubéole *(rubella)*. Faux ami encore !

La confusion que causent ces « faux amis » prend assez souvent des formes concrètes et provoque des difficultés sérieuses. C'est ainsi qu'une institution internationale, comme la Croix-Rouge à Genève, recevant de l'étranger une demande urgente de vaccin antityphique, peut hésiter à envoyer soit du vaccin antityphoïdique, soit du vaccin antiexanthématique.

De même, la transmission à sa famille du diagnostic hospitalier d'un prisonnier de guerre malade nécessite une traduction appropriée. Il en est de même de la cause éventuelle de son décès.

Ce sont de tels besoins pratiques qui ont incité, à la fin de 1942, la Commission mixte de secours de la Croix-Rouge internationale à nous demander d'établir, à l'usage des collaborateurs de cette institution, une liste de concordance des termes désignant les maladies contagieuses dans les principales langues européennes.

Depuis vingt ans déjà, le Service des Renseignements épidémiologiques et des Statistiques sanitaires de la Société des Nations reçoit de tous pays des relevés des maladies contagieuses et des statistiques de causes de décès en toutes langues. Il a dû naturellement établir pour ses propres besoins des listes d'équivalence des noms de maladie. Son personnel a été appelé à contribuer à la préparation et à la publication des trois dernières éditions de la « Nomenclature internationale des maladies et causes de décès » de 1920, 1929 et 1938.

Comme secrétaire de la Commission mixte de Nomenclature nosologique de l'Institut international de Statistique et de l'Organisation d'Hygiène de la Société des Nations, qui prépara la cinquième Conférence internationale de revision décennale de la nomenclature, en 1938, et comme secrétaire général adjoint de cette conférence, nous avons été personnellement à même d'apprécier à la fois la nécessité d'unifier les termes médicaux employés dans les divers pays et d'en préciser les sens.

Nous avons donc introduit dans l'index du manuel officiel

nyms, but — what is worse — sometimes a Latin term designates several different diseases. We have seen an instance in the word " typhus " ; we see another in *rubeola*, which is sometimes used as an equivalent of " measles " *(morbilli)* and sometimes of " German measles " *(rubella)* — yet another false friend !

The confusion such false friends may produce sometimes takes a concrete form and results in serious difficulties. Thus, an international institution like the Red Cross at Geneva may receive from abroad an urgent request for anti-typhus vaccine and may hesitate whether to send the vaccine employed against enteric or that used against typhus fever.

Similarly, the transmission to his family of the hospital diagnosis concerning a prisoner of war requires the appropriate translation. A similar necessity arises with regard to causes of death.

Such practical considerations prompted the request made at the end of 1942 by the Joint Relief Committee of the International Red Cross for lists of terms denoting the various communicable diseases in the main European languages, for the use of its staff.

For the past twenty years, the Service of Epidemiological Intelligence and Public Health Statistics of the League of Nations has been receiving from all countries reports in all languages on communicable diseases and statistics of causes of death. For its own requirements, it has accordingly had to draw up lists of names of diseases in the different languages. The staff of the Service was called upon to collaborate in the preparation and publication of the three latest editions of the International Nomenclature of Diseases and Causes of Death — 1920, 1929 and 1938.

As Secretary to the Mixed Committee on Nosological Nomenclature of the International Institute of Statistics and the Health Organisation of the League of Nations, which conducted the preparatory work for the Fifth International Conference for the Decennial Revision of the Nomenclature, in 1938, and as Deputy Secretary-General of that Conference, the present writer has had occasion to appreciate the need both for unifying the medical terms used in the different countries and for defining their meanings.

en français des « Nomenclatures internationales » [1], dont l'édition nous a incombé, de nombreux termes en langues étrangères et en latin et des notes sur les concordances ou divergences de sens de certains termes dans les divers pays.

Dans le présent lexique, nous avons dû aller au delà et donner, sous forme tabulaire, en regard des rubriques de la nomenclature internationale détaillée, une série de termes employés dans les publications officielles, la littérature médicale et le parler populaire des principales langues européennes.

Aux principales langues parlées en Europe, nous avons adjoint le latin, fréquemment employé pour la terminologie médicale.

Nous avons utilisé comme base principale de notre travail les termes contenus dans les documents officiels reçus par le Service des Renseignements épidémiologiques et des Statistiques sanitaires, et par la Bibliothèque de la Société des Nations : listes de maladies à déclaration obligatoire, statistiques de causes de décès, enfin, manuels nationaux de conversion des Nomenclatures internationales successives. Ces derniers documents, avec leurs index, nous ont été particulièrement précieux. Leurs lacunes, en ce qui concerne la médecine tropicale, ont été comblées dans une certaine mesure par la consultation des traités et périodiques traitant de cette branche de la médecine.

Les circonstances ne nous ont malheureusement pas permis de procéder par questionnaires auprès des autorités médicales des divers pays, ni même de leur soumettre les épreuves de ce glossaire ; ceci explique, en partie du moins, les lacunes et les erreurs qu'il peut contenir. Nous remercions d'avance tous ceux qui voudront bien nous signaler celles-ci, et ainsi nous aider à y remédier, soit dans une édition ultérieure, soit dans un autre ouvrage de nomenclature médicale.

Dès à présent, nous tenons à marquer ici notre reconnaissance à ceux de nos collègues et anciens collègues du Secrétariat de la Société des Nations qui nous ont aidés dans notre travail, en vérifiant et complétant nos listes de maladies, et en y ajoutant des expressions

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[1] *Nomenclatures internationales des causes de décès 1938* (classification Bertillon). Cinquième révision décennale effectuée par la Conférence internationale de Paris du 3 au 7 octobre 1938. Publié par l'Institut international de Statistique. Imprimerie Trio S. A., La Haye, 1940 (306 pp.).

We accordingly introduced into the Index of the French official handbook on " International Nomenclature " many terms in Latin and in languages other than French, together with notes on the similarities or divergencies of meaning in certain terms used in the different countries.

In the present glossary, we have gone further and have presented in tabular form, parallel to the detailed International List, a series of terms used in the principal European languages and found in official publications, medical literature and common speech.

To the principal languages spoken in Europe, we have added Latin, often used in medical terminology.

We have used, as the main basis of our work, terms contained in the official documents received by the Service of Epidemiological Intelligence and Public Health Statistics and by the Library of the League of Nations : lists of notifiable diseases, statistics of causes of death, and national handbooks containing translations of the successive International Nomenclatures. These last-mentioned documents, with their indexes, have been particularly useful. To a certain extent, we have filled in the lacunæ regarding tropical medicine by consulting treatises and periodicals covering that branch of medicine.

Circumstances did not allow of the sending of questionnaires to medical authorities of the various countries, or of submitting the proofs of this Glossary to them : this explains, in part at any rate, the gaps and errors which may be found in it. We thank in advance those readers who will be good enough to point them out and help us to remedy them in a further edition or in another publication regarding medical nomenclature.

We should like here to express our indebtedness to those colleagues and former colleagues of the League of Nations Secretariat who have helped us in our work in checking and completing our lists of diseases and adding to them popular terms which as a rule are not found in medical dictionaries. Their help has been particularly useful with regard to the Czech, Danish, Dutch, German, Latvian, Norwegian, Serbo-Croatian and Turkish languages.

We thank also Drs. T. Keller, A. Stocker and L. Weber-Bauler for their assistance regarding the Polish, Roumanian and Russian languages.

populaires qui, le plus souvent, échappent aux vocabulaires. Leur concours a été particulièrement utile pour les langues allemande, danoise, lettone, néerlandaise, norvégienne, serbo-croate, tchèque et turque.

Nous remercions également les docteurs T. Keller, A. Stocker et L. Weber-Bauler pour leur aide concernant les langues polonaise, roumaine et russe.

Nous regrettons de n'avoir pas été à même d'inclure dans ce glossaire les termes gaéliques, les listes de maladies contenues dans les documents médicaux officiels irlandais que nous avons pu consulter étant établies en anglais, autre langue nationale de l'Eire.

We regret to have been unable to include Gaelic terms in this Glossary ; the lists of diseases contained in official Irish medical documents that we were able to consult were in English, one of the national languages of Eire.

Première Partie

DESCRIPTION DU LEXIQUE ET DE SON MODE D'EMPLOI

1. Le lexique se compose de quatre parties. La *première* en est la description et le mode d'emploi ; la deuxième est constituée par une série de colonnes, une pour chaque langue [1], contenant, en face des titres des rubriques et sous-rubriques de la Nomenclature internationale détaillée de 1938, les noms de maladies dans les diverses langues ; la troisième est un index alphabétique ; la quatrième est constituée par quelques tableaux donnant des équivalences entre les numéros de rubriques des trois Nomenclatures détaillée, intermédiaire et abrégée de 1938, et des équivalences entre ces dernières et les Nomenclatures internationales de 1929 — c'est-à-dire de la revision décennale précédente — dont quelques pays n'ont pas encore abandonné l'usage.

2. Dans la *deuxième partie* de l'ouvrage, après une colonne consacrée à la Nomenclature internationale en français, nous avons placé le latin, qu'emploient de façon très courante les médecins des Etats scandinaves et baltes, et fréquemment aussi ceux d'autres pays, Allemagne et Pays-Bas, par exemple. Pour éviter des répétitions inutiles et de la perte de place, nous n'avons pas, en règle générale, répété les termes latins dans les colonnes des autres langues, à moins qu'ils ne soient les termes les plus couramment employés.

[1] Sauf pour les langues danoise et norvégienne, groupées en une seule colonne.

Part I

DESCRIPTION AND METHOD OF USE OF THE GLOSSARY

1. The glossary is made up of four parts. *Part I* contains a description of it and shows how it should be used ; Part II consists of a series of columns, one for each language[1], containing the names of diseases in the different languages, opposite the corresponding headings and sub-headings of the detailed International List of 1938 ; Part III is an alphabetical index, and Part IV is made up of some tables showing corresponding numbers of the headings of the three lists of the 1938 nomenclature, namely : the detailed, intermediate and abridged lists, and also the correspondence between the latter and the 1929 International Lists — *i. e.*, those of the previous decennial revision, which are still employed in some countries.

2. In *Part II*, after the official International List (in French), a column is devoted to Latin. Latin is in current use by physicians in Scandinavian and Baltic countries and frequently employed also by those of other countries — *e. g.*, Germany and the Netherlands. In some, Latin terms are used besides their equivalents in the national language ; in others, they are used to make up the deficiencies of the national language, particularly with regard to the exotic, the rarer or the more recently recognised diseases. In order to avoid repetition and to save space, we have not, as a rule, repeated the Latin terms in the columns for other languages, unless they are in fact the most commonly used and this explains many of the blanks left in the columns.

[1] With the exception of Danish and Norwegian, merged in a single column.

3. Après la colonne consacrée à la Nomenclature internationale en français et celle du latin, les langues sont données dans l'ordre alphabétique français, comme suit : allemand, anglais, bulgare, danois et norvégien, espagnol, estonien, finnois, français, grec, hongrois, islandais, italien, letton, lithuanien, néerlandais, polonais, portugais, roumain, russe, serbo-croate, suédois, tchèque et turc.

4. On s'est appliqué, par une présentation typographique appropriée, à mettre en face les uns des autres les termes les plus directement équivalents dans les diverses langues : ce sont toutefois les chiffres et lettres désignant les rubriques et sous-rubriques de la nomenclature qui constituent la véritable clef des équivalences de signification.

5. C'est pourquoi, dans l'index qui constitue la *troisième partie* de l'ouvrage, chaque nom de maladie, placé dans l'ordre alphabétique, est suivi de la désignation de la rubrique et éventuellement de la sous-rubrique sous lesquelles il figure dans les colonnes de la deuxième partie.

Il suffit de se reporter à la colonne de la langue dans laquelle on désire le terme équivalent au niveau correspondant à la rubrique et à la sous-rubrique indiquées pour trouver le ou les noms de la maladie en question.

On conçoit aisément que, dans un ouvrage couvrant vingt-quatre langues, on n'ait pu suivre la méthode convenant aux lexiques bilingues ordinaires et faire suivre chaque terme de ses équivalents étrangers. Il eût fallu en effet donner pour chaque terme les équivalents dans vingt-trois langues, d'où une perte de place considérable et des répétitions excessives.

6. Nous avons toutefois rencontré une difficulté, le fait que certaines rubriques ou sous-rubriques de la nomenclature désignent plusieurs maladies ; pour individualiser ces dernières et par conséquent présenter les équivalences de leurs désignations, nous avons dû multiplier les subdivisions au delà de ce qui avait été jugé pratique pour les statistiques ordinaires de causes de décès pour lesquelles la Nomenclature internationale a été établie. Ces subdivisions nouvelles sont désignées par des lettres entre parenthèses, pour les bien distinguer de celles approuvées par la

3. After the official International List (in French) and the column of Latin terms, the various languages are given in the French alphabetical order, as follows : German, English, Bulgarian, Danish and Norwegian, Spanish, Estonian, Finnish, French, Greek, Hungarian, Icelandic, Italian, Latvian, Lithuanian, Dutch, Polish, Portuguese, Roumanian, Russian, Serbo-Croatian, Swedish, Czech and Turkish.

4. An attempt has been made to place side by side those terms which are most nearly direct equivalents in the different languages. It is, however, the numbers and letters indicating the headings and sub-headings of the Nomenclature that constitute the real key to the equivalence of meanings between terms of all languages.

5. For this reason, in *Part III* of this work — *i. e.*, the index — the name of each disease, given in alphabetical order, is followed by the number of the heading, and often of the sub-headings, under which it is to be found in the different columns of Part II.

The name or names of a disease in any particular language may be found simply by referring to the column for that language opposite the headings and sub-headings of the International List.

It will readily be understood that, in a glossary covering 24 languages, it has not been possible for each term to be followed by all its foreign equivalents, as would have been the case in an ordinary bilingual lexicon. This would have meant giving 23 equivalents for each term, with consequent considerable loss of space and excessive repetition.

6. We were, however, confronted by the difficulty that some of the headings and sub-hcadings of the International List cover several diseases. In order to singlc these out and thus to be able to give their individual equivalents, wc have had to increase the number of sub-headings far beyond what had been found practical for the establishment of ordinary statistics of causes of death, for which purpose the International List was designed. These new sub-headings are indicated by letters in brackets in order to distinguish them clearly from the sub-headings which were approved by the International Conference for the Revision

Conférence internationale de Revision des Nomenclatures et incorporées dans la Nomenclature détaillée de 1938.

Nous nous sommes efforcés de leur donner une base logique, anatomique pour la tuberculose, les gonococcies, étiologique pour le typhus, etc.

Dans les quelques cas où ces subdivisions complémentaires ne permettaient encore point de distinguer assez clairement toutes les maladies, ou du moins leurs diverses formes, nous avons dans l'index fait précéder le numéro de la subdivision appropriée de l'équivalent latin.

On a, par contre, omis de la liste des rubriques de la Nomenclature celles qui n'avaient pas trait aux maladies contagieuses. On a omis de même tout ou partie des titres des rubriques se rapportant dans leur ensemble à des maladies non contagieuses. Dans ces cas, les numéros et les titres incomplets ont été indiqués par le signe (±).

7. En règle générale, les noms des maladies sont insérés dans l'index dans l'ordre alphabétique des substantifs (les adjectifs qui s'y rapportent étant placés après, même lorsqu'ils en sont normalement précédés dans la langue parlée. Ainsi « Laryngeal tuberculosis » est présentée comme « Tuberculosis [Laryngeal —] » et classée sous les « T »).

8. Dans l'index, chaque terme est suivi de l'indication abrégée de la langue (ou des langues) dans laquelle ou lesquelles ce terme est employé. Les abréviations employées sont les suivantes :

all.	= allemand.	*fr.*	= français.	*nor.*	= norvégien.
angl.	= anglais.	*gr.*	= grec.	*pol.*	= polonais.
bulg.	= bulgare.	*hong.*	= hongrois.	*port.*	= portugais.
dan.	= danois.	*isl.*	= islandais.	*roum.*	= roumain.
d. & n.	= danois et norvégien.	*it.*	= italien.	*rus.*	= russe.
		lat.	= latin.	*serb.*	= serbo-croate.
esp.	= espagnol.	*lett.*	= letton.	*suéd.*	= suédois.
est.	= estonien.	*lith.*	= lithuanien.	*tch.*	= tchèque.
fin.	= finnois.	*néer.*	= néerlandais.	*turc*	= turc.

9. La désignation des maladies en termes populaires est suivie de l'abréviation « (v) » pour « vulgaire ». Dans certains cas où des termes désuets ont été insérés, ils sont suivis de l'abréviation « (ant.) » pour « antique ».

of the International Lists, and were included in the Detailed List of 1938.

We have endeavoured to give to our extension of the sub-headings a logical basis, *viz.* : anatomical (for tuberculosis, gonococcal infections), etiological (for typhus), etc.

In the few cases in which the additional sub-headings do not make the distinction between all diseases, or between their various forms, sufficiently clear, the terms in the index are followed not only by the number of their appropriate sub-heading but also by the Latin equivalent.

Inversely, headings of the International List which do not refer to communicable diseases are omitted.

Those which refer chiefly to non-communicable diseases are omitted completely or in part. In these cases, the number or the curtailed title is followed by the symbol (†).

7. As a general rule, names of diseases are given in the index in the alphabetical order of nouns (adjectives being placed afterwards even when the adjective normally comes first in the spoken language. Thus " Laryngeal tuberculosis " is given as " Tuberculosis [Laryngeal —] " and classified under the letter T).

8. Each term in the index is followed by an abbreviation (in French) indicating the language or languages in which it is employed. The abbreviations used are the following :

all.	= German.	*fr.*	= French.	*nor.*	= Norwegian.
angl.	= English.	*gr.*	= Greek.	*pol.*	= Polish.
bulg.	= Bulgarian.	*hong.*	= Hungarian.	*port.*	= Portuguese.
dan.	= Danish.	*isl.*	= Icelandic.	*roum.*	= Roumanian.
d. & n.	= Danish & Norwegian.	*it.*	= Italian.	*rus.*	= Russian.
		lat.	= Latin.	*serb.*	= Serbo-Croatian.
esp.	= Spanish.	*lett.*	= Latvian.	*suéd.*	= Swedish.
est.	= Estonian.	*lith.*	= Lithuanian.	*tch.*	= Czech.
fin.	= Finnish.	*néer.*	= Dutch.	*turc*	= Turkish.

9. Popular designations of some diseases are followed by the abbreviation " (v) " for " *vulgare* ". In certain cases, some out-of-date terms have been inserted and they are followed by the abbreviation " (ant.) " for " antiquated ".

Certains termes dont l'emploi est susceptible d'engendrer une confusion sont marqués d'un astérisque « * ».

10. Un certain nombre de désignations de maladies sont vagues ; fréquemment, les sous-rubriques de la Nomenclature qui leur sont réservées comportent la mention « sans autre indication », abrégée en français « s. a. i. » et dans d'autres langues en ses équivalents :

latin :	n.s.	= non specificatus, a, um.
allemand :	o.n.B.	= ohne nähere Bezeichnung.
anglais :	u.	= unspecified.
danois :	u.o.	= uden oplysning.
espagnol :	s.o.i.	= sin otra indicación.
français :	s.a.i.	= sans autre indication.
italien :	s.a.i.	= senz'altra indicazione.
néerlandais :	z.n.a.	= zonder nadere aanduiding.
norvégien :	u.o.	= uten oplysning.
portugais :	s.o.i.	= sem outra indicação.
roumain :	f.a.s.	= fără altă indicaţie.
russe :	b.o.	= bez oboznatchenia.

11. Dans ce présent lexique, les langues slaves employant des caractères dérivés de l'alphabet cyrillique ont fait l'objet d'une translitération phonétique. Une telle translitération, pour être *parfaite*, doit être faite pour *une* seule langue. Or, le caractère polyglotte du lexique nous a obligé à adopter une forme de translitération convenant *à peu près à la plupart* des lecteurs des langues romanes et germaniques. Elle correspond plus particulièrement à la prononciation française, avec toutefois les exceptions suivantes :

la lettre « u » doit être lue avec le son du français « ou »,
la lettre « g » doit être lue avec le son du français « gu »,
la lettre « c » doit être lue avec le son du français « tss »,
la lettre « s » doit être lue avec le son du français « ss ».

On s'est efforcé de s'écarter le moins possible de l'orthographe slave originale.

Certain terms the use of which is undesirable as likely to lead to confusion are indicated by an asterisk " * ".

10. In a certain number of cases, the names of diseases are vague and accordingly the sub-headings of the Nomenclature reserved for them contain the mention " unspecified ", abbreviated as " u. " in English, equivalents in the different languages being as follows :

Latin :	n.s.	= non specificatus, a, um.
German :	o.n.B.	= ohne nähere Bezeichnung.
English :	u.	= unspecified.
Danish :	u.o.	= uden oplysning.
Spanish :	s.o.i.	= sin otra indicación.
French	s.a.i.	= sans autre indication.
Italian :	s.a.i.	= senz'altra indicazione.
Dutch :	z.n.a.	= zonder nadere aanduiding.
Norwegian :	u.o.	= uten oplysning.
Portuguese :	s.o.i.	= sem outra indicação.
Roumanian :	f.a.s.	= fără altă indicaţie.
Russian :	b.o.	= bez oboznatchenia.

11. In this glossary, Slav languages using symbols derived from the Cyrillic alphabet have been transposed phonctically into Latin characters. This transposition cannot be perfect for more than one language. In the present case, owing to the polyglot character of the glossary, we have had to adopt a form of transposition meeting only approximately the needs of readers in the Romance and Germanic languages. The form employed corresponds generally to the French pronunciation, with, however, the following exceptions :

the letter " u " should be pronounced as the English " oo " in " boot ",
the letter " g " as in the English " go ",
the letter " e " as the English " tss ",
the letter " s " as the English " ss ".

Except in those cases where phonetic rendering made some change necessary, the original Slav spelling has been respected as far as possible.

On trouvera à la page 220 un tableau emprunté à von Ostermann & Giegengack permettant le déchiffrage et la translitération en caractères latins de ceux dérivés de l'alphabet cyrillique.

12. Afin d'éviter de nombreuses répétitions, nous avons groupé dans une seule colonne de la deuxième partie, les termes appartenant aux langues danoise et norvégienne, qui sont très proches l'une de l'autre et dans lesquelles l'orthographe est assez variable. Nous avons indiqué dans l'index l'origine soit danoise *(dan.)*, soit norvégienne *(nor.)* des termes cités, soit leur appartenance aux deux langues *(d. & n.)*. Nous n'avons répété toutes les variantes orthographiques pour les termes qui existent parallèlement dans les deux langues ni dans cette partie, ni dans l'index. Nous indiquerons seulement qu'en règle générale :

« æ » danois correspond au « e » norvégien,
« g » danois correspond au « k » norvégien,
« t » danois correspond au « tt » norvégien,
« d » danois correspond au « dd » norvégien,
« b » danois correspond au « p » norvégien.

13. Nous avons fait figurer dans l'index, pour les diverses langues, des mots correspondant à : cas, décès, mortalité, maladie, épidémique, contagieux, parasitaire.

14. La *quatrième partie* contient des tables de correspondance entre les Nomenclatures abrégée et intermédiaire de 1938 et la détaillée de 1938 que nous employons comme nomenclature-clef (table 1).

Une table de correspondance similaire est donnée pour les trois Nomenclatures de 1929 (table 3) ; une autre table (2) montre la correspondance approximative entre les Nomenclatures détaillées de 1929 et de 1938.

L'ensemble de ces tables permet de rapporter à la Nomenclature-clef, la détaillée de 1938, les éléments de l'une quelconque des Nomenclatures internationales en usage entre 1931 et 1950.

A table on page 220, taken from von Ostermann & Giegengack, will enable the reader to decipher and transliterate into Latin characters those derived from the Cyrillic alphabet.

12. In the case of the Danish and Norwegian languages, which are very close to each other, and in which the spelling shows a good deal of variation, we have not repeated all the possible spellings either in the column where the two languages have been placed together or in the index. The following indications may, however, help the reader :

the Danish " æ " often corresponds to the Norwegian " e ",
the Danish " g " often corresponds to the Norwegian " k ",
the Danish " t " often corresponds to the Norwegian " tt ",
the Danish " d " often corresponds to the Norwegian " dd ",
the Danish " b " often corresponds to the Norwegian " p ".

13. For most languages, we have given in the index terms corresponding to : cases, deaths, mortality, disease, epidemic, communicable, parasitic.

14. *Part IV* contains tables of correspondence between the 1938 Abridged and Intermediate International Lists of diseases and the Detailed List which we are using as a standard (Table 1).

A similar table is given for the three 1929 International Lists (Table 3), while Table 2 shows the approximate correspondence between the 1929 and the 1938 Detailed Lists.

This Part makes it possible to refer to the standard 1938 Detailed List the items contained in any of the International Lists in use from 1931 to 1950.

Alphabets d'origine cyrillique. — Alphabets of Cyrillic Origin.

Cyrillique modifié Modified Cyrillic		Translitération	Russe Russian		Ukrainien Ukrainian		Blanc-Russien White Russian		Bulgare Bulgarian		Serbe Serbian	
А	а	*a*	А	а	А	а	А	а	А	а	А	а
Б	б	*b*	Б	б	Б	б	Б	б	Б	б	Б	б
В	в	*v*	В	в	В	в	В	в	В	в	В	в
Г	г	*g*	Г	г	Г	г	Г	г	Г	г	Г	г
Ґ	ґ	*ġ*			Ґ	ґ	Ґ	ґ				
Д	д	*d*	Д	д	Д	д	Д	д	Д	д	Д	д
Ђ	ђ	*đ*									Ђ	ђ
Е	е	*e*	Е	е	Е	е	Е	е	Е	е	Е	е
Є	є	*ē*			Є	є						
Ё	ё	*ë*	Ё	ё			Ё	ё				
Ж	ж	*zh*	Ж	ж	Ж	ж	Ж	ж	Ж	ж	Ж	ж
З	з	*z*	З	з	З	з	З	з	З	з	З	з
И	и	*ĭ*	И	и	И	и			И	и	И	и
І	і	*i*	І	і	І	і	І	і				
Ї	ї	*ĭ*			Ї	ї						
Й	й	*ĭ*	Й	й	Й	й	Й	й	Й	й		
Ј	ј	*j*									Ј	ј
К	к	*k*	К	к	К	к	К	к	К	к	К	к
Л	л	*l*	Л	л	Л	л	Л	л	Л	л	Л	л
Љ	љ	*ĺ*									Љ	љ
М	м	*m*	М	м	М	м	М	м	М	м	М	м
Н	н	*n*	Н	н	Н	н	Н	н	Н	н	Н	н
Њ	њ	*ń*									Њ	њ
О	о	*o*	О	о	О	о	О	о	О	о	О	о
П	п	*p*	П	п	П	п	П	п	П	п	П	п
Р	р	*r*	Р	р	Р	р	Р	р	Р	р	Р	р
С	с	*s*	С	с	С	с	С	с	С	с	С	с
Т	т	*t*	Т	т	Т	т	Т	т	Т	т	Т	т
Ћ	ћ	*ć*									Ћ	ћ
У	у	*u*	У	у	У	у	У	у	У	у	У	у
Ў	ў	*ŭ*					Ў	ў				
Ф	ф	*f*	Ф	ф	Ф	ф	Ф	ф	Ф	ф	Ф	ф
Х	х	*kh*	Х	х	Х	х	Х	х	Х	х	Х	х
Ц	ц	*t͡s* [1]	Ц	ц	Ц	ц	Ц	ц	Ц	ц	Ц	ц
Ч	ч	*ch*	Ч	ч	Ч	ч	Ч	ч	Ч	ч	Ч	ч
Џ	џ	*dzh*									Џ	џ
Ш	ш	*sh*	Ш	ш	Ш	ш	Ш	ш	Ш	ш	Ш	ш
Щ	щ	*shch*	Щ	щ	Щ	щ			Щ	щ	Щ	щ
Ъ	ъ	*″* [2]	Ъ	ъ					Ъ	ъ	Ъ [3]	ъ
Ы	ы	*y*	Ы	ы			Ы	ы				
Ь	ь	*′*	Ь	ь	Ь	ь	Ь	ь	Ь	ь	Ь [3]	ь
Ѣ	ѣ	*i͡e* [1]	Ѣ	ѣ					Ѣ	ѣ		
Э	э	*e*	Э	э			Э	э				
Ю	ю	*i͡u* [1]	Ю	ю	Ю	ю	Ю	ю	Ю	ю		
Я	я	*i͡a* [1]	Я	я	Я	я	Я	я	Я	я		
Ѳ	ѳ	*f*	Ѳ	ѳ								
Ѵ	ѵ	*ẏ*	Ѵ	ѵ								
	ж	*u*								ж		

[1] Comme initiales dans les noms propres, le premier mot d'une phrase, etc. : i͡a, i͡e, i͡u, t͡s.
[2] Finale muette.
[3] Desuète.
[4] D'après : — From : von Ostermann, G. F., & Giegengack, A. E.: « Manual of Foreign Languages ». 3rd ed. U. S. Govt. Print. Off., Washington, D.C., 1936.

[1] As initials in proper names, first word of a sentence, etc. : i͡a, i͡e, i͡u, t͡s.
[2] Final disregarded.
[3] Obsolete.

Deuxième Partie

LISTES DE NOMS DES MALADIES CONTAGIEUSES DANS LES DIVERSES LANGUES DANS L'ORDRE DE CLASSIFICATION DE LA NOMENCLATURE INTERNATIONALE DÉTAILLÉE DE 1938.

Part II

LISTS OF NAMES OF COMMUNICABLE DISEASES IN THE VARIOUS LANGUAGES FOLLOWING THE CLASSIFICATION ADOPTED IN THE DETAILED INTERNATIONAL LIST OF 1938.

N°	Nomenclature internationale International Nomenclature	Latin
I	**MALADIES INFECTIEUSES ET PARASITAIRES MALADIES BACTÉRIENNES**	
1	**Fièvre typhoïde**	Typhus abdominalis Febris typhoidea Ileotyphus
2	**Fièvres paratyphoïdes**	Paratyphus Febris paratyphoidea
3	**Peste**	Pestis
a(a)	*Peste bubonique*	Pestis bubonica
a(b)	*Peste septicémique*	Pestis septicemica
a(c)	*Localisation pulmonaire secondaire* (infection par les puces)	
b	Peste pneumonique primitive (infection par voie respiratoire)	Pneumonia pestis Pestis pulmonum
c	Non spécifiée	Pestis
4	**Choléra**	Cholera asiatica Cholera Cholera indica
5	**Fièvre ondulante**	Febris undulans
a	Infection par *Brucella melitensis* (mélitococcie)	Brucellosis Febris melitensis Melitococcosis
b	Infection par *Brucella abortus* Bang	Febris abortus Morbus Bangii
c	sans autre indication	

Allemand — German	Anglais — English	N°
Typhus Unterleibstyphus Bauchtyphus Darmtyphus	Typhoid fever Enteric fever Abdominal typhus	1
Paratyphus	Paratyphoid fevers	2
Pest	Plague	3
Beulenpest Bubonenpest	Bubonic plague	*a(a)*
Pestseptikaemie	Septicæmic plague	*a(b)*
	Pulmonary (secondary) plague	*a(c)*
Lungenpest	Pneumonic plague (primary)	*b*
Pest	Plague	*c*
Cholera Asiatische Cholera	Cholera Asiatic cholera Malignant cholera	4
Brucellose Febris undulans	Undulant fever Brucellosis	5
Maltafieber Mittelmeerfieber	Malta fever Mediterranean fever Melitococcus infection Febris melitensis	*a*
Bangsche Krankheit Seuchenhaftes Verkalben (vet.)	Abortus fever	*b*
Febris undulans Brucellose	Brucellosis	*c*

N°	Bulgare — Bulgarian	Danois et norvégien Danish and Norwegian
1	Коременъ тифъ Koremen tif	Tyfoid-feber
2	Паратифъ Paratif	Paratyfus
3	Чума Tchuma	Pest
a(a)		Byldepest
a(b)		Septikæmisk Pest
a(c)		
b		Lungepest
c	Чума Tchuma	Pest
4		Kolera
5		
a		Maltafeber
b		Svingefeber
c		

Espagnol — Spanish	Estonien — Estonian	N°
Fiebre tifoidea Tifus abdominal Fiebre entérica Ileo-tífus Fiebre mucosa*, Dotienentería	Kõhutõbi Kõhusoetõbi Kohutüüfus	1
Fiebres paratifoideas Paratifus	Paratüüfus	2
Peste	Kakt	3
Peste bubónica Peste de bubones Bubón pestoso		*a(a)*
Peste septicemica		*a(b)*
		a(c)
Peste pneumonica Peste pulmonar Pneumonia pestosa		*b*
Peste	Kakt	*c*
Cólera Cólera epidémico Cólera indio Cólera asiatico	Koolera	4
Fiebre ondulante Brucelosis	Unduleeruv palavik	5
Fiebre de Malta Melitococia Fiebre mediterranea Melitococosis	Malta-palavik	*a*
Infección por el *Bacillus abortus*	Unduleeruv palavik (Bang) Vahamere-palavik	*b*
Brucelosis		*c*

N°	Finnois — Finnish	Français — French
1	Lavantauti	Fièvre typhoïde Fièvre muqueuse (v) Dothienentérie Typhus abdominal (en Suisse et en Belgique seulement)
2	Paratyfus	Fièvres paratyphoïdes
3		Peste
a(a)		Peste bubonique Bubon pesteux
a(b)		Peste septicémique Septicémie pesteuse
a(c)		Peste pulmonaire secondaire
b		Peste pneumonique Pneumonie pesteuse Peste pulmonaire s.a.i.
c		Peste
4	Aasialainen kolera	Choléra asiatique Choléra indien
5		Fièvre ondulante
a		Mélitococcie Fièvre de Malte Fièvre méditerranéenne Fièvre ondulante caprine
b	Aaltoileva kuume	Avortement épizootique Maladie de Bang Fièvre ondulante bovine
c		Brucellose

Grec — Greek	Hongrois — Hungarian	Islandais — Icelandic	N°
Κοιλιακὸς τυφος Koiliakos typhos Τυφοειδὴς πυρετὸς Typhoides pyretos	Hastifusz Hasi hagymáz	Taugaveiki	1
Παρατυφοειδεις πυρετοι. Paratyphoeideis pyretoi Παρατυτος Paratyphos	Paratifusz		2
Πανωλης Panoles	Pestis		3
			a(a)
			a(b)
			a(c)
			b
			c
Χολέρα Cholera	Azsiai kolera		4
			5
Μελιταῖος πυρετος Melitaios pyretos	Máltai láz		*a*
	Bang-kór		*b*
			c

N°	Italien — Italian	Letton — Latvian
1	Febbre tifoidea Tifo abdominale Ileotifo Tifo* s.a.i.	Vēdera tīfs
2	Febbri paratifoidi Paratifo(i) Infezioni paratifiche Febbri paratifiche	Paratīfs
3	Peste	Mēris
a(a)	Peste bubbonica Bubbone pestoso	
a(b)	Peste setticemica Setticemia pestosa	
a(c)		
b	Peste pneumonica Polmonite pestosa	
c	Peste	Mēris Pestis
4	Colera asiatico Colera epidemico Colera s.a.i.	Cholera
5	Febbre ondulante Brucellosi	
a	Febbre di Malta Febbre mediterranea	
b	Infezione da *Bacillus abortus*	
c	Brucellosi	

Lithuanien — Lithuanian	Néerlandais — Dutch	N°
Vidurių šiltiné	Buiktyphus Typhoid Typhus Typheuze koorts	1
Paratyfas	Paratyphus Paratyphoïde koortsen	2
Maras	Pest Mensenpest	3
	Pest adenitis Parasitaire pest Builenpest Bubonenpest	*a(a)*
	Septichæmische pest	*a(b)*
	Secundaire longpest	*a(c)*
	Primaire longpest (contactpest)	*b*
Maras	Pest Mensenpest	*c*
	Cholera Aziatische cholera	4
		5
	Maltakoorts	*a*
	Ziekte van Bang	*b*
		c

N°	Polonais — Polish	Portugais — Portuguese
1	Dur brzuszny Tyfus brzuszny	Febre typhoide Tifoide
2	Paratyfus	Febre paratyphoide Paratifoide
3		Peste
a(a)	Dżuma gruczołowa	Peste bubonica Carbunculo pestilencial
a(b)		Peste septicemica
a(c)		
b	Dżuma płucna	Peste pneumonica Pneumonia pestilenciosa
c	Dżuma Mor	Peste
4	Cholera azjatycka	Cholera asiatico
5		Febre ondulante Brucelose
a	Choroba (gorączka) maltańska	Melitococcia Febre de Malta
b	Choroba Banga (zakaźne ronienie krow)	Infecção pela *Brucella abortus*
c		Brucelose

Roumain — Roumanian	Russe — Russian	N°
Febră tifoidă Tifus abdominal Lingoare	Брюшной тиф — Briuchnoï tif Тифозная горячка — Tifosnaïa goriatchka	1
Febre paratifoide Paratifus	Паратиф — Paratif	2
Pestă Ciumă (v)	Чума — Tchuma	3
Pestă bubonică	Чума бубонная — Tchuma bubonnaïa Чумный нарыв — Tchumnyï naryv (v)	*a(a)*
Pestă septicemică		*a(b)*
Pestă pulmonară secundară		*a(c)*
Pestă pneumonică primitivă	Чума легочная — Tchuma liogotchnaïa	*b*
Pestă Ciumă (v)	Чума б. у. — Tchuma b. y.	*c*
Holeră	Азиатская холера — Asiatskaïa kholera Холера — Kholera	4
Febră ondulantă Bruceloză	Брюсселез — Briusseloz	5
Melitococie Infecţie prin *B. melitensis*	Малтийская лихорадка — Maltiïskaïa likhoradka Мелитококкция — Melitokokkcia	*a*
Infecţie prin *B. abortus* Boala lui Bang		*b*
Bruceloză	Брюсселез — Briusseloz	*c*

N°	Serbo-croate — Serbo-Croatian	Suédois — Swedish
1	Цревни тифус Crevni tifus (Tsrevni tifus) Трбушни тифус Trbušni tifus (Trbushni tifus)	Tyfoid *Typhus* Abdominaltyfus Nervfeber (v) Tarmtyfus
2	Паратифус Paratifus	Paratyfoid *Paratyphus* Paratyfus
3	Куга Kuga	Pest
a(a)	Бубонска куга Bubonska kuga	
a(b)		
a(c)		
b	Плућна куга Plućna kuga (Pluchna kuga)	
c	Куга Kuga	Pest
4	Колера Kolera	Asiatisk kolera
5		Undulantfeber
a	Малтеска грозница Malteska groznica (Malteska grosnitsa)	
b		
c		

Tchèque — Czech	Turc — Turkish	N°
Týf břišní Týf střevní	Karahumma	1
Paratyf Paratyf	Paratifüs	2
Mor	Veba	3
Morová rána Morová nákaza		*a(a)*
		a(b)
		a(c)
Plicní mor		*b*
Mor	Veba	*c*
Cholera	Kolera	4
		5
	Malta hummasi	*a*
		b
		c

N°	Nomenclature internationale International Nomenclature	Latin
6	**Méningite cérébro-spinale (méningococcique)**	Meningitis cerebrospinalis epidemica Meningitis meningococcica
7	**Pustule maligne et charbon** *(Bacillus anthracis)*	Anthrax contagiosus Anthrax
a	Pustule maligne	Pustula maligna Carbunculus malignus Carbunculus anthracis
b	Formes septicémiques et localisations viscérales	Anthrax visceralis
c	sans autre spécification	Anthrax Anthrax contagiosus
8	**Scarlatine**	Febris scarlatinosa Scarlatina Febris rubra
9	**Coqueluche**	Tussis convulsiva
10	**Diphtérie**	Diphtheria crouposa Diphtheria
11	**Erysipèle**	Erysipelas
12	**Tétanos**	Tetanus Trismus neonatorum

Allemand — German	Anglais — English	N°
Übertragbare Genickstarre Genickstarre Epidemische cerebrospinal Meningitis	Cerebro-spinal meningitis Cerebro-spinal fever Meningococcal meningitis	6
Milzbrand	Anthrax	7
Pustula maligna	Malignant pustule	*a*
	Visceral anthrax	*b*
Anthrax contagiosus	Anthrax u.	*c*
Scharlach Scarlatina	Scarlet fever Scarlatina	8
Keuchhusten Pertussis	Whooping-cough Pertussis	9
Diphtherie Halsbräune (v) Krupp * Ansteckender Krupp *	Diphtheria Malignant angina (v) Croup *	10
Erysipel Rose (v) Rotlauf	Erysipelas	11
Tetanus Wundstarrkrampf Starrkrampf	Tetanus Lockjaw (v) Trismus neonatorum	12

N°	Bulgare — Bulgarian	Danois et norvégien Danish and Norwegian
6	Епидемиченъ менингитъ Epidemitchen meningit	Epidemisk Hjaerne-Rygmarvsbetændelse Epidemisk Cerebrospinalmeningitt
7	Антраксъ Antraks	Miltbrand
a	Синята пжпка Sinyata pjepka	Brandbyld
b		
c	Антраксъ Antraks	Miltbrand
8	Скарлатина Skarlatina	Skarlagensfeber
9	Лоша кашлица Locha kachlitsa Магарешка кашлица Magarechka kachlitsa	Kighoste Kikhoste
10	Лошо гърло Locho gerlo Дифтерия Diphteria	Difteri Strubehoste
11	Червенъ ветеръ Tcherven veter	Rosen
12	Тетанусъ Tetanus	Stivkrampe Stivkrampe hos nyfödte

Espagnol — Spanish	Estonien — Estonian	N°
Meningitis cerebro-espinal epidemica Meningitis meningocócica Meningococia	Epideemiline ajukelme meningiit Epideemiline tserebrospinaalne meningiit	6
Carbón	Antraks	7
Pústula maligna	Porntõbi	*a*
		b
Carbón	Antraks	*c*
Escarlatina Fiebre escarlatinosa	Sarlakid	8
Coqueluche Tos ferina	Läkaköha	9
Difteria Angina lardacea Laringitis Pseudo-membranosa Laringitis maligna Laringitis diftérica	Difteeria	10
Erisipela Flegmon erisipelatoso	Roos Erüsiipel	11
Tetanos Trismo de los recién nacidos	Kangestustõbi Teetanus	12

N°	Finnois — Finnish	Français — French
6	Kulkutaudinluontoinen aivokalvontulehdus	Méningite cérébrospinale épidémique Méningococcie Méningite basilaire
7		Charbon
a		Pustule maligne
b		Charbon interne Charbon alimentaire Fièvre splénique Septicémie charbonneuse
c		
8	Tulirokko	Fièvre scarlatine
9	Hinkuyskä	Coqueluche
10	Kurkkumätä Kuristustauti	Diphtérie Croup (vrai) * Angine à fausses membranes Angine couenneuse (v)
11	Ruusu	Erysipèle
12	Jäijkkäkouristus	Tétanos Trismus des nouveau-nés

Grec — Greek	Hongrois — Hungarian	Islandais — Icelandic	N°
Μηνιγγῖτις ἐγκεφαλονωτιαια ἐπιδημικὴ Meniggitis enkephalonotiaia epidemikè	Járvany agyhartyalob Járvany gerinczagyhártyalob Járvanyos gerinczvelőgyulladas Járvanyos agy- és gerincvelőburokgyulladas	Heilasótt	6
Κακοήθης φλύκταινα, Kakoedes phlyktaina	Lépfene		7
Ανθραξ Anthrax			
	Pokolvar		*a*
	Lépfene		*b*
	Lépfene		*c*
Οστρακιὰ Ostrakia	Vőrheny Skarlát	Skarlatsótt	8
Κοκκῦτις Kokkytis	Szamárhurut Hökhurut	Kikhósti	9
Διφθερῖτις Diphtheritis	Roncsoló toroklob Difteria Croup	Barnaveiki	10
Ερυσίπελας Erysipelas	Orbánc	Heimakoma	11
Τέτανος Tetanos	Tetanus Dermedés (fertőző) Fertőzeses merevgörcs		12

N°	Italien — Italian	Letton — Latvian
6	Meningite cerebrospinale epidemica Meningite meningococcica	Epidēmiskais galvas un muguras smadzenu plēves iekaisums
7	Carbonchio	Liesas sērgas bacillis
a	Pustola maligna	Liesas sērga ādā Karbunkuls
b	Carbonchio ematico Setticemia carbonchiosa	
c		
8	Scarlattina	Šarlaks
9	Pertosse Tosse convulsa Tosse ferina Tosse convulsiva Tosse asinina	Garais klepus
10	Difterite Croup* Laringite maligna (v.) Angina difterica Laringite pseudomembranosa	Difterija
11	Risipola Erisipela	Roze
12	Tetano Trisma dei neonati	Tetanos

Lithuanien — Lithuanian	Néerlandais — Dutch	N°
Epid. smegenų plévés uždegimas	Epidemische meningitis Nekkramp (v.)	6
Juodligé Sibiro maras	Miltvuur Anthrax	7
Sibiro maras	Carbunkel Miltvuurpuisten	*a*
	Septische miltvuur Gastrointestinale miltvuur Ingewandsmiltvuur	*b*
		c
Skarlatina	Roodvonk Scarlatina	8
Kokliušas Didysis kosulys	Kinkhoest	9
Difteritas	Diphtherie Diphtheria Diphteritis	10
Rože	Erysipelas Roos Belroos	11
Stabas (méšlungis)	Tetanus Klem Stijfkramp	12

N°	Polonais — Polish	Portugais — Portuguese
6	Zapalenie opon mózgowych nagminne (Epidemiczne)	Meningite cerebro-espinhal epidemica Molestia de Weichselbaum Meningite pelo meningococo
7	Wąglik	Carbunculo
a		Pustula maligna
b		
c		
8	Płonica Szkarlatyna	Escarlatina
9	Krztusiec Koklusz	Coqueluche Tosse convulsa
10	Błonica Dyfteria	Diphteria Angina membranosa (v) Crupe (v) * Difteria
11	Róża	Erysipéla
12	Tężek	Tetano Trismo Trismo dos recemnatos

Roumain — Roumanian	Russe — Russian	N°
Meningită cerebro-spinală (meningococică)	Менингококковый менингит — Meningokokkovyï meningit Эпидемический цереброспинальный менингит — Epidemitcheskiï cerebrospinalnyï meningit	6
Cărbune Infecţei prin *B. anthracis*		7
Pustulă malignă Buba neagră	Антракс — Antrax Сибирская язва — Sibirskaïa iazva Злокачественная язва — Zlokatchestvennaïa iazva	*a*
Carbune septicemico Carbune visceral		*b*
	Антракс — Antrax	*c*
Scarlatină	Скарлатина — Skarlatina Краснуха — Krasnukha (v)	8
Tuse convulsiva	Коклюш — Koklioucн	9
Difterie Crup* Angina difterică	Дифтерит — Difterit Круп — Krup Перепоночная жаба – Pereponotchnaïa jaba	10
Erizipel	Рожа — Roja	11
Tetanos	Столбняк — Stolbniak Тризм новорожденных — Trizm novorojdennykh	12

N°	Serbo-croate — Serbo-Croatian	Suédois — Swedish
6	Запаљење мозга Zapaljenje mozga	Hjärnfeber (Epidemisk —) Epidemisk Hjärnhinneinflammation
7	Антракс Antraks	Mjältbrand
a	Црни пришт Crni prišt (Tsrni prisht)	
b		
c	Антракс Antraks	Mjältbrand
8	Скрлет Skrlet Шарлах Šarlah (Sharlah)	Skarlakansfeber Scharlakansfeber
9	Велики кашаљ Veliki kašalj (Veliki kashalj) Pertusis	Kikhosta
10	Гушобоља Gušobolja (Gushobolja) Дифтерија Difterija	Difteri
11	Црвени ветар Crveni vetar (Tsrveni vetar)	Ros
12	Тетанус Tetanus	Stelkramp

Tchèque — Czech	Turc — Turkish	N°
Epidemický zánět plen mozkových a míšních Epidemické ztrnutí šíje	Sarî sehaya iltihabi	6
Uhlák	Şarbon	7
Uhlák		*a*
Sněť slezinná		*b*
Uhlák	Şarbon	*c*
Spála	Kizil	8
Zajikavý kašel Davný kašel Kašel zádušný Černý kašel (v)	Boğmaca öksürüğü	9
Záškrt Mázdřivka (v)	Difteri	10
Růže	Yilancik	11
Ztrnutí ranné Tetanus	Tetanoz	12

N°	Nomenclature internationale International Nomenclature	Latin
13	**Tuberculose de l'appareil respiratoire** (y compris ganglions trachéo-bronchiques)	Tuberculosis organorum respirationis
a	Avec mention de maladie professionnelle du poumon	
b	Sans mention de maladie professionnelle du poumon	
b(a)	*Larynx*	Tuberculosis laryngis
b(b)	*Trachée et bronches*	Bronchitis tuberculosa
b(c)	*Poumons*	Phtisis pulmonalis Pneumonia caseosa Pneumonia tuberculosa Pneumonia alba Tuberculosis pulmonum Caverna pulmonis Catarrhus apicis pulmonis Haemoptysis tuberculosa Haemoptysis n. s.
b(d)	*Ganglions trachéobronchiques*	
b(e)	*Localisation mixte ou non spécifiée*	
c	Tuberculose non spécifiée	Phtisis
14	**Tuberculose des méninges** et du système nerveux central	Tuberculosis systematis nervorum centralis et meningum
a	Méninges	Meningitis tuberculosa Tuberculosis meningum
b	Autres localisations	Encephalitis tuberculosa Tuberculosis cerebri

Allemand — German	Anglais — English	N°
Tuberkulose der Atmungsorgane	Tuberculosis of the respiratory system	**13**
		a
		b
Kehlkopftuberkulose Kehlkopfschwindsucht (v)	Laryngeal tuberculosis	*b(a)*
	Tubercular bronchitis	*b(b)*
Lungentuberkulose Tuberkulöse Lungenentzündung Käsige Lungenentzündung	Caseous pneumonia Hæmoptysis (u.) Tuberculosis of the lungs Pulmonary tuberculosis Pulmonary consumption	*b(c)*
Bronchialdrüsentuberkulose	Tuberculosis of mediastinal glands Hilum tuberculosis	*b(d)* *b(e)*
Schwindsucht (v) Phtise (v)	Tuberculosis (u.) Phtisis (v) Consumption (v) Tubercle (v)	*c*
Tuberkulose der Hirnhaut Tuberkulose des Gehirns	Tuberculosis of the meninges and central nervous system	**14**
Tuberkulöse Hirnhautentzündung Meningitis tuberculosa	Meningeal tuberculosis Tuberculous meningitis	*a*
Tuberkulose des Gehirns	Tuberculosis of the brain	*b*

N°	Bulgare — Bulgarian	Danois et norvégien Danish and Norwegian
13	Туберкулоза на дихателните органи Tuberkuloza na dikhatelnite organi	Tuberkulose i Aandedrætsorganerne
a		
b		
b(a)		Strubetuberkulose Strupetuberkulose
b(b)		
b(c)		Lungetuberkulose Brystsyke (v) Lungeblödning
b(d)		
b(e)		
c		Tæring (v) Svindsot (v)
14	Туберкулоза на менингите и центр. нервна система Tuberkuloza na meningite i centr. nervna sistema	Tuberkulose i Nervesystemet
a	Туберкулоза на менингите Tuberkuloza na meningite	Tuberkulose i Hjærnehinder Tuberkulös Meningitt
b		Tuberkulose i Hjærne Hjærnetuberkulose

Espagnol — Spanish	Estonien — Estonian	N°
Tuberculosis del aparato respiratorio	Hingamis organite tuberkuloos	**13**
		a
		b
Laringitis tuberculosa		*b(a)*
Bronquitis especifica* Bronquitis neoplásica* Bronquitis bacilar Hemoptisis Pneumonia especifica* Tuberculosis pulmonar Pneumonia caseosa Cavernas pulmonares*	Hingamisteede tuberkuloos	*b(b)* *b(c)*
Adenopatia traqueo-brónquica		*b(d)*
		b(e)
Consunción Tisis (v) Bacilosis* Fimatosis*	Tuberkuloos	*c*
Tuberculosis de las meningeas y del sistema nervoso central		**14**
Meningitis bacilar Meningitis tuberculosa Meningitis miliar	Tuberkuloosne meningiit	*a*
Tuberculosis del cerebro	Tuberkuloosne entsefaliit	*b*

N°	Finnois — Finnish	Français — French
13	Keuhkotauti	Tuberculose respiratoire
a		
b		
b(a)		Tuberculose laryngée Laryngite tuberculeuse
b(b)		Bronchite tuberculeuse
b(c)	Keuhkotauti	Tuberculose pulmonaire Pneumonie caséeuse Hémoptysie s.a.i. Hémorragies pulmonaires s.a.i. Cavernes pulmonaires s.a.i.
b(d)		Adénopathie trachéo-bronchique s.a.i.
b(e)		
c		Phtisie (v) Consomption (v) Bacillose
14	Tuberkuloottinen aivokalvontulehdus	
a		Méningite tuberculeuse Méningite bacillaire Méningite caséeuse Méningite miliaire Tuberculose méningée
b		

Grec — Greek	Hongrois — Hungarian	Islandais — Icelandic	N°
Φυματίωσις ἀναπνευτικοῦ συστήματος Phymatiosis anapneutikou systematos	A legzőszervek gümőkórja	Berklaveiki	**13**
			a
			b
			b(a)
			b(b)
	Tüdocsúcshurut Tüdőgümőkór Tüdővész		*b(c)*
			b(d)
			b(e)
Φτισις Phtisis	Gümőkór		*c*
Φυματίωσι, τῶν μηνιγγων Phymatiosis ton meniggon	Az agyvelőburkok és a központi idegrendszer gümőkórja		**14**
	Heveny agyvizkór Agyhártyagümősödes Agyhártyagümőkór Agygümőkór		*a*
			b

N°	Italien — Italian	Letton — Latvian
13	Tubercolosi dell'apparato respiratorio	Elpošanas organu tuberkuloze
a		
b		
b(a)	Laringite tubercolare	
b(b)	Bronchite bacillare Bronchite specifica*	
b(c)	Tubercolosi polmonare Polmonite caseosa Emottisi tubercolare Emottisi* s.a.i. Caverne polmonari *	Plaušu tuberkuloze
b(d)	Adenopatia tracheobronchiale	
b(e)		
c	Tisichezza (v) Tisi (v) Consunzione (v)	Dilonis
14	Tubercolosi delle meningi e del sistema nervoso centrale	
a	Meningite tubercolare Meningite miliare Meningite caseosa	Smadzeņu plèves tuberkuloze
b	Tubercolosi del cerebro Tubercolosi del cervello	Smadzeņu tuberkuloze

Lithuanien — Lithuanian	Néerlandais — Dutch	N°
Kvepuoj. org. tuberkul.	Tuberculose van de ademhalingsorganen en de bronchiaalklieren	**13**
		a
		b
	Keeltering (v)	*b(a)*
	Tuberculose van het strottenhoofd	*b(b)*
	Longtering (v) Tuberculose van de longen Longtuberculose Longblœdingen z.n.a. Hemoptoe	*b(c)*
	Tuberculose van de longklieren Tuberculose van de bronchiaalklieren	*b(d)* *b(e)*
Dziova	Tering (v) T. B. C. Vliegende tering (v) Algemeene tuberculose	*c*
	Tuberculose van de meningen en van het centraal zenuwstelsel	**14**
	Tuberculose van de meningen Tuberculeuze hersenvliesontsteking	*a*
	Tuberculose hersenontsteking Hersentuberculose	*b*

N°	Polonais — Polish	Portugais — Portuguese
13	Gruźlica narządu oddechowego	Tuberculose do aparelho respiratorio
a	Z wyszczególnieniem choroby zawodowej płuc	
b	Bez wyszczególnienia choroby zawodowej płuc	
b(a)		Laryngite tuberculosa
b(b)		Bronquite tuberculosa
b(c)		Pneumonia caseosa Tuberculose pulmonar Tuberculose dos pulmões
b(d)		
b(e)		
c	Suchoty	Consumpção * Tisica* Phtisica* Bacilose*
14	Gruźlica opon mózgowych i systemu nerwowego ośrodkowego	Tuberculose dos meninges ou do systema nervoso central
a		Meningite tuberculosa Meningite miliar
b		

Roumain — Roumanian	Russe — Russian	N°
Tuberculoza aparatului respirator (aici sunt cuprinşi si ganglionii traheobronşici)	Бугорчатка органов дыхания — Bugortchatka organov dykhania	**13**
		a
		b
Laringită tuberculoasă	Бугорчатка гортани — Bugortchatka gortani	*b(a)*
Bronşită tuberculoasă	Бронхит туберкулезный — Bronkhit tuberkulioznyï	*b(b)*
Tuberculoza pulmonară Hemoptizie*	Бугорчатка легких — Bugortchatka legkikh Катарр верхушек легких — Katarr verkhuchek legkikh Верхушечная пневмония — Verkhuchetchnaïa pnevmonia Кровохаркание туберкулезное — Krovokharkanie tuberkulioznoie	*b(c)*
		b(d)
		b(e)
Ftizie (v) Oftică Slăbire (v) Uscare Topire	Чахотка — Tchakhotka (v) Сухотка — Sukhotka (v) Чахлость — Tchakhlost (v)	*c*
Tuberculoza meningelui şi a sistemului nervos central	Бугорчатка мозговых оболочек и центральной нервной системы — Bugortchatka mozgovykh obolotchek i centralnoi nervnoï sistemy	**14**
Tuberculoza meningelor Meningită tuberculoasa	Туберкулезный менингит — Tuberkuliosnyï meningit	*a*
		b

N°	Serbo-croate — Serbo-Croatian	Suédois — Swedish
13		
a		
b		
b(a)		Struptuberkulos
b(b)		
b(c)	Туберкулоза плућа Tuberkuloza pluća (Tbk. plucha)	Lungtuberkulos Lungsot
b(d)		
b(e)		
c	Туберкулоза Tuberkuloza Сушица Sušica (Sushitsa) Јектика Jektika Јехтика Jehtika	Tuberkulos
14		
a	Туберкулозно запаљење мозга Tuberkulozno zapaljenje mozga	Hjärnhinnetuberkulos
b		Hjärntuberkulos

Tchèque — Czech	Turc — Turkish	N°
Tuberkulosa ústrojí dýchacího	Verem	**13**
		a
		b
		b(a)
		b(b)
Tuberbulosa plic Hæmoptœ	Verem	*b(c)*
		b(d)
		b(e)
Úbytě Souchotiny Tuberkulosa		*c*
Tuberkulosa ústřední soustavy čivové		**14**
Tuberkulosa mozkových blan		*a*
		b

N°	Nomenclature internationale International Nomenclature	Latin
15	**Tuberculose des intestins et du péritoine** (y compris ganglions mésentériques et rétropéritonéaux)	Tuberculosis viscerum abdominalium
(a)	*Intestin*	Tuberculosis intestini
(b)	*Péritoine*	Ascites tuberculosus Tuberculosis peritonei Tabes mesaraica Tabes mesenterica
(c)	*Localisation mixte ou non spécifiée*	Tuberculosis abdominalis
16	**Tuberculose de la colonne vertébrale**	Tuberculosis vertebralis Tuberculosis columnae vertebrarum Malum Pottii, Morbus Pottii
17	**Tuberculose des os et des articulations** (excepté colonne vertébrale)	Tuberculosis ossium et articulationum
a	Os (excepté colonne vertébrale)	Tuberculosis ossium Osteomyelitis tuberculosa Arthritis tuberculosa Ostitis tuberculosa
b	Articulations	Tuberculosis articulationum Morbus coxae Tumor albus Coxitis tuberculosa
c	Mixte ou non spécifiée	Abscessus frigidus
18	**Tuberculose de la peau et du tissu cellulaire sous-cutané**	Tuberculosis cutis Tuberculosis telae cellulosae Lupus Lupus vulgaris Lupus exedens

Allemand — German	Anglais — English	N°
Tuberkulose der Unterleibsorgane	Tuberculosis of the intestines and peritoneum	**15**
Darmtuberkulose	Intestinal tuberculosis Tuberculosis enteritis	*(a)*
Peritoneal Tuberkulose Tuberkulose des Peritoneums Schwindsucht der Gekrösedrüsen Tabes mesaraica	Peritoneal tuberculosis Tabes mesenterica	*(b)*
Tuberkulose des Bauchfells Bauchfelltuberkulose	Abdominal consumption (v) Phtisis enterica	*(c)*
Tuberkulose der Wirbelsäule Pottsche Krankheit	Tuberculosis of the vertebral column Pott's disease, Tuberculous spondylitis Vertebral caries Psoas abscess Vertebral tuberculosis	**16**
Tuberkulose der Knochen	Tuberculosis of other bones and joints	**17**
Tuberkulöser Knochenfrass (v)	Cold abscess (v) Tuberculous osteomyelitis	*a*
Gelenktuberkulose	Articular tuberculosis Tuberculosis of joints Tuberculous arthritis Morbus coxae	*b* *c*
Tuberkulose der Haut Tuberkulose des Unterhautzellgewebes Lupus	Tuberculosis of the skin and subcutaneous cellular tissue Lupus Scrofuloderma Tuberculide	**18**

N°	Bulgare — Bulgarian	Danois et norvégien Danish and Norwegian
15		
(a)		Tarmtuberkulose
(b)		Bukhindetuberkulose
(c)		Buktuberkulose Bugtuberkulose
16		Rygmarvstuberkulose Ryggmarvstuberkulose
17		Tuberkulose i knogler og led
a		Tuberkulose i Knokler Tuberkulose i Knogler Tuberkulös Benmarvsbetændelse
b		Tuberkulose i Ledd Ledtuberkulose
c		
18		Tuberkulose i Hud og det subkutant Vaev Lupus Hudtuberkulose

Espagnol — Spanish	Estonien — Estonian	N°
Tuberculosis de los intestinos y del peritoneo	Soolte ja kõhukelme tuberkuloos	**15**
Enteritis tuberculosa		*(a)*
Atrofia mesenterica Ascitis bacilar Peritonitis bacilar Peritonitis fibro-caseosa		*(b)*
Tuberculosis abdominal		*(c)*
Tuberculosis de la columna vertebral Carie vertebral Mal de Pott Mal vertebral*	Lülisamba tuberkuloos	**16**
Tuberculosis de los huesos y de las articulaciones	Luude ja liigeste tuberkuloos	**17**
Absceso frió Absceso osifluente Absceso por congestión Tuberculosis ósea	Luude tuberkuloos	*a*
Artrocace Tumore blanco Artritis tuberculosa	Liigeste tuberkuloos	*b*
		c
Tuberculosis de la piel y del tejido celular sub-cutaneo Absceso bacilar Lupus Ulcera tuberculosa Ulcera bacilar	Naha tuberkuloos Nahaaluse koe tuberkuloos põletik	**18**

N°	Finnois — Finnish	Français — French
15		
(a)		Tuberculose intestinale Entérite tuberculeuse
(b)		Péritonite tuberculeuse Péritonite bacillaire Carreau (v) Ascite tuberculeuse Ascite bacillaire
(c)		Tuberculose abdominale
16		Tuberculose vertébrale Mal de Pott Spondylite tuberculeuse Abcès du psoas
17		Tuberculose des os et des articulations
a		Ostéomyélite tuberculeuse Tuberculose osseuse Abcès ossifluent
b		Arthrite tuberculeuse Coxalgie tuberculeuse Tumeur blanche
c		Rhumatisme tuberculeux Abcès froid Abcès par congestion* Ostéo-arthrite tuberculeuse
18		Tuberculose cutanée Lupus Scrofulodermie

Grec — Greek	Hongrois — Hungarian	Islandais — Icelandic	N°
Φυματιωσις τῶν εντερων Phymatiosis ton enteron			15
Φυματιωσις τοῦ περιτοναιου Phymatiosis tou peritonaiou			
	Bélgümőkór		*(a)*
	Hashártyagümőkór		*(b)*
	Belfodormirigysorvadás		*(c)*
Φυματιωσις της σπονδυλικῆς στηλης Phymatiosis tes spondylikes steles	Csigolyagümőkór		16
	A csontok és az izületek gümőrkórja		17
Φυματιωσις τῶν ὀστῶν Phymatiosis ton oston	Csontgümokór		*a*
Φυματιωσις τῶν αρθρωσεων Phymatiosis ton arthroseon	Az izütelek gümokórja		*b*
			c
Φυματιωσις τοῦ δερματος Phymatiosis tou dermatos	A bőr és bőralatti kötőszövet gümőkórja Bőrgümősödés		18

N°	Italien — Italian	Letton — Latvian
15	Tubercolosi degli intestini e del peritoneo	
(a)	Tubercolosi degli intestini Enterite tubercolare	
(b)	Tubercolosi del peritoneo Ascite tubercolare Tubercolosi mesenterica	
(c)	Tubercolosi addominale	
16	Tubercolosi della colonna vertebrale Morbo di Pott	
17	Tubercolosi delle ossa e delle articolazioni	Kaulu un locitavu tuberkuloze
a	Tubercolosi ossea Ascesso ossifluente Ascesso congestizio*	Kaulu tuberkuloze
b	Ascesso delle articolazione Artrite tubercolare Tumori bianchi Artrocace	Locitavu tuberkuloze
c		
18	Tubercolosi della pelle e tubercolosi sottocutaneo Lupus Tubercolosi cutanea Ulcera tubercolare	Ādas tuberkuloze

Lithuanien — Lithuanian	Néerlandais — Dutch	N°
		15
	Tuberculose van de ingewanden	*(a)*
	Buikvliestuberculose Tuberculose van het buikvlies	*(b)*
	Buiktuberculose Tuberculose van de buikklieren	*(c)*
	Tuberculose van de wervelkolom Pottsche ziekte	**16**
	Beenderentuberculose	**17**
	Tuberculose van de beenderen	*a*
	Tuberculose van de gewrichten Gewrichtstuberculose	*b*
		c
	Tuberculose van de huid en van het onderhuidsche celweefsel Lupus Huidtuberculose	**18**

N°	Polonais — Polish	Portugais — Portuguese
15		Tuberculose dos intestinos ou do peritoneo
(a)	Gruźlica jelit	Enterite tuberculosa
(b)	Gruźlicze zapalenie otrzewnej	Tuberculose mesenterica Peritonite tuberculosa
(c)		
16	Gruźlica kręgosłupa Próchnica kręgów Choroba Potta	Tuberculose da columna vertebral Doença de Pott
17	Gruźlica kości i stawów	Tuberculose dos ossos
a	Próchnica kości (zimny ropień)	Abcesso frio
b	Gruźlicze zapalenie stawów	Tuberculose articular Arthritis tuberculosa
c		Tumor branco
18	Gruźlica skóry i tkanki podskórnej wilk	Tuberculose da pelle ou do tecido cellular subcutaneo Loba

Roumain — Roumanian	Russe — Russian	№
Tuberculoza intestinelor şi a peritoneului	Бугорчатка кишек и брюшины — Bugortchatka kichek i briuchiny	**15**
Tuberculoza intestinelor	Бугорчатка кишек — Bugortchatka kichek	*(a)*
Tuberculoza peritoneului	Бугорчатка перитонита — Bugortchatka peritonita Туберкулезный перитонит — Tuberkulioznyï peritonit	*(b)*
Tuberculoza abdominal		*(c)*
Tuberculoza coloanei vertebrale Morbul (Boala) lui Pott	Бугорчатка позвоночника — Bugortchatka pozvonotchnika Болезнь Поттова — Bolezn Pottova	**16**
Tuberculoza oaselor şi a articulaţiilor	Бугорчатка костей и суставов — Bugortchatka kosteï i sustavov	**17**
Tuberculoza oaselor	Бугорчатка костей – Bugortchatka kosteï Туберкулезный нарыв — Tuberkulioznyï naryv	*a*
Tuberculoza a articulaţiilor	Бугорчатка сочленений — Bugortchatka sotchleneniï Бугорчатка суставов — Bugortchatka sustavov	*b*
Tuberculoza osteo-articulară		*c*
Tuberculoza pielei şi a ţesutului celular subcutanat Lupus	Бугорчатка кожи и подкожной клетчатки — Bugortchatka koji i podkojnoi klettchatki Волчанка — Voltchanka	**18**

N°	Serbo-croate — Serbo-Croatian	Suédois — Swedish
15		
(a)	Туберкулоза црева Tuberkuloza creva (Tbk. tsreva)	Tarmtuberkulos
(b)		Bukhinnetuberkulos
(c)		
16	Туберкулоза хрптењаче Tuberkuloza hrptenjače (Tuberkuloza hrptenjatche) Туберкулоза кичме Tuberkuloza kičme (Tuberkuloza kitchme)	Pottssjukdom
17		
a	Туберкулоза костију Tuberkuloza kostiju	Bentuberkulos
b	Туберкулоза зглобова Tuberkuloza zglobova	Ledgångstuberkulos
c		
18	Туберкулоза коже Tuberkuloza kože (Tbk. koje)	Hudtuberkulos

Tchèque — Czech	Turc — Turkish	N°
	Barsak veremi	**15**
Tuberkulosa střev		*(a)*
Tuberkulosa pobřišnice		*(b)*
		(c)
Tuberkulosa páteře Tuberkulosa obratlů	Amudi fukari veremi	**16**
Tuberkulosa kostí a kloubů	Kemik veremi	**17**
Tuberkulosa kostí		*a*
Tuberkulosa kloubů		*b*
		c
Tuberkulosa kůže a podkožního vaziva Tuberkulosa kůže Lupus	Cilt veremi	**18**

N°	Nomenclature internationale International Nomenclature	Latin
19	**Tuberculose du système lymphatique**	Scrofulosis Adenitis tuberculosa Tuberculosis systematis lymphatici Lymphangitis tuberculosa
20	**Tuberculose de l'appareil génito-urinaire**	Tuberculosis systematis urogenitalis Tuberculosis urogenitalis Epididymitis tuberculosa Orchitis tuberculosa
21	**Tuberculose : autres localisations**	Ceterae affectiones tuberculosae
a	Maladie d'Addison spécifiée comme d'origine tuberculeuse	Morbus Addisonii tuberculosus
b	Autres	
22	**Tuberculose disséminée**	Tuberculosis disseminata
a	Aiguë (miliaire)	Tuberculosis miliaris acuta Tuberculosis miliaris n. s.
b	Chronique	Tuberculosis miliaris chronica
c	Non spécifiée	Tuberculosis disseminata
23	**Lèpre**	Lepra Elephantiasis Graecorum

Allemand — German	Anglais — English	N°
Tuberkulose der Drüsen Skrofulose (v) Skrofeln (v)	Tuberculosis of the lymphatic system Scrofula* Struma* Glandular tuberculosis Lymphatic tuberculosis Tuberculous adenitis	**19**
Tuberkulose der Harn- und Geschlechtsorgane	Tuberculosis of the genito-urinary system Genital tuberculosis Urinary tuberculosis Tuberculous epididymitis	**20**
		21
Tuberkulöse Addisonsche Krankheit	Tuberculous Addison's disease Tuberculosis of adrenals	*a*
		b
Miliartuberkulose	Disseminated tuberculosis	**22**
Akute Miliartuberkulose	Acute miliary tuberculosis Acute generalised tuberculosis	*a*
Chronische Miliartuberkulose	Chronic miliary tuberculosis	*b*
	Diffuse tuberculosis Generalised tuberculosis	*c*
Lepra Aussatz (v)	Leprosy	**23**

N°	Bulgare — Bulgarian	Danois et norvégien Danish and Norwegian
19		Skrofulose
20		Urogenitaltuberkulose Tuberkulose i Könsorganerne og Urinorganerne Tuberkulös Blærebetændelse Tuberkulose i Urinveiene
21	Всички други видове туберкулоза Vsitchki drugi vidove tuberkulosa	
a		Tuberkulös Addisons Sygdom
b		
22		
a		Miliærtuberkulose
b		
c		
23	Проказа Prokaza	Spedalskhet Spedalskhed

Espagnol — Spanish	Estonien — Estonian	N°
Tuberculosis del sistema linfatico Absceso escrofuloso Absceso estrumoso Bubón escrofuloso Tuberculosis ganglionar Adenitis estrumosa Escrofula*	Tuberkuloosne mahlanäärmete	**19**
Tuberculosis del aparato genito-urinario Epididimitis caseosa Nefritis tuberculosa Cistitis tuberculosa	Uro-genitaalne tuberkuloos	**20**
Tuberculosis en otras localizaciones	Tuberkuloos mujal lokaliseerunud	**21**
Enfermedad de Addison de origen tuberculoso		*a*
		b
Tuberculosis diseminada	Laialikülvunud tuberkuloos	**22**
Tuberculosis diseminada aguda Granulia Tuberculosis miliar	Äge laialikülvunud tuberkuloos	*a*
	Krooniline laialikülvunud tuberkuloos	*b*
Tuberculosis generalizada s.a.i.		*c*
Lepra	Pidalitõbi Leepra	**23**

N°	Finnois — Finnish	Français — French
19	Risatauti	Tuberculose ganglionnaire Tuberculose lymphatique Adénite tuberculeuse Adénite bacillaire Abcès bacillaire Abcès scrofuleux* Abcès strumeux*
20		Tuberculose génitale Tuberculose urinaire Épididymite tuberculeuse Salpingite tuberculeuse Cystite tuberculeuse
21	Muu tuberkuloosi	Tuberculose ; autres localisations
a		Paralysie agitante tuberculeuse Tuberculose surrénale
b		
22		
a		Tuberculose miliaire Granulie
b		
c		Tuberculose multiple Tuberculose diffuse Tuberculose généralisée
23	Spitaalitauti	Lèpre Lèpre tuberculeuse

Grec — Greek	Hongrois — Hungarian	Islandais — Icelandic	N°
Φυματιωσις τοῦ λεμφικοῦ συστηματος Phymatiosis tou lemphikou systematos	Görvélykor vagy a nyirokerrendszer gümőkórja Görvélykór Mirigygümő		19
Φυματιωσις τοῦ οὐροποιογεννητικοῦ συστηματος Phymatiosis tou ouropoiogennetikou systematos	A hugyiverszervek gümőkórja		20
	Egyeb szervek gümókórja Összes egyeb gümőkór Egyeb gümőkóri bántalmak		21
Φυματιωσις ἑτέρων οργανων Phymatiosis eteron organon	Gumős Addisonkór		*a*
			b
Φυματιωσις διεσπαρμένη Phymatiosis diesparmeni	Altalános gümőkór		22
	Heveny altalános gümőkór		*a*
	Idült altalános gümőkór		*b*
			c
Λέπρα Lepra	Bélpoklosság Lepra	Holdsveiki	23

N°	Italien — Italian	Letton — Latvian
19	Tubercolosi del sistema linfatico Tubercolosi ganglionare Adenite tubercolare Adenite scrofolosa* Adenite strumosa* Scrofola* (v)	Dziedzeru tuberkuloze
20	Tubercolosi dell'apparato genito-urinario Epididimite caseosa Pielonefrite tubercolare Cistite tubercolare	
21	Altre localizzazioni della tubercolosi	Pārejo organu tuberkuloze
a	Malattia (tubercolare) di Addison Tubercolosi delle ghiandole surrenali	
b		
22		
a	Tubercolosi disseminata acuta Tubercolosi miliere acuta Tifobacillosi di Landouzy	
b	Tubercolosi disseminata cronica	
c	Tubercolosi disseminata Tubercolosi generalizzata	
23	Lebbra Elefantiasi dei Greci (v)	Spitālība Lepra

Lithuanien — Lithuanian	Néerlandais — Dutch	N°
	Tuberculose van het lymphvatenstelsel Klierziekte Scrophulose	**19**
	Tuberculose van de urogenitaalorganen Tuberculose van de nier Tuberculose van de blaas Tuberculose van de geslachtsorganen	**20**
Kitos Tuberkulozes	Tuberculose van de overige organen	**21**
	Morbus addisonii tuberculosus Tuberculeuze ziekte van Addison	*a*
		b
	Gedissemineerde tuberculose	**22**
	Acute miliair tuberculose	*a*
	Chronische gedissemineerde tuberculose	*b*
	Gedissemineerde tuberculose z. n. a.	*c*
Raupsai	Lepra Melaatschheid	**23**

Nº	Polonais — Polish	Portugais — Portuguese
19	Gruźlica gruczołów limfatycznych zołzy	Tuberculose do systema lymphatico Escrofula* Alporcas*
20	Gruźlica narządu moczo-płciowego	Tuberculose do aparelho genito-urinario Nephritis tuberculosa
21	Gruźlica nadnercza	Tuberculose de outros orgãos
a	Choroba Addisona na tle gruźliczym (cisawica)	Molestia de Addison tuberculosa
b		
22	Gruźlica rozsiana	Tuberculose disseminada
a	Ostra prosowkowa	Tuberculose disseminada aguda Tuberculose miliar
b	Chroniczna	Tuberculose disseminada chronica
c		
23	Trąd Lepra	Lepra

Roumain — Roumanian	Russe — Russian	N°
Tuberculoza sistemului limfatic (fără ganglionii traheobronşici, mezenterici si retroperitoneali)	Бугорчатка лимфатической системы — Bugortchatka limfatitcheskoï sistemy Холодный нарыв — Kholodnyï naryv Натечный нарыв — Natechnyï naryv Лимфатизм — Limfatizm Золотуха — Zolotukha (v) Скрофулез — Skrofuloz (v)	19
Tuberculoza aparatului genito-urinar	Бугорчатка мочеполовых органов — Bugortchatka motchepolovych organov	20
Tuberculoza altor localizări	Бугорчатка : другие локализации — Bugortchatka : druguie lokalizatsii	21
Boala lui Addison specificată de origină tuberculoasă	Бугорчатка надпочечных желез — Bugortchatka nadpotchechnykh jelez Болезнь Аддисона — Bolezn Addisona Бронзовая болезнь — Bronzovaïa bolezn	*a*
		b
Tuberculoza diseminată	Бугорчатка разсеянная — Bugortchatka rasseiannaia	22
Tuberculoza diseminată acuta Tuberculoza miliară	Бугорчатка разсеянная острая — Bugortchatka rasseiannaia ostraia	*a*
Tuberculoza diseminată cronică	Бугорчатка разсеянная хроническая — Bugortchatka rasseiannaia khronitcheskaia	*b*
	Бугорчатка милиарная — Bugortchatka miliarnaia	*c*
Lepră	Проказа — Prokaza	23

N°	Serbo-croate — Serbo-Croatian	Suédois — Swedish
19	Туберкулоза жљезда Tuberkuloza žljezda (Tbk. jlezda) Скрофулоза Skrofuloza	Skroffer
20	Туберкулоза мокраћних органа Tuberkuloza mokraćnih organa (Tbk. mokrachnih organa)	Tuberkulos i könsorganen Tuberkulos i urinvägarna
21		Tuberkulos i andra organen Annan tuberkulos
a		Tuberkulös Addisons sjukdom
b		
22		Allmän tuberkulos
a	Галопирајућа туберкулоза Galopirajuća tuberkuloza	Allmän tuberkulos
b		
c		
23	Шуга Šuga (Shuga) Лепра Lepra	Spetälska

Tchèque — Czech	Turc — Turkish	N°
Tuberkulosa soustavy mízní Tuberkulosa lymfatickeho systemu Tuberkulosa žlaz		19
Tuberkulosa ústrojí pohlavního a močového Tuberkulosa ústrojí urogenitalního Tuberkulosa ústrojí močového		20
Tuberkulosa jiných míst		21
		a
		b
Tuberkulosa roztroušená		22
Tuberkulosa miliární prudká		*a*
Tuberkulosa miliární vleklá		*b*
Tuberkulosa miliární		*c*
Malomocenství	Cüzam	23

N°	Nomenclature internationale International Nomenclature	Latin
24	**Infection purulente et septicémie** (sans rapport avec la grossesse, l'accouchement ou l'état puerpéral)	Infectio purulenta et septicaemia non puerperalis
a	Septicémie	Septichaemia Septicaemia Pyoseptichaemia
b	Pyémie ou pyohémie	Pyaemia
c	Gangrène gazeuse	Gangraena aerogenes Gangraena emphysematosa
d	Colibacillose généralisée	Colibacillosis disseminata
25	**Infection gonococcique** (toutes formes et localisations)	Morbi gonorrhoici
(a)	*Génitale*	Gonorrhea vulvae Metritis gonorrhoica Urethritis gonorrhoica Vaginitis gonorrhoica Colpitis gonorrhoica Endometritis gonorrhoica Epididymitis gonorrhoica Orchidis gonorrhoica
(b)	*Oculaire*	Neonatorum blennorrhoea gonorrhoica Ophthalmia neonatorum gonorrhoica
(c)	*Localisations autres et non spécifiées*	Arthritis gonorrhoica Cystitis gonorrhoica Endocarditis gonorrhoica
26	**Autres maladies bactériennes** (sauf dysenterie)	
a	Morve et farcin	Malleus humidus Malleus farciminosus Maliasmus
b	Tularémie	Tularaemia Tularemia
c	Autres	

Allemand — German	Anglais — English	N°
Sepsis	Purulent infection and septicæmia (non-puerperal)	**24**
Septikaemie Blutvergiftung (v)	Septicæmia* Blood poisoning (v) Sepsis* Bacteriæmia* Streptococcus infection	*a*
Pyaemie	Pycemia Staphylococcus infection	*b*
Gasoedem Gasbrand	Gas gangrene Malignant œdema (v)	*c*
Colisepsis	Colibacillosis Generalised infection by *B. Coli*	*d*
	Gonococcal infection	**25**
Gonorrhoe Tripper (v) Blennorrhoe	Blennorrhagia Blennorrhœa Gleet (v) Gonorrhœa Clap (v)	*(a)*
Gonorrhoische Augenentzündung der Neugeborenen Augentripper (v)	Purulent conjunctivitis of the newly-born Ophthalmia neonatorum	*(b)*
		(c)
		26
Rotz Malleus	Glanders	*a*
Tularämie	Tularæmia Tularemia	*b*
		c

N°	Bulgare — Bulgarian	Danois et norvégien Danish and Norwegian
24		
a		Septikæmi Blodforgiftning (v)
b		Pyæmi Gasbrand
c		
d		
25		
(a)		Gonorrĕ Uretritt u.o. Dryppert (v) Gonorroisk Orchitt
(b)		Gonorroisk oftalmi Spebarn-oftalmi Spædebarn Oftalmi
(c)		Gonorreisk Ledebetændelse Gonorroisk Leddbetendelse Gonorroisk Arthritis
26		
a	Сапъ Sap	Snive
b		Tularæmi
c		

Espagnol — Spanish	Estonien — Estonian	N°
Infeccion purulenta y septicemia no puerperal		**24**
Infeccion por estreptococos Infeccion generalizada Septicemia Infeccion septica	Roiskveresus	*a*
Piemia Piohemia Infeccion por estafilococos	Mädaveresus	*b*
Gangrena gaseosa Infeccion putrida	Gaasgangreen	*c*
Colibacilosis generalizada		*d*
		25
Blenorragia Gota militar (v) Purgación (v) Vaginite s.o.i.	Gonorröa Tripper	*(a)*
Oftalmía blenorragica Oftalmía gonocócica		*(b)*
Gonococia		*(c)*
		26
Muermo Farcin Lamparones	Tatitõbi	*a*
Tularemia	Tularæmia	*b*
		c

Nº	Finnois — Finnish	Français — French
24		Infection purulente
a	Verenmyrkytys	Septicémie, Bactériémie Empoisonnement du sang (v) Infection généralisée* Infection septique* Infection streptococcique
b		Pyémie Résorption purulente (v) Infection staphylococcique
c		Gangrène gazeuse Œdème malin (v) Œdème gazeux (v)
d		Septicémie colibacillaire Colibacillose généralisée
25		
(a)	Tippuri	Uréthrite gonococcique Gonococcie Blennorragie Blennorrée Chaudepisse (v) Goutte militaire (v) Vaginite s.a.i.
(b)		Conjonctivité purulente des nouveau-nés Ophtalmie des nouveau-nés
(c)		Arthrite gonococcique
26		
a		Morve Farcin Infection à *B. Mallei*
b		Tularémie Maladie de Francis Fièvre des lièvres
c		

Grec — Greek	Hongrois — Hungarian	Islandais — Icelandic	N°
Λοιμωξις πυώδης Loimoxis pyodes	Genyes fertőzes és vérmérgezés (nem gyermekagyi)		**24**
Σηψαιμια Sepsaimia	Vermérgezés Sepsis		*a*
	Genyesvérűség		*b*
			c
			d
			25
Γονοκοκκικαι Gonokokkikai	Gonorrhœa Kankó		*(a)*
			(b)
	Fertőző hugycsőlob kovetk		*(c)*
			26
Μἀλις Malis	Takonykór		*a*
			b
			c

N°	Italien — Italian	Letton — Latvian
24	Infezione purulenta e setticemia non puerperale	
a	Sepsi generalizatta Infezione settica Setticemia s.a.i. Infezioni settiche Streptococcemia	
b	Piemia Stafilococcemia Pioemia	
c	Cangrena gassosa	
d	Febbre da bacillo coli Colibacillosi	
25		
(a)	Blenorragia Goccetta militare (v) Uretrite blennorragica Uretrite gonococcica Vaginite s.a.i.	Tripers Gonoreja
(b)	Congiuntivite gonococcica dei neonati Oftalmia blennoragica	
(c)	Infezione gonococcica	
26		
a	Morva Farcino Cimurro Moccio (v)	Ļaunie ienāsi
b	Tularemia	
c		

Lithuanien — Lithuanian	Néerlandais — Dutch	N°
Kraujo užkretimas	Sepsis behalve puerperale	24
Septicemija	Septichaemie Bloedvergiftiging (v)	*a*
	Pyaemie	*b*
	Gasgangreen	*c*
	Colibacillose [gedissemineerde —]	*d*
		25
Triperís	Gonococcie Gonorrhoe Druiper (v)	*(a)*
	Gonorrhoische oogontsteking van de zuigelingen	*(b)*
	Gonorrhoische gewrichtsontsteking	*c*
		26
Iplautimas	Kwade droes Huidworm Malleus humidus Malleus farciminosus	*a*
	Tularaemie	*b*
		c

N°	Polonais — Polish	Portugais — Portuguese
24	Zakażenie ropne i posocznicze (nieporodowe)	Infecção purulenta (não puerperal)
a	Posocznica	Septicemia Infecção septica
b	Ropnica Pyemia	Infecção staphylococica
c	Zgorzel gazowa	Gangrena gazosa
d		Colibacilose generalisada
25		
(a)	Rzeżączka Wiewiór Tryper	Gonorrhea Esquentamento (v)
(b)		Ophtalmia blenorrhagica dos recemnatos
(c)		Gonococcia Infecção gonococica
26		
a	Nosacizna	Mormo Lamparão
b	Tularemia	Tularemia
c		

Roumain — Roumanian	Russe — Russian	N°
Infecţie purulentă şi septicemică (nepuerperală)	Гнойное заражение — Gnoinoe zarajenie	**24**
Septicemie	Септицемия — Septicemia Стрептококкцемия — Streptokokkcemia	*a*
Piemie Piohemie	Пиемия — Piemia	*b*
Gangrenă gazoasă	Газовая гангрена — Gazovaïa gangrena	*c*
Colibaciloză generalizată	Колибациллез — Colibacillez	*d*
		25
Blenoragie Gonococie	Бленорея — Blenoreïa Триппер — Tripper (v) Сляветечение — Sliazetetchenie (v)	*(a)*
Oftalmia noilor-născuti	Бленорройное (гнойное) воспаление глаза новорожденных — Blenoroïnoe (gnoïnoe) vospalenie glaza novorojdennykh	*(b)*
Infecţie gonococică Gonococie	Гонококкция — Gonokokkcia	*(c)*
Alte boli bacteriene (fara dizenterie)		**26**
Morvă Rapciugă	Сап — Sap Чилчак — Tchiltchak	*a*
Tularemie	Туларемия — Tularemia	*b*
		c

N°	Serbo-croate — Serbo-Croatian	Suédois — Swedish
24		
a	Септично заражење Septično zaraženje Тровање крви Trovanje krvi (Trovanié khrvi)	Varfeber Blodförgiftning
b	Гнојно заражење Gnojno zaraženje	
c	Гасна гангрена Gasna gangrena	Gasbrand
d		
25		
(a)	Гонореја Gonoreja Трипер Triper Капавац Kapavac	Gonorré Dröppel
(b)	Гнојно запаљење очију новорођених Gnojno zapaljenje očiju novorodjenih	
(c)		
26		
a	Сакагија Sakagija Малеус Maleus	Rots
b		Tularemi
c		

Tchèque — Czech	Turc — Turkish	N°
Hnísavá nákaza a otrava krve		**24**
Otrava krve	Septisemi	*a*
Rozklad krve		*b*
Snět plynová		*c*
		d
		25
Kapavka	Bel soğukluğu	*(a)*
Zánět očí novorozenců		*(b)*
		(c)
		26
Ozhřivka	Ruam	*a*
		b
		c

N°	Nomenclature internationale International Nomenclature	Latin
	DYSENTERIES	
27	**Dysenteries**	Dysenteria
a	Bacillaire	Dysenteria bacillaris
b	Amibienne	Dysenteria epidemica
c	Par d'autres protozoaires	Dysenteria amaebica
d	Non spécifiée ou due à d'autres causes	Dysenteria n.s.
	MALADIES DUES A DES PROTOZOAIRES	
28	**Paludisme** (malaria)	Malaria
a	Tierce bénigne	Malaria [Febris] terciana benigna
b	Quarte	Malaria [Febris] quartana
c	Tierce maligne (tropicale) et	Malaria tropica Malaria [Febris] quotidiana

Allemand — German	Anglais — English	N°
Ruhr	Dysentery	**27**
Bazilläre Ruhr	Bacillary dysentery	*a*
Amoebenruhr	Amoebic dysentery	*b*
	Giardiasis Flagellate diarrhea Lambliasis	*c*
Ruhr Dysenterie	Catarrhal dysentery Tropical dysentery	*d*
Malaria	Malaria	**28**
Dreitagefieber Vivax Malaria	Benign tertian fever	*a*
Viertagefieber	Quartan fever	*b*
Falciparum Malaria Malaria tropica Quotidiana Tägliches Wechselfieber	Quotidian fever* Tropical malignant tertian fever Æstivo-autumnal fever Subtertian malaria	*c*

N°	Bulgare — Bulgarian	Danois et norvégien Danish and Norwegian
27	Дизентерия Disenteria	Dysenteri
a		Paradysenteria Bacillær Disenteri Dysenteria bacillaris Basillœrdysenteri
b		Amöbedysenteri
c		
d		
28	Малария Malaria	Malaria
a		
b		
c		

Espagnol — Spanish	Estonien — Estonian	N°
Disenterías	Verine kõhutõbi Kõhutõbi Düsenteeria	**27**
Disentería bacilar Disentería epidemica Diarrea disenteriforme Disentería coleriforme	Batsillaarne düsenteeria	*a*
Disentería amebiana Disentería amibiana Disentería crónica	Amööbne düsenteeria	*b*
		c
Disentería s.o.i. Disentería de los paises cálidos Disentería tropical	Düsenteeria eristlemata või teistest põhjustest tingitud	*d*
Paludismo	Soopalavik Malaaria	**28**
Fiebre terciana Terciana benigna		*a*
Fiebre cuartana		*b*
Terciana precoz Fiebre sub-terciana Terciana espuria Fiebre perniciosa Paludismo maligno Fiebre estío-otoñale Fiebre cotidiana Fiebre maligna		*c*

N°	Finnois — Finnish	Français — French
27	Punatauti	Dysenterie
a		Dysenterie bacillaire Dysenterie à bacilles de Flexner, Hiss, Shiga, Sonne, V, W, X, Y, Z
b		Dysenterie amibienne Amibiase s.a.i.
c		Balantidiose intestinale Giardose Lambliase Diarrhée à flagellés
d		Diarrhée dysentériforme Dysenterie épidémique Dysenterie tropicale Dysenterie chronique
28	Vuorokuume Vilutauti	Paludisme Malaria
a		Tierce bénigne, Infection à *Plasmodium vivax*
b		Fièvre quarte, Infection à *P. malariae*
c		Tierce maligne, Infection à *P. falciparum* Fièvre estivo-automnale

Grec — Greek	Hongrois — Hungarian	Islandais — Icelandic	N°
Δυσεντερια Dysenteria	Vérhas Dysenteria	Blódsótt	27
	Bacillusos vérhas		*a*
	Amœbás vérhas Entamœbiasis		*b*
			c
	Külön meg nem nevezett, vagy más okból szarmazó		*d*
Ἑλονοσια Elonosia	Malária Mocsárláz Váltóláz		28
			a
			b
			c

Nº	Italien — Italian	Letton — Latvian
27	Dissenteria	Dizenterija
a	Dissenteria bacillare Dissenteria coleriforme	
b	Dissenteria amebica	
c	Flagellosi Lambliasi Giardasi intestinale	
d	Diarrea dissenteriforme Dissenteria s.a.i. Dissenteria dei paesi caldi	
28	Malaria	Malarija
a	Febbre terzana Terzana benigna	
b	Quartana Febbre quartana Infezione da *P. malariae*	
c	Malaria tropicale Terzana maligna Infezione da *P. falciparum*	

Lithuanien — Lithuanian	Néerlandais — Dutch	N°
Kruvinoji	Dysenterie	**27**
	Bacillaire dysenterie	*a*
	Amoeben-dysenterie Amoebiasis	*b*
		c
		d
Drugys Malarija	Malaria Moeraskoorts	**28**
	Tertiana	*a*
	Quartana Derdedaagsche koorts	*b*
	Tropica Pernicieuze malaria	*c*

Nº	Polonais — Polish	Portugais — Portuguese
27	Czerwonka Dysenteria	Dysenteria Disenteria
a	Dysenteria bakteryjna	Dysenteria bacillar Disenteria epidemica Disenteria choleriforme
b	Dysenteria amebowa	Dysenteria amebica Disenteria amebiana
c		
d		Disenteria tropicál
28	Malaria Zimnica	Malaria Paludismo
a	Malaria trzeciaczka	Febre terçã Terçã benigna
b	Malaria czwartaczka	Febre quartã Quartã
c	Malaria tropikalna	Febre perniciosa Febre quotidiana Terçã maligna Terçã tropical

Roumain — Roumanian	Russe — Russian	N°
Dizenterie	Дизентерия — Dizenteria	**27**
Dizenterie bacilară Dizenterie microbiană	Бациллярная дизентерия — Bazilliarnaïa dizenteria	*a*
Dizenterie amoebiană	Амебная дизентерия — Amebnaïa dizenteria	*b*
		c
		d
Malarie	Малярия — Malaria	**28**
Terța benignă	Трехдневная малярия — Trekhdnevnaïa malaria	*a*
Quartă	Четырехдневная малярия — Tchetyrekhdnevnaïa malaria	*b*
Terța malignă Terță tropicală	Ежедневная малярия — Ejednevnaïa malaria Злокачественная малярия — Zlokatchestvennaïa malaria Тропическая малярия — Tropitcheskaïa malaria	*c*

N°	Serbo-croate — Serbo-Croatian	Suédois — Swedish
27	Срдобоља Srdobolja Грижа Griža Дизентерија Dizenterija	Rödsot
a	Бациларна дизентерија Bacilarna dizenterija	Epidemisk rödsot
b	Тропска дизентерија Tropska dizenterija	
c		
d		
28	Маларија Malarija Барска грозница Barska groznica (v) (Barska groznitsa)	Malaria
a	Тродневна маларија Trodnevna malarija	
b	Четверодневна маларија Četverodnevna malarija (Chetverodnevna malarija)	
c	Тропска маларија Tropska malarija	

Tchèque — Czech	Turc — Turkish	N°
Červenka Úplavice Úplavice střevní	Dizanteri	27
Úplavice střevní bacilarní		*a*
Úplavice střevní amoebová		*b*
		c
Úplavice střevní nerozlišená nebo z jiné příčiny		*d*
Zimnice malarická Malaria	Malarya Sitma	28
		a
		b
		c

N°	Nomenclature internationale International Nomenclature	Latin
28c(b)	Fièvre bilieuse hémoglobinurique	Febris biliosa hemoglobinurica
d	Forme autre ou non spécifiée	Malaria Febris malariae Febris malarica Febris intermittens Cachexia malariae
29	**Autres maladies dues à des protozoaires parasites** (sauf spirochètes)	
a	Coccidiose	Coccidiosis
b	Leishmaniose	Leishmaniosis
b(a)	*Leishmaniose viscérale*	Leishmaniosis visceralis Kala azar
b(b)	*Leishmaniose cutanée méditerranéenne*	Helkosoma tropicum Leishmaniosis cutanea Leishmaniosis tropica
b(c)	*Leishmaniose cutanée américaine*	Leishmaniosis cutis americana
c	Ulcère phagédénique	Ulcus phagedaenicum
d	Trypanosomiase américaine	Trypanosomiasis americana
e	Trypanosomiase africaine	Trypanosomiasis africana
f	Bartonellose	Bartonellosis Verruca peruviana

Allemand — German	Anglais — English	N°
Schwarzwasserfieber	Blackwater fever Hæmoglobinuric bilious fever	28c(b)
Wechselfieber (v) Febris intermittens *	Marsh fever (v) Ague (v) Intermittent fever (v) Remittent fever (v)	d
		29
Coccidiosis Coccidien	Coccidiosis	a
Leishmaniosen	Leishmaniasis	b
Kala-azar	Kala-azar	b(a)
Orientbeule Helkosoma tropicum	Oriental sore	b(b)
	American dermal leishmaniasis	b(c)
Phagedänische Geschwüre	Phagedenic ulcer	c
Chagassche Krankheit	Chagas' disease	d
Schlafkrankheit	Sleeping-sickness Trypanosomiasis	e
Verruga peruviana	Bartonellosis Carrion's disease Oroya fever	f

N°	Bulgare — Bulgarian	Danois et norvégien Danish and Norwegian
28c(b)		
d	Малария Malaria	Koldfeber Malaria Febris intermittens
29		
a		
b		
b(a)		
b(b)		
b(c)		
c		
d		
e		Sovesyke Sovesyge
f		

Espagnol — Spanish	Estonien — Estonian	N°
Fiebre biliosa hemoglobinurica Fiebre hemoglobinurica		28c(b)
Malaria Impaludismo Anemia palustre Fiebre intermitente (v) Calentura intermitente (v) Fiebre maregmatica* Fiebre palustre Caquexia paludica	Malaaria Malaaria palavik Kaheksia malaariast	*d*
		29
Coccidiosis	Coccidioses	*a*
Leishmaniosis	Leishmanioses	*b*
Kala-azar Leishmaniosis esplénica infantil Leishmaniosis visceral		*b(a)*
Boton (o Clavo) de Alepo, de Oriente Leishmania furunculosa Leishmaniosis cutanea (mediterranea)	Orientaal muhk	*b(b)*
Bubas brasilianas		*b(c)*
Ulcera fagedenica Absceso fagedenico de los paisos calidos	Soojail mail Soemail	*c*
Simbiosis fuso-espirilar Enfermedad de Chagas Tripanosomiasis americana		*d*
Enfermedad del sueño Tripanosomiasis africana	Aafrika unehaigus	*e*
Verruga peruana Enfermedad de Carrion Fiebre de Oroya Fiebre verrucosa del Guaitara Bartonellosis		*f*

N°	Finnois — Finnish	Français — French
28*c*(*b*)		Fièvre bilieuse hématurique
d	Vuorokuume Vilutauti	Accès pernicieux Cachexie palustre Fièvre paludéenne Fièvre pernicieuse* Fièvre intermittente* (v)
29		
a		Coccidiose
b		Leishmaniose Infection à *L. donovani*
b(*a*)		Kala-azar Leishmaniose splénique infantile
b(*b*)		Bouton d'Alep, de Biskra, d'Orient Leishmaniose cutanée Infection à *L. tropica*
b(*c*)		Leishmaniose forestière Leishmaniose brésilienne
c		Pourriture d'hôpital Ulcère phagédénique Ulcère fuso-spirillaire
d		Trypanosomose américaine Infection à *Schyzotrypanum cruzi*
e		Trypanosomiase africaine Trypanosomiase s.a.i. Maladie du sommeil
f		Fièvre d'Oroya Maladie de Carrion Bartonellose péruvienne Verruga peruviana

Grec — Greek	Hongrois — Hungarian	Islandais — Icelandic	N°
Χολώδης αιμοσφαιρινουρικὸς πυρετός Cholodes aimosphairinoyrikos pyretos	Heveny mocsárláz		28c(b)
	Mocsárlázas senyv		d
	Elősködő veglények által okozott egyeb betegségek		29
			a
			b
			b(a)
			b(b)
			b(c)
			c
			d
			e
			f

N°	Italien — Italian	Letton — Latvian
28*c(b)*	Febbre biliosa emoglobinurica Febbre ittero-emoglobinurica Febbre emoglobinurica	
d	Malaria s.a.i. Febbre malarica Febbre palustre Cachessia palustre	Purva drudzis
29		
a	Coccidiosi	
b	Leishmaniosi	
b(a)	Leishmaniosi interna Leishmaniosi splenica infantile	
b(b)	Kala-azar Bottone di oriente Bottone di Aleppo Chiodo di Biskra Leishmanide cutanea	
b(c)	Leishmaniosi brasiliana	
c	Ulcera fagedenica	
d	Malattia di Chagas Schizotripanosomiasis Tripanosi del Brasile	
e	Malattia del sonno Tripanosomiasi africana Castellanosi Malattia del Castellani	
f	Verruca peruviana Febbre di Oroja Morbo di Carrion	

Lithuanien — Lithuanian	Néerlandais — Dutch	N°
	Zwartwaterkoorts	28c(b)
Drugys Malarija	Moeraskoorts Malaria cachexie	d
		29
		a
		b
		b(a)
		b(b)
		b(c)
		c
	Ziekte van Chagas	d
	Afrikaansche slaapziekte	e
		f

Nº	Polonais — Polish	Portugais — Portuguese
28c(b)		Febre biliosa hemoglobinurica Pirexia biliosa hemoglobinurica
d	Malaria Zimnica	Paludismo Malaria Febre palustre Febre* (v) Cachexia palustre Febre intermitente Sezonismo Febre remitente vulgar (v) Impaludismo
29		
a		Coccidiose
b		Leishmaniosa
b(a)		Leishmaniosa viscerosa
b(b)		Leishmaniosa cutanea
b(c)		Leishmaniosa brasileira Bóubas brasileiras Bubas Ulcera de Bauru
c		Ulcera phagedenica
d		Doença de Chagas
e		Doença do sono
f		Verruga peruana Bartonellose

Roumain — Roumanian	Russe — Russian	N°
Febra bilioasa hemoglobinurică	Билиозная лихорадка — Bilioznaïa likhoradka	28c(b)
Paludism Friguri palustre (v) Friguri de balta (v) Caşexie paludică	Малярия — Malaria	d
Alta boli datorite protozoarelor paraziţi (fara spirocheţi)		29
Coccidioză	Коккидии — Kokktsidii	a
Leishmanioză		b
Leishmanioză visceralâ		b(a)
Leishmanioză cutanee mediterraneană		b(b)
Leishmanioză cutanee americană		b(c)
Ulcer fagedenic		c
Tripanosomiaza americană	Трипанозомиазис американский — Tripanozomiazis amerikanskiï	d
Tripanosomiaza africană Boala somnului	Трипанозомиазис африканский — Tripanozomiazis afrikanskiï Сонная болезнь — Sonnaïa bolezn	e
Bartoneloză		f

N°	Serbo-croate — Serbo-Croatian	Suédois — Swedish
28c(b)		
d	Маларија Malarija	Frossa
29		
a		
b		
b(a)	Кала-азар Kala-azar	
b(b)		
b(c)		
c		
d		
e	Болест спавања Bolest spavanja	
f		

Tchèque — Czech	Turc — Turkish	N°
		28c(b)
Kachexie malarická Malaria	Malarya Sitma	*d*
Jiné nemoci zaviněné cizopasnými prvoky		**29**
		a
		b
	Haşevi leyşmanyoz	*b(a)*
	Halep cibani	*b(b)*
		b(c)
		c
		d
		e
		f

N°	Nomenclature internationale International Nomenclature	Latin
	MALADIES DUES A DES SPIROCHÈTES	
30	**Syphilis**	Syphilis Lues
a	Ataxie locomotrice (tabes dorsalis)	Tabes dorsalis Ataxia motoria progressiva
b	Paralysie générale	Paralysis generalis progressiva alienorum Lues cerebri Dementia paralytica
c	Anévrisme de l'aorte	Aneurysma aortae Aortitis syphilitica Roseola syphilitica
d	Autres formes de syphilis (=) dont :	Syphilis primaria Syphilis secundaria Syphilis tertiaria Ulcus durum Gumma syphiliticum
d(a)	*Congénitale*	Syphilis congenita Syphilis hereditaria Pemphigus syphiliticus Lues infantum
31	**Fièvres récurrentes**	Febris recurrens
a	Fièvre récurrente épidémique à poux *(Sp. obermeieri)*	Typhus recurrens Typhus recurrens Obermeieri Spirochetosis Obermeieri
b	Fièvres récurrentes transmises par d'autres vecteurs : Fièvre récurrente à tiques	Spirochetosis Duttoni Febris recurrens Duttoni
c	Fièvre récurrente non spécifiée	Febris recurrens

Allemand — German	Anglais — English	N°
Syphilis	Syphilis	**30**
Tabes dorsalis Rückenmarkschwindsucht	Progressive locomotor ataxia Progressive ataxy Tabes dorsalis	*a*
(Fortschreitende) Gehirnerweichung Progressive Paralyse	General paralysis of the insane General paresis Dementia paralytica	*b*
Schlagadererweiterung Aortenaneurysma Hauptschlagaderentzündung Aortitis syphilitica	Aneurysm of the aorta	*c*
Lues Lustseuche (v)	Gumma Rupia Hard chancre Lues venerea Luetic disease Primary, secondary, tertiary syphilis*	*d*
Erb Syphilis Angeborene Syphilis	Congenital syphilis Congenital disease (v) Snuffles (v) Lues infantum Hereditary syphilis	*d(a)*
Rückfallfieber	Relapsing fever Spirillosis	**31**
Typhus recurrens	Epidemic (louse-borne) relapsing fever	*a*
Febris recurrens	Tick-fever *(Sp. duttoni)* Central African tick-fever	*b*
		c

N°	Bulgare — Bulgarian	Danois et norvégien Danish and Norwegian
30	Сифилисъ Sifilis	Syfilis
a		Rygmarvstæring Ryggmargstœring
b		Generel Paralyse Paralysis generalis
c		Aortaaneurysme
d		Erhvervet Syfilis Haard Chanker
d(a)		Medfödt Syfilis
31		
a	Възвратенъ тифъ Vezvraten tif	Tilbakefallsfeber
b		
c		

Espagnol — Spanish	Estonien — Estonian	N°
Sifilis	Süüfilis Luues	**30**
Ataxia locomotriz progresiva Tabes dorsal Enfermedad de Duchenne	Seljataabes	*a*
Paralisis general Marasmo paralítico Demencia paralítica	Üldine paralüüs Halvatus	*b*
Aneurisma aortica Ectasia aortica		*c*
Accidentes primitivos, secundarios, terciarios Chancro indurado, infectante, sifilítico Vérole (v) Gomas Bubón infectante	Omandatud süüfilis Lihtsad juhud sekundaar-nähtused Tertsiaarsed nähtused	*d*
Sifilis congénita	Kaasasündinud süüfilis	*d(a)*
Fiebre recurrente		**31**
Fiebre recurrente de Obermeier Tifus recurrente	Korduv palavik Taastuv soetõbi	*a*
Fiebre recurrente africana Enfermedad de Dutton Fiebre recurrente de garrapatas		*b*
Fiebre recurrente hispanoafricana		*c*

N°	Finnois — Finnish	Français — French
30	Kuppa Kuppatauti	Syphilis Vérole (v) Spécificité*
a		Ataxie locomotrice progressive Tabès
b		Paralysie générale Démence paralytique Cachexie paralytique
c		Anévrisme aortique
d	Veres kuppa Piileva kuppa	Chancre infectant Chancre induré Gommes Accidents primaires, secondaires, tertiaires
d(a)	Synnynnäinen kuppatauti	Hérédo-syphilis Syphilis congénitale
31		
a	Toisintakuume	Typhus récurrent Fièvre récurrente à poux
b		Fièvre récurrente espagnole Fièvre récurrente à tiques Fièvre récurrente à *Ornithodorus*
c		Spirochétose africaine

Grec — Greek	Hongrois — Hungarian	Islandais — Icelandic	N°
Σύφιλις Syphilis	Bujakór Szifilisz Verbaj		**30**
Αταξια κινητική προϊοῦσα Ataksia kinetike proiousa	Hátgerincsorvadás		*a*
Παραλυσια γενική Paralysia genike	Benulásos elmezavar Elmebajosok bénulása		*b*
Ανεύρυσμα Aneurysma	Ertágulás Verőértágulás		*c*
	Szerzett szifilisz		*d*
	Veleszületett szifilisz		*d(a)*
Ὑποστροφοι πυρετοι Upostrophoi pyretoi			**31**
	Visszatérő laz		*a*
			b
			c

N°	Italien — Italian	Letton — Latvian
30	Sifilide Lue Peste (vv)	Sifiliss
a	Atassia locomotrice progressiva Tabe dorsale	Mugurkaula smadzenu degenerācija
b	Paralisi progressiva (degli alienati) Demenza paralitica	Vispārējā paralīze
c	Aneurisma delle aorta	
d	Ulcera infettante Bubbone infettante Manifestazioni primitive, secondarie, terziarie Manifestazioni specifiche Gomme Ulcera dura	
d(a)	Sifilide congenita	Sifiliss iedzimts
31	Febbre ricorrente	
a	Tifo ricorrente Febbre ricorrente da pidocchio Febbre ricorrente europea	Atguļas tīfs
b	Febbre ricorrente da zecche Febbre ricorrente spagnuola	
c		

Lithuanien — Lithuanian	Néerlandais — Dutch	N°
Syfilis	Syphilis	**30**
Tabes dorsalis	Progressive locomotorische ataxie Rugemergstering	*a*
Paralysis progressiva	Lues cerebri Dementia paralytica Taboparalyse Progressive paralyse	*b*
	Aneurysma van het aorta	*c*
	Verkregen syphilis Harde schanker	*d*
	Aangeboren syphilis	*d(a)*
		31
Atokrytiné šiltiné		*a*
		b
	Recurreerende koorts Recurrens spirochaetose	*c*

Nº	Polonais — Polish	Portugais — Portuguese
30	Syfilis Lues Kiła	Syphilis
a	Tabes Wiąd rdzenia	Ataxia locomotriz progressiva Tabes dorsal Ataxia locomotora progressiva
b	Paraliż postępowy	Paralysie geral Paralisia geral
c	Tętniak aorty	Aneurysma da aorta
d		Gallico (v)
d(a)	Kiła wrodzona	Syphilis congenita
31		
a	Tyfus powrotny Dur powrotny	Febre recurrente epidemica Typho recurrente
b		Febre recurrente de Dutton Febre recurrente dos carrapatos
c		

Roumain — Roumanian	Russe — Russian	N°
Sifilis	Сифилис — Sifilis	**30**
Ataxie locomotrice Tabes dorsalis	Сухотка спинного мозга — Sukhotka spinnogo mozga Болезнь Дюшенна — Bolezn duchenna	*a*
Paralizie generală	Общий прогрессивный паралич — Obstchiï progressivnyï paralitch Прогрессивный паралич помешанных — Progressivnyï paralitch pomechannykh	*b*
Anevrism al aortei	Аневризма аорты — Anevrizma aorty	*c*
		d
Sifilis congenital	Наследственный врожденный сифилис — Nasledstvennyï vrojdennyï sifilis	*d(a)*
Febre recurente	Возвратныя лихорадки — Vozvratnya likhoradki	**31**
Febră recurentă epidemica cu păduchi Tifus recurent	Эпидемический возвратный тиф — Epidemitcheskiï vozvratnyï tif Возвратный тиф (вшивый) — Vozvratnyï tif (vchivyï)	*a*
Febră recurentă cu căpuşe	Клещевый возвратный тиф — Klestchiovyï vozvratnyï tif	*b*
	Возвратный тиф б. у. — Vozvratnyï tif b. u.	*c*

N°	Serbo-croate — Serbo-Croatian	Suédois — Swedish
30	Сифилис Sifilis	Syfilis
a	Сушење кичме Sušenje kičme (Sushenje kichme)	Ryggmärgstvinsot
b	Општа парализа Opšta paraliza (opshta paraliza)	Allmän paralysi
c		Aortabråck
d		Förvärvad syfilis
d(a)	Наследни сифилис Nasledni sifilis	Ärvd syfilis
31		Återsfallsfeber
a	Рекуренс Rekurens Тифус рекуренс Tifus recurens	Återsfallsfeber
b		
c		

Tchèque — Czech	Turc — Turkish	N°
Příjice	Frengi	**30**
Úbyt míchy		*a*
Postupná obrna povšechná		*b*
Výdut tepen Aneurysma aorty Výdut aorty		*c*
Příjice získaná		*d*
Příjice vrozená		*d(a)*
		31
Týf zvratný Týf vratný Vrativka	Hummayi racia	*a*
		b
		c

N°	Nomenclature internationale International Nomenclature	Latin
32	**Autres maladies dues à des spirochètes**	
a	Spirochétose ictéro-hémorragique	Icterus haemorrhagicus (spirochaetosus) Icterus infectiosus Morbus Weilii Spirochaetosis ictero-hemorrhagica
b	Autres	
b(a)	*Angine de Vincent*	Angina Vincenti
b(b)	*Sodoku*	Febris morsusmuris
b(c)	*Pian*	Framboesia tropica
b(d)	*Fièvre des marais* (infection à Leptospira grippo-typhosa)	Leptospirosis grippo-typhosa
	MALADIES DUES OU ATTRIBUÉES A DES VIRUS FILTRANTS	
33	**Grippe** (influenza)	Influenza
a	Avec complications respiratoires mentionnées	Bronchitis gripposa Bronchopneumonia gripposa Pneumonia influenzae Broncho-pneumonia influenzae
b	Sans complications respiratoires mentionnées	Encephalitis influenzae

Allemand — German	Anglais — English	N°
		32
Weil'sche Krankheit Übertragbare Gelbsucht Icterus infectiosus	Spirochætosis ictero-hemorragica Weil's disease Leptospirosis ictero-hemorragica Spirochætal jaundice	*a*
		b
Angina Plaut-Vincenti Geschwürige Mandelentzündung nach Plaut-Vincent Plaut-Vincentsche Angina	Vincent's angina Trench mouth Spirochætal stomatitis	*b(a)*
Sodoku Japanische Rattenbisskrankheit	Rat-bite fever Sodoku	*b(b)*
Framboesie Framboesia	Yaws Framboesia Goundou	*b(c)*
Feldfieber Schlammfieber Erntefieber Sumpffieber Infektion durch *Leptospira grippo-typhosa*	Mud-fever Mud field fever	*b(d)*
Influenza Grippe	Influenza Grippe Flu (v)	33
Influenzische Pneumonie Influenzische Bronchopneumonie Influenzische Lungenentzündung	Influenzal pneumonia Influenzal broncho-pneumonia	*a*
Influenzische Encephalitis Influenzische Gehirnentzündung	Epidemic catarrh (v) Febrile catarrh (v)	*b*

N°	Bulgare — Bulgarian	Danois et norvégien Danish and Norwegian
32		
a		Weil Syge Weilsyke Weils sykdom
b		
b(a)		Vincent angina
b(b)		Rottebidsygdom
b(c)		
b(d)		
33	Инфлуенца Influentsa	Influensa Influenza
a		Influensa med Lungekomplikationer Influensa Pneumonie Influensa Bronkopneumonie
b		Influensa uden Lungekomplikationer Influensa Encephalitt

Espagnol — Spanish	Estonien — Estonian	N°
		32
Espiroquetosis icterohemorrágica Enfermedad de Mathieu-Weil Enfermedad de Weil Ictericia epidemica Ictericia de recaidas	Spirochætoses muud Morbus Weilii	*a*
		b
Angina de Vincent Angina ulceromembranosa fusoespirilar	Angina Vincenti	*b(a)*
Sodoku Espiroquetosis por mordedura de rata		*b(b)*
Pian Frambesia Polypapilloma tropicum Framboesia tropica		*b(c)*
		b(d)
Grippe Influenza	Influentsa Gripp	**33**
Pneumonia gripal Bronco-pneumonia gripal		*a*
Encefalitis gripal		*b*

N°	Finnois — Finnish	Français — French
32		
a		Ictère épidémique Maladie de Weil Leptospirose ictéro-hémorragique
b		
b(a)		Angine fuso-spirillaire de Vincent
b(b)		Sodoku Fièvre par morsure de rat
b(c)		Pian
b(d)		Fièvre des marais Leptospirose à *L. grippotyphosa*
33	Influensa	Grippe Influenza
a		Pneumonie grippale Broncho-pneumonie grippale
b		Encéphalite grippale

Grec — Greek	Hongrois — Hungarian	Islandais — Icelandic	N°
			32
			a
			b
			b(a)
			b(b)
			b(c)
			b(d)
Γρίππη Grippe	Influenza Nátha	Influensa	**33**
	Megmelitett legzőszervi szövődményekkel		*a*
	Azok nélkul		*b*

N°	Italien — Italian	Letton — Latvian
32		
a	Leptospirosi Spirochetosi ittero-emorragica Morbo di Weil Ittero infettivo Ittero castrense grave	
b		
b(a)	Angina fuso-spirillare di Vincent	
b(b)	Sodoka Sodoku Febbre da morso di topo	
b(c)	Pian Frambœsia tropicale Treponemosi di Castellani	
b(d)		
33	Influenza Influenza epidemica	Gripa Grippa Influenca Influenza
a	Polmonite influenzale Bronco-polmonite grippale	
b	Encefalite influenzale Grippe s.a.i.	

Lithuanien — Lithuanian	Néerlandais — Dutch	N°
		32
	Ziekte vạn Weil Leptospirosis	*a*
	Bronchospirochætosis	*b*
	Plaut-Vincentsche angina	*b(a)*
	Rattebeetspirillosis Rattebeetziekte	*b(b)*
	Framboesia tropica Pian Yaws Paerae (Sumatra) Pateh (Java)	*b(c)* *b(d)*
Gripas Influenza	Influenza Griep	**33**
	Griepale broncho-pneumonie Griepale pneumonie	*a*
	Griepale hersenontsteking	*b*

N°	Polonais — Polish	Portugais — Portuguese
32		
a	Choroba Weila	Espiroquetose de Weil Espirochetose icterohemorrhagica Doença de Weil
b		
b(a)	Angina Vincenta	Angina de Vincent
b(b)		Sodoku
b(c)		Framboezia tropicál
b(d)		
33	Grypa Influenza	Grippe Influenza Gripe
a	Z komplikacjami płucnymi	
b	Bez komplikacji płucnych	

Roumain — Roumanian	Russe — Russian	N°
Alte boli datorite spirocheților		**32**
Spirochetoza ictero-hemoragică Boala lui Weil	Желчно-геморрагический спирокетоз — Jeltchno-gemorragitcheskiĭ spiroketoz Болезнь Вейля — Bolezn Weila Епидемическая желтуха — Epidemitcheskaïa jeltoukha	*a*
		b
Angina lui Vincent		*b(a)*
Sodoku	Содоку — Sodoku	*b(b)*
Pian	Пиана — Piana	*b(c)*
Leptospiroza gripo-tifică	Болотная лихорадка гриппотифозная — Bolotnaïa likhoradka grippotifoznaïa	*b(d)*
Gripă Influență	Грипп — Gripp Инфлюэнца — Infliuenza	**33**
Bronho-pneumonie gripală Pneumonie gripală	С обозначением легочных осложнений — S oboznatcheniem legotchnykh oslojneniĭ Гриппозная пневмония — Grippoznaïa pnevmonia Гриппозная бронхопневмония — Grippoznaïa bronkhopnevmonia	*a*
Encefalită gripală	Без обозначения легочных осложнений — Bez oboznatchenia legotchnykh oslojneniĭ	*b*

N°	Serbo-croate — Serbo-Croatian	Suédois — Swedish
32		
a	Morbus Weyli	Weils sjukdom
b		
b(a)		
b(b)		
b(c)		
b(d)		
33	Грип Grip Грипа Gripa Инфлуенца Influenca	Influensa
a	Грипозно запаљење плућа Gripozno zapaljenje pluća (gripozno zapaljenje plucha)	
b		

Tchèque — Czech	Turc — Turkish	N°
		32
Spirochetosa iktero-hœmorrhagiæ Infekční žloutenka		*a*
		b
		b(a)
		b(b)
		b(c)
		b(d)
Chřipka Influenza	Grip	**33**
Chřipka s komplikacemi dýchadel Chřipkový zánět plic		*a*
Chřipka bez údaje takových komplikací		*b*

N°	Nomenclature internationale International Nomenclature	Latin
34	**Variole**	Variola
a	Variola major	Variola vera Variola major Purpura variolosa Variola hemorrhagica
b	Variola minor	Variola minor Variolois
c	Non spécifiée	Variola
35	**Rougeole**	Morbilli Rubeola
36	**Poliomyélite aiguë et polioencéphalite aiguë**	Poliomyelitis anterior acuta Polioencephalitis acuta Paralysis infantum
37	**Encéphalite infectieuse aiguë** (léthargique ou épidémique)	Encephalitis epidemica acuta
a	Encéphalite léthargique (ou épidémique) aiguë	Encephalitis lethargica acuta
b	Séquelles d'encéphalite léthargique	Sequelae encephalitis lethargicae Paralysis agitans encephalitica
c	Encéphalite léthargique (ou épidémique) sans autre spécification	Encephalitis lethargica n.s.
38	**Autres maladies dues ou attribuées à des virus filtrants**	
a	Fièvre jaune	Febris flava Febris biliosa
b	Rage	Lyssa humana Rabies Hydrophobia

Allemand — German	Anglais — English	N°
Pocken Blattern (v)	Smallpox	**34**
Echte Blattern Schwarze Blattern (v) Schwarze Pocken	Variola major Severe smallpox	*a*
Variolois Milde Form der Blattern	Variola minor Mild smallpox Alastrim Amaas (South Africa)	*b*
		c
Masern Morbilli	Rubeola* (cf. note in the index) Measles Morbilli	**35**
Spinale Kinderlähmung Rückenmarkslähmung der Kinder Rückenmarkskinderlähmung Heine-Medinsche Krankheit	Acute poliomyelitis Acute polioencephalitis Infantile paralysis	**36**
		37
Übertragbare Gehirnentzündung Epidemische Gehirnentzündung Gehirngrippe (v)	Acute infectious encephalitis Epidemic encephalitis Infective encephalitis	*a*
Parkinsonsche Krankheit (nach Gehirnentzündung)	Sequelæ of encephalitis Parkinsonism	*b*
Schlafsuchtkrankheit (v)	Lethargic encephalitis Sleepy sickness	*c*
		38
Gelbfieber	Yellow fever	*a*
Tollwut Rabies Hundswut (v) Wut Lyssa	Rabies Hydrophobia	*b*

N°	Bulgare — Bulgarian	Danois et norvégien Danish and Norwegian
34	Едра шарка Edra charka	Kopper
a	Едра шарка Edra charka	Kopper
b		
c		
35	Брусница Brusnitsa	Mæslinger Meslinger
36	Детски паралич Detski paralitch	Börnelammelse Akutt Poliomyelitt Akut Barnelammelse
37	Епидемиченъ енцефалитъ Epidemitchen entsephalit	
a		Akut Hjærnebetændelse Akutt epidemisk encephalitt
b		
c		Hjærnebetændelse Hjærnbetendelse Epidemisk Encephalitt Epidemisk Encephalitis
38		
a		Gulfeber
b	Буе Bue	Hundgalskab Hundgalskap

Espagnol — Spanish	Estonien — Estonian	N°
Viruela	Rõuged	**34**
Viruela mayor	Rõuged	*a*
Viruela menor Alastrim	Tuulerõuged	*b*
Varioloide		*c*
Sarampión Alfombrilla Rugeola Erupción morbilosa	Leetrid	**35**
Poliomielitis aguda Polioencefalitis aguda Paralisis esencial de la infancia Paralisis espinal aguda Enfermedad de Heine-Médin	Äge poliomüeliit Äge polioentsefaliit	**36**
		37
Encefalitis letargica aguda		*a*
		b
Encefalitis letargica Encefalitis epidemica	Letargiline entsefaliit Epideemiline entsefaliit Unehaigus	*c*
		38
Fiebre amarilla Vómito negro (v)	Kollane palavik	*a*
Rabia Hidrofobia Lyssa	Marutõbi	*b*

N°	Finnois — Finnish	Français — French
34	Isorokko	Variole
a	Isorokko	Petite vérole (v) Variole s.a.i. Variole grave Variole hémorragique
b		Varioloïde Alastrim
c		
35	Tuhkarokko	Rougeole Infection morbilleuse
36	Lapsihalvaus	Poliomyélite antérieure aiguë Paralysie infantile Maladie de Heine-Medin
37	Unitauti	Encéphalite épidémique
a		Encéphalite infectieuse aiguë
b		Parkinsonisme
c	Unitauti	Encéphalite léthargique s.a.i.
38		
a		Fièvre jaune Typhus amaril Vomito negro
b		Rage Hydrophobie*

Grec — Greek	Hongrois — Hungarian	Islandais — Icelandic	N°
Εὐλογία Eulogia	Himlő Holyagos himlő		34
Εὐλογία Eulogia	Himlő		*a*
			b
			c
Ιλαρὰ Ilara	Kanyaró	Mislingar	35
Πολυομυελιτις ὀξεῖα Poliomyelitis okseia Πολυοεγκεφαλιτις ὀξεῖα Polioenkephalitis okseia	Heine-Medin-kór Gyermekhüdes	Maenusott	36
Εγκεφαλιτις ληθαργικὴ Enkephalitis lethargikè	Jarvanyos agyvelő-gyulladás	Svefnsýki	37
Εγκεφαλιτις επιδημικὴ ὀξεῖα Enkephalitis epidemikè okseia			*a*
			b
Εγκεφαλιτις ληθαργικὴ Enkephalitis lethargike			*c*
			38
Κίτρινος πυρετος Kitrinos pyretos	Sárgaláz		*a*
Λύσσα Lyssa	Ebdüh Lyssa Veszettség		*b*

N°	Italien — Italian	Letton — Latvian
34	Vaiuolo	Bakas
a	Vaiolo	Bakas
b	Vaiuoloide Vaioloide Alastrim	
c		
35	Morbillo	Masalas
36	Malattia di Heine-Medin Poliomielite anteriore acuta Paralisi infantile (v) Polioencefalite acuta	Akūtais muguras smadzenu iekaisums Akūtais galvas smadzenu iekaisums Bērnu trieka Heine-Medin slimība
37		
a	Encefalite letargica acuta Encefalite epidemica acuta	Akūtais letargiskais smadzenu iekaisums
b		
c	Encefalite epidemica s.a.i. Encefalite letargica s.a.i.	Epidēmiskais smadzenu iekaisums Letargiskais smadzenu iekaisums
38		
a	Febbre gialla	
b	Rabbia Lissa Idrofobia (v)	Trakuma sērga Lyssa

Lithuanien — Lithuanian	Néerlandais — Dutch	N°
Raupai	Pokken	**34**
Raupai	Variola major Pokken Variola vera	*a*
	Variola minor Alastrim Variolois	*b*
		c
Tymai	Mazelen	**35**
Ūminis nugaros ir Galvos smegenų uždegimas	Poliomyelitis anterior acuta Kinderverlamming	**36**
		37
		a
		b
Letargiškas smegenų uždegimas	Encephalitis lethargica (Europeesche) Slaapziekte Slaapzucht	*c*
		38
	Gele koorts	*a*
Pasiutimas	Hondsdolheid Lyssa Rabies	*b*

N°	Polonais — Polish	Portugais — Portuguese
34	Ospa	Variola Bexigas (v)
a	Ospa	Variola major
b		Alastrim Variola minor
c		
35	Odra	Sarampo (v) Sarampão Sarampello
36	Zapalenie rogów przednich rdzenia Choroba Heine-Medina Paraliż dziecięcy	Poliomyelite aguda Paralysia infantil Polioencefalite aguda
37	Zapalenie mózgu	
a	Zapalenie mózgu ze śpiączką	Encephalite infecciosa aguda
b		
c	Spiączka	Encephalite lethargica
38		
a	Żółta febra	Febre amarella Febre amaréla
b	Wścieklizna	Raiva

Roumain — Roumanian	Russe — Russian	N°
Variolă Vărsat		34
Variola major	Оспа — Ospa Оспа натуральная — Ospa naturalnaïa	*a*
Variola minor Alastrim	Вариолоид — Varioloid	*b*
		c
Rugeolă Pojar (y)	Корь — Kor	35
Poliomielită acută Polioencefalită acută Paralizie infantilă	Острый полиомиэлит — Ostryï poliomielit Детский паралич — Detskiï paralitch Полиоэнцефалит — Polioencefalit	36
Encefalită infectioasa acută		37
Encefalită letargică acută Encefalită letargică epidemică	Острый эпидемический энцефалит — Ostryï epidemitcheskiï encefalit Острый летаргический энцефалит — Ostryï letargitcheskiï encefalit	*a*
Parkinsonism Sindromul lui Parkinson	Остатки летаргического энцефалита — Ostatki letargitcheskogo encefalita	*b*
Encefalită letargică	Летаргический энцефалит б. у. — Letargitcheskiï encefalit b.u. Эпидемический энцефалит б. у. — Epidemitcheskiï encefalit b.u.	*c*
Alte boli datorite sau atribuite virusurilor filtrante		38
Febră galbenă	Желтая лихорадка — Jeltaïa likhoradka	*a*
Turbare	Водобоязнь — Vodoboïazn Лисса — Lyssa	*b*

N°	Serbo-croate — Serbo-Croatian	Suédois — Swedish
34		
a	Велике богиње Velike boginje Велике оспице Velike ospice	Smittkoppor
b		
c		
35	Црвен Crven (Tsrven) Мразе Mraze Morbili	Mässling
36	Дечија парализа Dečija paraliza (Dechija paraliza)	Akut barnforlamning
37		Sömnssjuka
a		Akut sömnssjuka
b		
c	Летаргични енцефалитис Letargični encefalitis (Letargichni entsefalitis)	Sömnssjuka
38		
a	Жута грозница Žuta groznica (Juta groznitsa)	Gula febern
b	Беснило Besnilo	Vattuskräck

Tchèque — Czech	Turc — Turkish	N°
Neštovice	Çiçek	**34**
Neštovice pravé	Çiçek	*a*
		b
Neštovice		*c*
Osýpky Spalničky	Kizamik	**35**
Prudký zánět šedé hmoty míšní Prudký zánět šedé hmoty mozkové Prudký zánět předních rohů míšních Medinová nemoc	Poliyomiyelitt	**36**
Zánět mozku epídemický	Ansefalit	**37**
Prudký zánět mozku lethargický		*a*
		b
Zánět mozku epidemický		*c*
		38
Žlutý mor	Sari humma	*a*
Vzteklina	Kuduz	*b*

N°	Nomenclature internationale International Nomenclature	Latin
38*c*	Herpes zoster (Zona)	Herpes zoster Herpes febrilis Febris herpetica
d	Rubéole	Rubeola * Rubella
e	Varicelle	Varicella
f	Autres	
f(*a*)	*Dengue*	Febris dengue
f(*b*)	*Fièvre à pappataci* (fièvre de 3 jours)	Febris phlebotomorum Febris pappataci
	RICKETTSIOSES	
39	**Typhus et autres maladies apparentées au typhus (Rickettsioses)**	
a	Typhus exanthématique (à poux)	Typhus exanthematicus Typhus petechialis Febris hungarica
a(*b*)	*Maladie de Brill*	Morbus Brillii
b	Maladies apparentées au typhus transmises par d'autres vecteurs :	
b(*a*)	à puces : *Typhus endémique bénin* (murin)	Typhus endemicus benignus Typhus endemicus murinus

Allemand — German	Anglais — English	N°
Gürtelrose Zona	Herpes zoster Shingles (v) Zona Herpes febrilis	38*c*
Röteln Rubeolae	German measles Roseola Rubella Epidemic rose rash (v)	*d*
Windpocken (v) Varizellen Spitzpocken Wasserpocken Schafpocken Schafblattern	Chicken-pox Varicella	*e*
Denguefieber Siebentagefieber	Dengue	*f* *f*(*a*)
Pappatacifieber Hundskrankheit * Dreitagefieber	Pappataci fever Sandfly fever Phlebotomus fever	*f*(*b*)
Rickettsiosen	Rickettsioses	39
Flecktyphus Petechialtyphus Fleckfieber Hungertyphus Epidemisches Fleckfieber	Louse-borne typhus fever Exanthematic typhus Epidemic typhus Gaol fever (v)	*a*
Brillsche Krankheit	Brill's disease	*a*(*b*) *b*
Rattenfleckfieber Muriner Flecktyphus Mäuseflecktyphus Durch Flöhe übertragener Flecktyphus	Rat-flea typhus Benign endemic typhus Murine typhus Flea-borne typhus Hone's disease Shop (tropical) typhus	*b*(*a*)

N°	Bulgare — Bulgarian	Danois et norvégien Danish and Norwegian
38c		Herpes Zoster
d	Рубеола Rubeola	Röde Hunde Rödlinger
e	Лешенка Leschenka	Vannkopper Variceller Skoldkopper
f		
f(a)		Denguefeber
f(b)		Pappatacifeber
39		
a	Петнистъ тифъ Petnist tif	Eksantematisk Tyfus Flekkfeber
a(b)		
b		
b(a)		

Espagnol — Spanish	Estonien — Estonian	N°
Zona Herpes zoster		**38***c*
Rubeola Erupción rubeólica	Rubeola	*d*
Varicela	Varicella	*e*
Dengue Fiebre dengue	Febris dengue	*f* *f*(*a*)
Fiebre de pappataci Fiebre de tres dias Fiebre de los flebótomos	Febris pappataci	*f*(*b*)
		39
Tifus petequial Tifus exantematico Tifo negro Tifo europeo Fiebre petequial	Plekitüüfus, Tähniline sœtõbi Tähniline tüüfus	*a*
Enfermedad de Brill		*a*(*b*) *b*
Fiebre de los marinos de Tolón Tifus (tifo) endemico benigno (por pulga de rata) Tifus murino Tabardillo Tifo mejicano Fiebre de Mejico Pinareño (Cuba)	Mexiko-palavik	*b*(*a*)

N°	Finnois — Finnish	Français — French
38*c*		Herpès zoster Zona
d	Vihurirokko	Rubéole
e	Vesirokko	Varicelle
f		
f(a)		Dengue
f(b)		Fièvre de trois jours Fièvre à pappataci Fièvre à phlébotomes
39		Rickettsioses
a	Pilkkukuume	Typhus exanthématique à poux Typhus classique Typhus épidémique Typhus européen Typhus historique Typhus pétéchial Infection à *Rickettsia prowazeki*
a(b)		Maladie de Brill
b		
b(a)		Fièvre nautique de Toulon Typhus murin Typhus endémique à puces Typhus méxicain Infection à *Rickettsia mooseri*

Grec — Greek	Hongrois — Hungarian	Islandais — Icelandic	Nº
		Ristill	38c
Ερύθρωσις Erythrosis		Raudir hundar	d
Ανεμοευλογια Anemoeulogia	Bárányhimlő Varicella		e
			f
Δαγγειοη Daggeion			f(a)
			f(b)
	Kiűteses tifusz ez egyeb hasonló betegsegek		39
Εξανθηματικὸς τῦφος Exanthematikos typhos	Kiűtéses tifusz Kiűtéses hagymáz		a
			a(b)
			b
			b(a)

Nº	Italien — Italian	Letton — Latvian
38c	Erpete zoster Zona Herpes zoster	
d	Rosolia	
e	Varicella	
f		
f(a)	Dengue Febbre dei sette giorni Febbre rossa	
f(b)	Febbre da papataci Febbre di tre giorni Febbre estiva* Febbre da canapa (v)	
39	Rickettsiosi Tifo e tifopetecchialesimili	
a	Tifo petecchiale Tifo esantematico	Izsitumu tifs
a(b)	Febbre di Brill	
b		
b(a)	Tifo endemico benigno Tifo murino Tifo da pulce Dermotifo sporadico (di Catania)	

Lithuanien — Lithuanian	Néerlandais — Dutch	N°
	Herpes zoster Gordelroos	**38***c*
Raudonligé	Rubeola Roode hond Rubeolæ	*d*
Vejaraupsiai	Waterpokken Varicella	*e*
		f
	Dengue (Tropische) Knokkelkoorts Denguekoorts	*f(a)*
		f(b)
		39
Beriamoji šiltiné	Vlektyphus Hongertyphus	*a*
	Ziekte van Brill	*a(b)*
		b
		b(a)

N°	Polonais — Polish	Portugais — Portuguese
38*c*	Polpasiec	Herpes zoster
d	Rozyczka	Rubeola
e	Ospa wietrzna	Catapora Varicella Varicéla
f		
f(a)	Dengue	Dengue
f(b)		Febre de tres dias
39		Typho
a	Tyfus osutkowy Tyfus plamisty Dur osutkowy Dur plamisty	Typho exanthematico Febre petechial Rickettsiose epidemico Tifus epidemico classico Tifo exantematico (piolko)
a(b)		Doença de Brill
b		
b(a)		Riquetsiose murina Tifus murino

Roumain — Roumanian	Russe — Russian	N°
Herpes zoster Zona	Опоясывающий лишай — Opoïasyvaïustchiï lichaï Герпес зостер — Herpes zoster Поясовидная рожа — Poïasovidnaïa roja (v)	38c
Rubeolă	Краснуха — Krasnukha	d
Varicelă	Ветряная оспа — Vetrianaïa ospa	e
	Денга — Denga	f f(a)
Febra de trei zile	Трехдневная лихорадка (паппатаки) — Trekhdnevnaïa likhoradka (Pappataki)	f(b
Rickettsioze	Риккетциозы — Riketsiozy	39
Tifus exantematic (prin păduchi)	Сыпной тиф (от вшей) — Sypnoï tif (ot vcheï)	a
Boala lui Brill	Бриллиева болезнь — Brillieva bolezn	a(b)
Boli inrudite cu tifusul transmise prin alti vectori :		b
Prin purici :		
Tyfus endemic benin (murin)	Легкий эндемический тиф (от блох) — Lekhkiï endemitcheskiï tif (ot blokh) Крысный тиф — Krysnyï tif	b(a)

N°	Serbo-croate — Serbo-Croatian	Suédois — Swedish
38c		
d	Рубеола Rubeola	Röda hund
e	Мале богиње Male boginje Козице Kozice (kozitse)	Vattenkoppor
f *f(a)*	Денга Denga	Denguefeber
f(b)	Вардарска грозница Vardarska groznica (Vardarska groznitsa)	
39		
a	Пегавац Pegavac (Pegavatz) Пегави тифус Pegavi tifus	Fläcktyfus Fläckfeber
a(b) *b*		
b(a)		

Tchèque — Czech	Turc — Turkish	N°
		38c
		d
Neštovice vodní Neštovice plané Vodnatky	Luçiçeği	*e*
		f *f(a)*
		f(b)
		39
Týf skvrnitý Skvrnivka	Lekeli humma	*a*
		a(b) *b*
		b(a)

N°	Nomenclature internationale International Nomenclature	Latin
39*b*(*b*)	à Trombididés (acariens) : *Fièvre fluviale du Japon*	Febris fluvialis japonica
b(*c*)	à tiques :	
b(*ca*)	*Fièvre boutonneuse*	Febris exanthematosa mediterranea Febris verrucosa mediterranea
b(*cb*)	*Fièvre par piqûre de tiques* (Afrique du Sud)	
b(*cc*)	*Fièvre pourprée des Montagnes Rocheuses*	Febris purpurea montium saxosorum
b(*cd*)	*Typhus de São-Paulo*	Typhus Sancti Pauli
b(*ce*)	*Fièvre « Q »* du Queensland	Febris « Q » Febris Reginæ Terræ
c	Rickettsioses autres et non spécifiés	
c(*a*)	*Fièvre de 5 jours*	Febris wolhynica Rickettsiosis quintana Febris neuralgica periodica

MALADIES DUES A DES HELMINTHES

40	**Ankylostomiase**	Ankylostomiasis Ankylostomum duodenale

Allemand — German	Anglais — English	N°
		39*b*(*b*)
Japanisches Flussfieber Tsutsugamushi	Japanese river fever Tsutsugamushi mite fever Jungle (tropical) typhus Scrub typhus	
		b(*c*)
Beulenfieber	Boutonneuse fever Exanthematous fever	*b*(*ca*)
Süd-Afrikanisches exanthematisches Zeckenfieber	South African tick-bite fever	*b*(*cb*)
Rocky-Mountain Fleckfieber	Rocky Mountains spotted fever	*b*(*cc*)
Exanthematisches Zeckenfieber von São-Paulo	São Paulo tick-typhus	*b*(*cd*)
Q-Fieber	Q-fever Queensland fever Mossman fever	*b*(*ce*)
		c
Fünftagefieber Wolhynisches Fieber Febris neuralgica periodica Schützengrabenfieber Periodisches Fieber	Trench fever	*c*(*a*)
Wurmkrankheit der Bergleute (v) Hakenwurmkrankheit Ankylostomiasis	Hookworm Uncinariasis Miners' anæmia	40

N°	Bulgare — Bulgarian	Danois et norvégien Danish and Norwegian
39*b*(*b*)		
b(*c*)		
b(*ca*)		
b(*cb*)		
b(*cc*)		
b(*cd*)		
b(*ce*)		
c		
c(*a*)		
		Helminthiases
40		Anchilostomiase Ankylostomiase

Espagnol — Spanish	Estonien — Estonian	N°
		39*b(b)*
Fiebre fluvial del Japón		
		b(c)
Fiebre botonosa		*b(ca)*
Fiebre exantematica mediterranea (de Olmer)		
		b(cb)
Fiebre maculosa de las Montañas Rocosas		*b(cc)*
Typho de S. Paulo (por carrapatas)		*b(cd)*
Fiebre « Q »		*b(ce)*
		c
Fiebre de cinco dies		*c(a)*
Fiebre de las trincheras		
Anquilostomiasis	Ankülostomiaas	**40**
Amarellao		
Anemia epidemica		
Anemia de Egipto		
Uncinariasis		

N°	Finnois — Finnish	Français — French
39*b*(*b*)		Tsutsugamushi Fièvre fluviale du Japon Typhus tropical de brousse Maladie de Kedani Infection à *Rickettsia orientalis*
b(*c*)		
b(*ca*)		Fièvre boutonneuse Fièvre exanthématique Maladie de Conor et Bruch Infection à *Rickettsia conori*
b(*cb*)		Typhus à tiques sud-africain
b(*cc*)		Fièvre tachetée (ou pourprée) des Montagnes Rocheuses Infection à *Rickettsia rickettsi*
b(*cd*)		Fièvre pourprée de São Paulo, de Minas Geraes
b(*ce*)		Fièvre « Q » Fièvre des coupeurs de cannes du Queensland Infection à *Rickettsia burneti* (= *R. diasporica*)
c		
c(*a*)		Fièvre de cinq jours Fièvre des tranchées Fièvre de Volhynie Infection à *Rickettsia quintana*
		Helminthiases
40		Ankylostomiase Uncinariose Anémie des mineurs Anémie d'Egypte

Grec — Greek	Hongrois — Hungarian	Islandais — Icelandic	N°
			39*b(b)*
			b(c)
			b(ca)
			b(cb)
			b(cc)
			b(cd)
			b(ce)
			c
			c(a)
Ελμινθας Elminthas	Fergek okozta betegségek = Helminthiases		
Αγκυλοστομιασεις Ankylostomiaseis	Bányászaszály Anchylostomiasis		**40**

N°	Italien — Italian	Letton — Latvian
39*b(b)*	Febbre fluviale del Giappone Febbre di tsutsugamushi Tifo da acaro	
b(c)		
b(ca)	Febbre bottonosa Febbre di Marsiglia Tifo da zecca minore Dermotifo benigno Dermotifo endemico*	
b(cb)		
b(cc)	Tifo da zecca maggiore Tifo (febbre) delle Montagne Rocciose Febbre maculata Febbre purpurea delle Montagna Rocciose	
b(cd)	Tifo (da zecca maggiore) di São Paulo	
b(ce)	Febbre « Q »	
c		
c(a)	Febbre quintana Febbre volinica Febbre delle trincee Febbre dei cinque giorni	
	Elminthiasi	
40	Anchilostomiasi Ipoemia dei minatori (v) Uncinariosi Anemia epidemica*	

Lithuanien — Lithuanian	**Néerlandais — Dutch**	N°
		39*b*(*b*)
	Sumatraansche mijtekoorts	
		b(*c*)
		b(*ca*)
		b(*cb*)
		b(*cc*)
		b(*cd*)
	« Q » koorts	*b*(*ce*)
		c
	Vijfdaagsche koorts	*c*(*a*)
	Wormziekte	
	Ankylostomiasis Anchylostomiasis Haakworm ziekte	**40**

N°	Polonais — Polish	Portugais — Portuguese
39*b*(*b*)		Tsutsugamushi
b(*c*)		
b(*ca*)		Febre botonosa
b(*cb*)		
b(*cc*)		Febre maculosa dos Montanhas Rochosas
b(*cd*)		Typho endemico (exanthematico) de São Paulo Rickettsiose neotropica paulista Febre maculosa brasileira Riquetsiose maculosa neotropica
b(*ce*)		Febre « Q »
c		
c(*a*)	Gorączka pięciodniowa	Febre de cinco dias
40		Ancylostomiase Amarellão Opilação Ancilostomiase

Roumain — Roumanian	Russe — Russian	N°
Prin acarieni : Febră fluviala japonă		**39***b(b)*
Prin căpuse : Febră butonoasă		*b(c)* *b(ca)*
Febra prin intepaluri de căpuse (Africa de Sud)		*b(cb)*
Febra purpurie Muntilor Stancoşi		*b(cc)*
Tifusul din São-Paulo		*b(cd)*
Febra « Q » (Queensland)		*b(ce)*
		c
Febra de cinci zile	Пятидневная лихорадка — Piatidnevnaïa likhoradka Волынская лихорадка — Wolynskaïa likhorada	*c(a)*
Anchilostomiază	Анкилостомиаз — Ankilostomias Анкилостомидоз — Ankilostomidoz	**40**

N°	Serbo-croate — Serbo-Croatian	Suédois — Swedish
39*b(b)*		
b(c)		
b(ca)		
b(cb)		
b(cc)		
b(cd)		
b(ce)		
c		
c(a)		
40	Анкилостомија Ankilostomija	Ankylostomasjukdom

Tchèque — Czech	Turc — Turkish	N°
		39*b(b)*
		b(c)
		b(ca)
		b(cb)
		b(cc)
		b(cd)
		b(ce)
		c
		c(a)
Ankylostomiasa Červivost		**40**

N°	Nomenclature internationale International Nomenclature	Latin
41	**Maladie hydatique**	Hydatides echinococci Echinococcosis Echinococcus
a	Foie	Hydatides hepatis Echinococcosis hepatis Toenia echinococcus hepatis
b	Localisations autres et non spécifiées	Hydatides pulmonum
42	**Autres maladies dues à des helminthes**	Ceteræ helminthiases
(a)	*Ascaridiose*	Ascaridiosis
(b)	*Bilharziose*	Bilharziosis
(c)	*Cysticercose*	Cysticercus
(d)	*Distomatoses*	Distomatosis Distomum hepaticum Distomum pulmonale
(e)	*Filariose*	Filariasis Elephantiasis arabum Filaria sanguinis
(f)	*Oxyurose*	Oxyurasis
(g)	*Toenia*	Toeniasis, Tænia Toenia solium Toenia saginata

Allemand — German	Anglais — English	N°
Echinokokkus Hydatiden Cyste Hundewurm Krankheit	Hydatid disease Echinococcus infection Hydatid cysts	**41**
Echinokokkus der Leber	Hydatid cysts (disease) of the liver	*a*
	Tænia echinococcus	*b*
	Helminthiasis	**42**
Ascaridiosis	Ascariasis	*(a)*
Bilharziasis Schistosomiasis	Bilharziasis Schistosomiasis Chyluria	*(b)*
Blasenwurm (v) Cysticercus	Cysticercosis	*(c)*
Distomatosis Menschliche Eingeweidewürmer Distoma	Distomiasis Fluke disease Paragonimiasis	*(d)*
Filariasis Filaria Fadenwurm	Filariasis Elephantiasis (filarial)	*(e)*
Oxyurasis Madenwurm (v) Springwurm (v)	Oxyurus vermicularis Pin worms (v) Threadworms	*(f)*
Bandwurm (v) Bandwurmkrankheit Taeniasis	Tæniasis Tape worms (v)	*(g)*

N°	Bulgare — Bulgarian	Danois et norvégien Danish and Norwegian
41		Hundeorm Echinokokksykdom
a		
b		
42		Andre Ormsykdommer
(a)		Ascaridiose
(b)		Bilharziose
(c)		Cysticercose
(d)		Distomatose
(e)		Filariose
(f)		Oxyurose
(g)		Bændelorm Bendelorm

Espagnol — Spanish	Estonien — Estonian	N°
Quiste hidatidico Hidatides Echinocosis Echinococos	Ehhinokokk Põistang	**41**
Quiste hidatidico del higado	Põistang maksas	*a*
	Põistang mujal	*b*
Otras enfermedades debidas a los helmintos	Teised haigused	**42**
Ascaridiosis	Ascaridiosis	*(a)*
Bilharziosis Quiluria Hematuria de los paises calidos Lipemia	Piimataoline uriin	*(b)*
Cisticercosis Ladrería	Cysticercus	*(c)*
Distomatosis	Distomatosis	*(d)*
Filariosis Elefantiasis de los árabes	Vaïke-aasias	*(e)*
Oxiurosis	Oxyurosis	*(f)*
Teniasis Lombriz solitaria	Tæniasias Siseelundkonna parasiidid	*(g)*

N°	Finnois — Finnish	Français — French
41		Kystes hydatiques Hydatides Echinococcose
a		Echinococcose du foie
b		
42		Helminthiases
(a)		Ascaridiose
(b)		Bilharziose Schistosomiase Chylurie* Hématurie des pays chauds*
(c)		Cysticercose Ladrerie (v)
(d)		Distomatose Cirrhose périportale Douves Fasciolase Hétérophiase
(e)		Filariose Dracunculose Eléphantiasis Ver de Médine
(f)		Oxyurose
(g)		Tœnia Ver solitaire (v)

Grec — Greek	Hongrois — Hungarian	Islandais — Icelandic	N°
Κύστις ὑδατική Kystis hydatikè	Hólyagféreg Echinococcus	Sullaveiki	**41**
	Hólyagféreg majban		*a*
	Hólyagféreg	Sullaveiki	*b*
Αλλαι νοσοι οφειλομεναι εις ἑλμινθας Allai nosoi opheilomenai eis elminthas Ελμινθας Elminthas	Bélférgek okozta egyeb betegségek		**42**
	Ascaris gyakorisága		*(a)*
	Bilharzia gyakorisága		*(b)*
	Cysticercus gyakorisága		*(c)*
	Distoma gyakorisága		*(d)*
	Filaria gyakorisága		*(e)*
	Oxyuriasis Oxyurus gyakorisága		*(f)*
Ταινια Tainia			*(g)*

N°	Italien — Italian	Letton — Latvian
41	Cisti idatica (idatiche) Echinococcosi	
a	Cisti idatica del fegato Echinococcosi epatica	
b		
42	Altre malattie dovute a elminti Elmintiasi	
(a)	Ascaridiosi	
(b)	Bilharziosi Schistosomiasi Malattia di Kotayama	
(c)	Cisticercosi	
(d)	Distomatosi Distomiasi Paragonimiasi polmonare	
(e)	Filariosi Elefantiasi	
(f)	Ossiuriosi Enterobiasi	
(g)	Teniasi Verme solitario (v)	

Lithuanien — Lithuanian	Néerlandais — Dutch	N°
	Hydatiden Blaasworm	**41**
	Hydatiden van de lever	*a*
	Hydatiden van de overige organen Draaiziekte (vet.)	*b*
	Overige wormziekte	**42**
	Ascaris-infectie	*(a)*
	Bilharziosis	*(b)*
	Cysticercosis	*(c)*
	Distomatosis	*(d)*
	Filariasis	*(e)*
	Oxyurosis	*(f)*
	Lintworm Tœniasis	*(g)*

Nº	Polonais — Polish	Portugais — Portuguese
41	Bąblowiec	Molestia hidatica
a		Tumor hydatico do figado
b		
42		Outras molestias devidas a helmintos
(a)	Glista	
(b)	Bilcharcia	
(c)	Bąblowiec	
(d)		
(e)		Elephantiasis
(f)		
(g)	Tasiemiec	Tenia Lombriga solitaria

Roumain — Roumanian	Russe — Russian	N°
Boala hidatică Chist hidatic		**41**
Boala hidatică Ficatului	Эхинококк печени — Ekhinokokk petcheni	*a*
	Эхинококк б. у. — Ekhinokokk b.u. Гидатиды б. у. — Gidatidy b.u.	*b*
Alte boli datorite helmintilor	Болезни причиненныя гелминтамы — Bolezni pritchinionnya guelmintamy	**42**
Ascaridioză	Аскаридиозы — Askaridiozy	*(a)*
Bilharzioză	Билярциозы — Biliartsiozy	*(b)*
Cisticercoză	Цистеркозы — Tsisterkozy	*(c)*
Distomatoză (galbază)	Дистоматозы — Distomatozy	*(d)*
Filarioză	Филяриозы — Filiariozy	*(e)*
Oxyuroză	Нитевидная глиста — Nitevidnaïa glista Оксириозис — Oksiriosis	*(f)*
Tenie Panglică	Тэния — Taenia Солитер — Soliter Ленточная глиста — Lentotchnaïa glista Цепень — Tsepen	*(g)*

N°	Serbo-croate — Serbo-Croatian	Suédois — Swedish
41		Blåsmasksjukdom
a		
b		
42		Andra masksjukdomar
(a)		
(b)		
(c)		
(d)		
(e)		
(f)		
(g)	Пантљичара Pantljičara (Pantljichara) Тракавица Trakavica (Trakavitsa)	Binnikemasksjukdom

Tchèque — Czech	Turc — Turkish	N°
Boubel mechožilová		**41**
Boubel mechožilová játer		*a*
Boubel mechožilová jiných míst		*b*
Jiné nemoci zaviněné červy		**42**
		(a)
		(b)
		(c)
		(d)
		(e)
Škrkavka		*(f)*
Tasemnice	Şirit	*(g)*

N°	Nomenclature internationale International Nomenclature	Latin
42(h)	*Trichinose*	Trichinosis Trichina spiralis Trichinellosis
(i)	*Autres vers*	
(j)	*Vers non spécifiés*	Vermes n.s. Helminthiasis n.s. Verminatio n.s.
	MYCOSES	
43	**Mycoses**	Mycoses
(a)	*Muguet*	Stomatomycosis
(b)	*Teigne*	Herpes tonsurans Trychophyton tonsurans
(c)	*Autres mycoses*	
	AUTRES MALADIES INFECTIEUSES OU PARASITAIRES	
44	**Autres maladies infectieuses ou parasitaires**	
a	Maladies vénériennes, sauf syphilis et gonococcie	
a(a)	*Chancre mou*	Ulcus molle
a(b)	*Lymphogranulome vénérien*	Lymphogranuloma venereum Lymphogranuloma inguinale
b	Lymphogranulomatose maligne (maladie de Hodgkin)	Lymphogranuloma malignum Lymphogranulomatosis maligna

Allemand — German	Anglais — English	N°
Trichinenkrankheit Trichinellenkrankheit Trichinosis Haarwurm Krankheit (v)	Trichiniasis Trichina spiralis	42(h)
	Myiasis	(i)
Wurmkrankheit	Worms* Helminthiasis*	(j)
Mykosis	Mycoses	43
Mundschwämmchen (v) Stomatomycosis	Thrush Stomatomycosis	(a)
Trichophytie Mikrosporie Herpes tonsurans	Ringworm Trichophytosis	(b)
		(c)
		44
		a
Weicher Schanker Ulcus molle	Soft chancre Soft sore	a(a)
Lymphogranuloma venereum	Esthiomene Poradenitis Granuloma pudendorum Climatic bubo Lymphogranulome inguinale	a(b)
Hodgkinsche Krankheit Infektiöses Granulom	Pernicious lymphogranulomatosis Hodgkin's disease	b

N°	Bulgare — Bulgarian	Danois et norvégien Danish and Norwegian
42(h)	Трихиноза Trichinoza	Trichinose Trikinose
(i)		
(j)		Ormesygdomme
43		Mykoser
(a)		
(b)	Трихофитија Trichofitiya	Ringorm
(c)		
44	Други заразни и паразитни болести Drugi zarazni i parazitni bolesti	
a		
a(a)		Veneriske Saar Veneriske Sår Ulcus molle Blödchanker Blöt-chanker
a(b)		Venerisk Lymfogranulom
b		Malign Lymfogranulomatose

Espagnol — Spanish	Estonien — Estonian	N°
Triquinosis	Trichinosis	**42***(h)*
	Teised	*(i)*
Lombrices intestinales Cólico verminoso Helmintos s.o.i.		*(j)*
Micosis	Mükoos Seentõbi	**43**
Muguet		*(a)*
Tiñas Tricoficia	Mitmesugused seenhaigused	*(b)*
		(c)
		44
		a
Chancrillo Chancro simple Potro (v) Chancro blando Chancro venereo	Bubo ulceris mollis	*a(a)*
Linfogranulomatosis inguinal Enfermedad poradenica Bubon virulento Bubon de absorción Bubón venereo Adenitis venerea	Adenitis venerea	*a(b)*
Linfogranulomatosis s.o.i. Enfermedad de Hodgkin		*b*

N°	Finnois — Finnish	Français — French
42(h)		Trichinose
(i)		Myiase
(j)		Vers intestinaux Infection vermineuse Helminthiase Coliques vermineuses (v)
43		Mycoses
(a)		Muguet
(b)		Teigne Trichophytie
(c)		
44		
a		
a(a)	Pehmeä haava Epäiltiva haava	Chancre mou Chancrelle (v) Poulain (v) Adénite vénérienne
a(b)		Bubon phagédénique Esthiomène Maladie de Nicolas-Favre Bubon vénérien* Bubon climatique Quatrième maladie vénérienne
b		Lymphogranulomatose maligne Lymphadénie maligne Maladie de Hodgkin

Grec — Greek	Hongrois — Hungarian	Islandais — Icelandic	N°
			42(h)
			(i)
Ελμινθας Elminthas			(j)
Μυκώσεις Mykoseis	Fonalgombák által okozott betegségek	Geislasveppsbólga	43
	Szájpenesz		(a)
			(b)
	Sugár gombabetegs	Geitur	(c)
	Fertőző es elős okozta egyeb betegsegei		44
			a
	Lágyfekély		a(a)
			a(b)
			b

N°	Italien — Italian	Letton — Latvian
42(h)	Trichinosi	
(i)	Miasi Myasis	
(j)	Vermi intestinali Colica verminosa	
43	Micosi	
(a)	Mighetto Afte parassitiche Mughetto	
(b)	Tigna Dermatomicosi Tricofizie	
(c)		
44	Altre malattie infettive e parassitarie	
a		
a(a)	Cancro molle Ulcera molle Ulcera venerea Ulcera semplice	Mīkstais Šankers
a(b)	Linfogranuloma inguinale Quarta malattia venerea Bubbone climatico Bubbone fagedenico	
b	Linfogranulomatosi Morbo di Hodgkin	

Lithuanien — Lithuanian	Néerlandais — Dutch	N°
	Trichinellosis	**42**(*h*)
		(*i*)
	Wormziekte	(*j*)
	Mycosen	**43**
	Spruw	(*a*)
	Herpes tonsurans Schurft (v) Trichophytie Hoofdzeer (v)	(*b*)
		(*c*)
Kitos infekcines ligos		**44**
		a
Minkštasis šankeris	Ulcus molle Weke schanker Zachte schanker	*a*(*a*)
	Venerisch granuloom	*a*(*b*)
	Ziekte van Hodgkin	*b*

Nº	Polonais — Polish	Portugais — Portuguese
42(h)	Trychinoza	Trichina Gafeira (v)
(i)		
(j)		Helminthes s.o.i. Helmintos
43		Mycoses Micoses
(a)		
(b)		Tinha Pellada
(c)		
44		
a		
a(a)	Wrzód miękki	Cancro molle Cavallo (v) Boubas venereas
a(b)		Lymphogranuloma venerea
b	Ziarnica złośliwa	Molestia de Hodgkin Lymphadenoma de Hodgkin Linfogranulomatose maligna

Roumain — Roumanian	Russe — Russian	№
Trichinoza	Трихина — Trikhina	**42**(*h*)
Alti helminti		(*i*)
Vermi nespecificati	Черви б. у. — Tchervi b.u.	(*j*)
Micoze	Микозы — Mikozy	**43**
	Молочница — Molotchnitsa	(*a*)
	Стригучий лишай — Strigutchiï lichaï Парша — Parcha	(*b*)
		(*c*)
Alte boli infectioase sau parazitare		**44**
Boli venerice, fără sifilis și gonococie		*a*
Sancăr moale	Мягкий шанкр — Miagkiï chankr	*a*(*a*)
Limfogranulom veneric	Венерический лимфогранулом — Veneritcheskiï limfogranulom	*a*(*b*)
Limfogranulomatoză malignă Boala lui Hodgkin	Болезнь Годкина — Bolezn Godkina	*b*

N°	Serbo-croate — Serbo-Croatian	Suédois — Swedish
42(h)	Трихноза Trihnoza	Trikinsjukdom
(i)		
(j)		
43		
(a)		
(b)		Skorv
(c)		
44		
a		
a(a)	Мекани чангир Mekani čangir (Mekani changir)	Schanker Chancre
a(b)		
b		Lymfogranulomat

Tchèque — Czech	Turc — Turkish	N°
		42(*h*)
		(*i*)
Červivka Hlísti		(*j*)
Mykosy		**43**
Moučník		(*a*)
Strupovitost		(*b*)
Aktinomykosa		(*c*)
Jiné nemoci nakažlivé nebo cizopasníky působené		**44**
		a
Hlíza Měkký vřed		*a*(*a*)
		a(*b*)
Nemoc Hodkinova		*b*

N°	Nomenclature internationale International Nomenclature	Latin
44*c*	Oreillons	Parotitis epidemica Parotis
d	Autres maladies infectieuses ou parasitaires	
d(a)	*Fièvre aphteuse*	Aphtae epizooticae Stomatitis aphthosa epidemica
d(b)	*Mononucléose infectieuse*	Mononucleosis infectiosa
d(c)	*Myosite épidémique* (maladie de Bornholm)	Myositis epidemica Myalgia epidemica Morbus Boringiæ
d(d)	*Psittacose*	Psittacosis
d(e)	*Quatrième maladie* (rubéole scarlatiniforme)	Rubeola scarlatinosa Morbus quartus
d(f)	*Suette miliaire*	Febris miliaris Sudor miliaris Sudor anglicus
d(g)	*Ictère épidémique*	Hepatitis epidemica Icterus epidemicus
d(h)	*Hoquet épidémique*	Singultus epidemicus
II	**AUTRES AFFECTIONS DE CARACTÈRE INFECTIEUX, NON INCLUSES DANS LE CHAPITRE I DE LA NOMENCLATURE INTERNATIONALE**	
58	**Rhumatisme articulaire aigu** (‡)	Rheumatismus articulorum acutus Febris rheumatica

Allemand — German	Anglais — English	N°
Ziegenpeter (v) Mumps	Mumps Epidemic parotitis	**44***c*
		d
Maul- und Klauenseuche (vet.)	Foot and mouth disease (vet.)	*d(a)*
Pfeiffersche Krankheit Drüsenfieber	Infectious mononucleosis Glandular fever	*d(b)*
Bornholmer Krankheit	Epidemic myositis Bornholm disease	*d(c)*
Psittakosis Papageikrankheit	Psittacosis	*d(d)*
Vierte (exanthematische) Krankheit	Fourth disease Dukes's disease Rubella scarlatinosa	*d(e)*
Frieseln Schweissfrieseln Schweissfieber	Miliary fever Sweating sickness	*d(f)*
Epidemische Gelbsucht Hepatitis epidemica	Epidemic hepatitis Epidemic catarrhal jaundice	*d(g)*
	Epidemic hiccup	*d(h)*
Akuter fieberhafter Gelenkrheumatismus	Rheumatic fever Acute rheumatic endocarditis	**58**

N°	Bulgare — Bulgarian	Danois et norvégien Danish and Norwegian
44c	Заушка Zauchka	Kusma Faaresyge
d		
d(a)		
d(b)		
d(c)		Bornholmsk Sygdom Epidemisk Myalgi
d(d)		Papagöjesygdom
d(e)		Fjerde Sygdom
d(f)		
d(g)		Epidemisk Gulsot
d(h)		Hikkesyge
58	Остеръ ставенъ ревматизъмъ Oster staven revmatizem	Gigtfeber Giktfeber

Espagnol — Spanish	Estonien — Estonian	N°
Orejones Ourles	Infektsioon kõrvanäärmeis	**44***c*
		d
		d(a)
		d(b)
		d(c)
Psitacose Psitacosis	Psittacosis	*d(d)*
Cuarta enfermedad Enfermedad de Duke	Morbus quartus	*d(e)*
Sudor miliar Fiebre miliar	Sudatio miliaris	*d(f)*
Ictericia epidemica		*d(g)*
		d(h)
Reumatismo articular agudo febril Artritis reumática aguda Reumatismo febril	Äge liigeste reumatism	**58**

N°	Finnois — Finnish	Français — French
44*c*	Sikotauti	Oreillons Parotidite épidémique Ourles (v)
d		
d(a)		Fièvre aphteuse
d(b)		Mononucléose infectieuse Angine à monocytes Fièvre glandulaire de Pfeiffer
d(c)		Myosite épidémique Maladie de Bornholm
d(d)		Psittacose
d(e)		Rubéole scarlatiniforme Maladie de Dukes-Filatow Quatrième maladie
d(f)		Suette miliaire Fièvre miliaire*
d(g)		Ictère épidémique Hépatite épidémique
d(h)		Hoquet épidémique
58	Akillinen nivelreumatismi	Rhumatisme articulaire aigu Maladie de Bouillaud Rhumatisme fébrile

Grec — Greek	Hongrois — Hungarian	Islandais — Icelandic	N°
Παροτιτις ἐπιδημική Parotitis epidemikè	Fültőmirigylob (Jarvány —)	Hettusótt	**44***c*
			d
			d(a)
			d(b)
			d(c)
			d(d)
			d(e)
			d(f)
		Gulusótt Umferdargula	*d(g)*
			d(h)
Αρθριτικὸς ρευματισμος Arthritikos rheumatismos	Heveny lázas sokizületű csúz	Gigtsótt	**58**

N°	Italien — Italian	Letton — Latvian
44c	Orecchioni Parotite epidemica	Epidēmiskais pieausses dziedzera iekaisums
d		
d(a)		
d(b)	Febbre ghiandolare Mononucleosi infettiva Febbre di Pfeiffer	
d(c)		
d(d)	Psittacosi	
d(e)	Quarta malattia Malattia di Duke	
d(f)	Febbre miliare	
d(g)	Itterizia epidemica Ittero castrense lieve Itterizia catarrale epidemica	
d(h)		
58	Reumatismo articolare acuto (febbrile) Reumatismo febbrile Artrite reumatica acuta	Akūtais locitāvu reimatisms

Lithuanien — Lithuanian	Néerlandais — Dutch	N°
Epid. seilės liaukų uždegimas	Bof	**44***c*
		d
Snukio ir nagų liga	Mondzeer Mond en klauwzeer (vet.) Tongblaar	*d(a)*
	Klierkoorts	*d(b)*
	Bornholmsche ziekte	*d(c)*
	Psittacosis Papagaaienziekte	*d(d)*
	Vierde ziekte	*d(e)*
	Zweetkoorts	*d(f)*
	Epidemische geelzucht	*d(g)*
		d(h)
	Acute gewrichtsrheumatiek	**58**

N°	Polonais — Polish	Portugais — Portuguese
44c	Zapalenie przyusznicy Swinka	Parotidite infecciosa Papeira Parotidite epidemica
d		
d(a)	Gorączka opryszczkowa	
d(b)	Mononukleoza zakaźna	
d(c)	Epidemiczne zapalenie mięśni	
d(d)	Choroba papuzia	
d(e)	Choroba czwarta	Quarta doença
d(f)		Suor maligno miliar
d(g)	Żółtaczka epidemiczna	Ictericia epidemica
d(h)		
58	Gościec stawowy ostry Zapalenie reumatyczne stawów	Rheumatismo articular agudo febril Reumatismo articular agudo Molestia de Buillaud

Roumain — Roumanian	Russe — Russian	N°
Oreioane Oreion	Заушница — Zaouchnitsa Эпидемический паротит — Epidemitcheskiĭ parotit	**44***c*
		d
Febra aftoasă	Афтная лихорадка — Aftnaïa likhoradka Молочница — Molotchnitsa (v) Ящур — Iastchur (v)	*d(a)*
Mononucleoza infectioasă	Инфекционный мононуклеоз — Infektioznyï mononukleoz	*d(b)*
Miozita epidemică Boala din Bornholm	Эпидемическое воспаление мышец — Epidemitcheskoïe vospalenie mychets Болезнь Борнгольма — Bolezn Borngolma	*d(c)*
Psitacoza	Пситтакоз — Psittakoz	*d(d)*
Maladia a patra Rubeolă scarlatiniformă	Четвертая болезнь — Tchetvertaïa bolezn Скарлатинообразная краснуха — Skarlatino obraznaïa krasnukha	*d(e)*
	Потовая горячка — Potovaïa goriatchka (v) Миллиарная лихорадка — Milliarnaïa likhoradka Потница — Potnitsa	*d(f)*
Icter epidemic Galbinare epidemică	Эпидемическая желтуха — Epidemitcheskaïa jeltukha	*d(g)*
		d(h)
Reumatism articular acut febril Boala lui Bouillaud Pericardită reumatismală acută Endocardită reumatismală acută Miocardită reumatismală acută Pleurezie reumatismală	Острый сочленовный ревматизм — Ostryï sotchlenovnyï revmatizm Суставной ревматизм — Sustavnoï revmatizm	**58**

N°	Serbo-croate — Serbo-Croatian	Suédois — Swedish
44c	Заушњаци Zaušnjaci (Zaushnjaci)	Påssjuka
d		
d(a)	Шап Šap (Shap)	
d(b)		
d(c)		Bornholmsjukdom
d(d)	Пситакоза Psitakoza	
d(e)		Fjärde sjukdom
d(f)		
d(g)	Заразна жутица Zarazna žutica (Zarazna jutitsa)	Infektiös ikterus Smittsam gulsot
d(h)		
58	Зглобни реуматизам Zglobni reumatizam	Reumatisk feber

Tchèque — Czech	Turc — Turkish	N°
Zánět přiušnice	Kabakulak	**44***c*
		d
Slintavka a kulhavka (vet.)		*d(a)*
		d(b)
		d(c)
		d(d)
		d(e)
		d(f)
Epidemická žloutenka		*d(g)*
		d(h)
Rheumatismus kloubní prudký horečnatý. Akutní hostek kloubní		**58**

N°	Nomenclature internationale International Nomenclature	Latin
81	**Méningite non méningococcique**	Meningitis non meningococcica
a	Méningite simple	Meningitis simplex Pachymeningitis
b	Méningite cérébro-spinale aiguë non méningococcique	
88(†)*(a)*	*Trachome*	Trachoma Conjunctivitis granosa Conjunctivitis trachomatosa Conjunctivitis follicularis
89	**Maladies de l'oreille et des sinus mastoïdiens**	Morbi auris
a	Otite et autres maladies de l'oreille sans mention d'affection mastoïdienne	
a(a)	*Otite*	Otitis media acuta Otitis media purulenta
b	Maladies des sinus mastoïdiens	
b(a)	*Mastoïdite*	Mastoiditis
91	**Endocardite aiguë non rhumatismale**	
a	Endocardite bactérienne aiguë	Endocarditis bacteriana acuta
b	Endocardite bactérienne subaiguë (à *Streptococcus viridans*)	Endocarditis bacteriana subacuta Endocarditis lenta
c	Autres formes d'endocardite aiguë ou subaiguë (non artérioscléro-tique)	

Allemand — German	Anglais — English	N°
Hirnhautentzündung	Meningitis (non-meningococcal)	**81**
Einfache Hirnhautentzündung	Simple meningitis	*a*
Akute Hirnhautentzündung	Pachymeningitis	*b*
Körnerkrankheit Trachom Ägyptische Augenkrankheit	Trachoma Granular conjunctivitis	**88***(a)*
		89
		a
Ohrenentzündung Otitis Mittelohrentzündung	Otitis Otitis media Abscess of middle ear	*a(a)*
		b
Warzenfortsatzentzündung	Mastoiditis Mastoid abscess Mastoid disease	*b(a)*
		91
Akute Endokarditis (bakterielle)	Acute bacterial endocarditis (non-rheumatic)	*a*
Endokarditis lenta	Subacute bacterial endocarditis Endocarditis lenta	*b*
		c

N°	Bulgare — Bulgarian	Danois et norvégien Danish and Norwegian
81		
a		Hjærnehindbetændelse
b		Purulent Meningitt
88*(a)*	Трахома Trachoma	Trakom
89		
a		
a(a)		Örebetændelse Örebetennelse
b *b(a)*		Mastoidit
91		
a		Akutt bakterisk Endokarditt
b		*Str. viridans* subakutt Endokarditt
c		

Espagnol — Spanish	Estonien — Estonian	N°
		81
Meningitis simple	Lihtne ajukelme põletik	*a*
Meningitis infecciosa Meningitis no-meningococica Meningitis s.o.i. Meningitis purulentá s.o.i.		*b*
Tracoma Conjuntivitis folicular Conjuntivitis tracomatosa	Silmamarjad	**88***(a)*
		89
		a
Otitis Catarro del oido	Korvapõletik	*a(a)*
		b
Mastoiditis Absceso del seno mastoideo	Sinus mastoideuse põlitik	*b(a)*
		91
Endocarditis aguda Endocarditis infecciosa	Äge batsillaarne südame sisekesta-põletik	*a*
Endocarditis lenta	*Str. viridans* südame sisekesta-põletik	*b*
		c

N°	Finnois — Finnish	Français — French
81	Aivokalvontulehdus	
a		Méningite simple
b		Méningite aseptique aiguë Méningite à B. de Pfeiffer Méningite à pneumocoques Méningite à streptocoques, etc. Pachyméningite
88*(a)*		Trachome Conjonctivite phlycténulaire
89		
a		
a(a)		Otite
b		
b(a)		Mastoïdite
91		Endocardite aiguë non rhumatismale
a		Endocardite infectieuse aiguë Endocardite bactérienne aiguë
b		Endocardite lente Endocardite maligne Endocardite à streptocoques
c		

Grec — Greek	Hongrois — Hungarian	Islandais — Icelandic	N°
			81
Μηνιγγιτις απλῆ Meniggitis aplè	Agyvelőburogyulladas (nem járvany) Agyhárlyagyulladas (nem járvany)		*a*
			b
	Egyiptomi szembaj Trachoma Egyiptomi szembeteség		**88***(a)*
Νοσοι τοῦ ὠτος και λοιμυτιδες τοῦ μαστοειδοῦς Nosoi tou otos kai lomytides tou mastoeidous			**89**
			a
Ωτιτις Otitis	Füllob Fülfolyás Fülgyulladás		*a(a)*
			b
Μαστοειδιτις Mastoeiditis			*b(a)*
	Heveny szivbelhártyagyulladás		**91**
Ενδοκαρδῖτις Endokarditis	Heveny bacillusos szivbelhártyagyulladás		*a*
			b
			c

N°	Italien — Italian	Letton — Latvian
81		
a	Meningite semplice	
b	Meningite infettiva s.a.i. Meningite purulenta s.a.i.	
88*(a)*	Tracoma Congiuntivite folliculare Congiuntivite tracomatosa	Trachoms
89		
a		
a(a)	Otite	
b		
b(a)	Mastoidite	
91		
a	Endocardite acuta (bacillare) Endocardite lenta	Akutais sirds iekšejas plēves iekaisums (nereimātiskais)
b		
c		

Lithuanien — Lithuanian	Néerlandais — Dutch	N°
		81
	Meningitis simplex	*a*
	Hersenvliesontsteking z.n.a. Hersenontsteking (v) z.n.a.	*b*
Trachoma	Trachoom	**88***(a)*
		89
		a
	Otitis Oorontsteking Middenoorontsteking	*a(a)*
		b
	Mastoiditis	*b(a)*
	Endocarditis acuta	**91**
	Septische endocarditis	*a*
		b
		c

N°	Polonais — Polish	Portugais — Portuguese
81	Zapalenie opon mózgowych zwykłe	Meningite não meningocócica
a		Meningite simples
b		
88*(a)*	Jaglica	Trachoma Tracoma
89		
a		
a(a)	Zapalenie ucha środkowego	Otite
b	Zapalenie wyrostka sutkowego	
b(a)		Mastoidite
91	Zapalenie wsierdzia ostre	Endocardite aguda (não-reumatismal)
a	Ostre bakt. zapalenie wsierdzia	Endocardite bacteriana aguda
b	Podostre zapalenie wsierdzia Wywołane gronkowcem zieleniejącym	Endocardite streptococica Endocardite bacteriana subaguda
c	Zapalenie wsierdzia ostre	

Roumain — Roumanian	Russe — Russian	N°
Meningită nemeningococică	Воспаление мозговых оболочек — Vospalenie mozgovykh obolotchek	81
Meningită simplă	Менингит простой — Meningit prostoï Не-менингококковый менингит — Nemeningokokkovyï meningit	*a*
Meningită cerebro spinală acută nemeningococică	Острый не-менингококковый менингит — Ostryï ne-meningokokkovyï meningit	*b*
Trahom Conjunctivita granuloasă	Трахома — Trakhoma	88*(a)*
		89
		a
Otita	Отит — Otit Воспаление уха — Vospalenie ukha	*a(a)*
Bolile sinusului mastoidian Mastoidită Sinusită mastoidiană	Костоеда сосцевидного отростка височной кости — Kostoeda sostsevidnogo otroska visotchnoï kosti	*b* *b(a)*
Endocardită acută nereumatismală	Острый эндокардит (не-ревматический эндокардит) — Ostryï endokardit (nerevmatitcheskiï endokardit)	91
Endocardită bacteriană acută	Острое воспаление эндокардия — Ostroïe vospalenie endocardia	*a*
Endocardită bacteriană subacută cu *S. viridans*	Эндокардит бактерийный — Endokardit bakteriïnyï	*b*
		c

N°	Serbo-croate — Serbo-Croatian	Suédois — Swedish
81		
a		Hjärnhinneinflammation
b		
88*(a)*	Трахома Trahoma	Smittsam ögonsjukdom Trachom
89		
a		
a(a)	Запаљење средњега ува Zapaljenje srednjega uva	Mellanöreinflammation
b		
b(a)		
91		
a		Akut. bakterien hjärtvalveinflammation
b		*Str. viridans* subakut hjärtvalveinflammation
c	Бациларни ендокардит Bacilarni endokardit	

Tchèque — Czech	Turc — Turkish	N°
		81
Prostý zánět plen mozkových		*a*
		b
Trachom	Trahom	**88***(a)*
		89
		a
Zánět ušní Záněty stredoušní	Otit	*a(a)*
		b
	Mastoidit	*b(a)*
		91
Prudký bacilarní zánět nitroblány srdeční	Ăndokardit	*a*
		b
		c

N°	Nomenclature internationale International Nomenclature	Latin
104	**Maladies des fosses nasales et annexes**	
a	Maladies des fosses nasales	
a(a)	*Coryza*	Rhinitis Coryza
b	Autres, y compris sinusites (spécifier le siège)	
b(a)	*Sinusite*	Sinusitis
105	**Maladies du larynx**	
(a)	*Laryngite*	Laryngitis catarrhalis Laryngotracheitis
106	**Bronchite**	Bronchitis Tracheitis Tracheobronchitis
a	Aiguë	Bronchitis acuta Bronchitis catarrhalis
b	Chronique	Bronchitis chronica Bronchiectasis Bronchitis purulenta
c	Non spécifiée	Bronchitis n.s.
107	**Bronchopneumonie**	Pneumonia catarrhalis Bronchopneumonia Bronchitis capillaris

Allemand — German	Anglais — English	N°
		104
		a
Coryza Schnupfen Rhinitis Gravedo	Coryza Nasal catarrh Rhinitis Cold in the head (v) Cold u. (v)	*a(a)*
		b
Sinusitis Nebenhöhlenentzündung	Sinusitis Abscess of... sinus	*b(a)*
		105
Laryngitis Kehlkopfentzündung	Laryngitis Laryngo-tracheitis	*(a)*
Luftröhrenentzündung	Bronchitis	**106**
Akute Bronchitis Frische Luftröhrenentzündung	Acute tracheitis Acute bronchitis Acute bronchial catarrh	*a*
Chronische Bronchitis	Chronic bronchitis Bronchiectasis Purulent bronchitis	*b*
Bronchitis Bronchialkatarrh Luftröhrenentzündung	Bronchitis u. Bronchial catarrh u.	*c*
Bronchopneumonie (lobuläre) Katarrhalische Lungenentzündung	Broncho-pneumonia Capillary pneumonia Lobular pneumonia Secondary pneumonia*	**107**

N°	Bulgare — Bulgarian	Danois et norvégien Danish and Norwegian
104		
a		
a(a)		Snue
b		
b(a)		Sinusit
105		
(a)		Akutt Strupekatarrh
106	Бронхитъ Bronkhit	Bronkitt
a	Остеръ бронхитъ Oster bronkhit	Akutte Katarrer i Åndedrettsorganene Akutt Bronkitt Akut Brystkatarh Akut Bronchitis Akut Luftrörskatarr
b	Хрониченъ бронхитъ Khronitchen bronkhit	Kronisk Bronchitis
c		Bronkitt Bronchitis Bronkit
107		Bronkpnevmonie Katarrhalsk Lungebetændelse Kapillær Bronkitt

Espagnol — Spanish	Estonien — Estonian	N°
		104
		a
Coriza Rinitis Romadizo (v) Catarro* s.a.i. Resfriado* Constipado (v)		*a(a)*
		b
Sinusitis Absceso del seno...	Sinus põletik	*b(a)*
		105
Laringitis Laringotraqueitis	Kõri põletik	*(a)*
Bronquitis	Kopsutoru põletik Bronhiit	**106**
Bronquitis aguda Bronquitis catarral aguda Traqueobronquitis Traqueitis s.a.i.	Äge kopsutoru põletik	*a*
Bronquitis cronica Bronquectasia	Krooniline kopsutoru põletik	*b*
		c
Bronconeumonia Bronquitis capilar Pneumonia lobular	Bronho-pneumonia Kapillaarne bronhiit	**107**

N°	Finnois — Finnish	Français — French
104		
a		
a(a)		Coryza Rhume de cerveau (v) Rhume s.a.i. (v)
b		
b(a)		Sinusite Abcès du sinus...
105		
(a)	Akillinen kurkunpääntulehdus	Laryngite Faux croup Laryngo-trachéite
106	Henkitorventulehdus	Bronchite
a	Akillinen henkitorventulehdus	Bronchite aiguë Trachéobronchite aiguë
b		Bronchite chronique Bronchiectasie Dilatation des bronches
c	Henkitorventulehdus	Trachéo-bronchite s.a.i. Catarrhe s.a.i. Trachéite s.a.i.
107		Bronchopneumonie Bronchite capillaire Pneumonie secondaire*

Grec — Greek	Hongrois — Hungarian	Islandais — Icelandic	N°
	Az orr és melléküregeinek betegségei		**104**
Ρινιτις Rhinitis			*a* *a(a)*
			b
			b(a)
	Gégebetegségek		**105**
Λαρυγγιτις Laryggitis			*(a)*
Βρογχῖτις Bronchitis	Hörghurut Légcsőhurut Légcsőgyulladás		**106**
Βρογχιτις οξεια Bronchitis oxeia	Heveny hörghurut	Kvefsótt	*a*
Βρογχιτις χρονια Bronchitis kronia	Idult hörghurut		*b*
	Hörggyulladás		*c*
Βρονγχοπνευμονια Bronchopneumonia Τριχοειδὴς βρογχῖτις Trichoeides bronchitis	Tüdőhurut Hurutos tüdőgyulladás Hajszalhörgök gyulladás	Kveflungnabolga	**107**

N°	Italien — Italian	Letton — Latvian
104		
a		
a(a)	Corizza Rafreddore di testa (v) Infreddatura (v)	
b		
b(a)	Sinusite Ascesso del seno...	
105		
(a)	Laringite Laringo-tracheite	
106	Bronchite	Bronchitu iekaisums
a	Bronchite acuta Bronchite catarrale acuta Tracheo-bronchite	Akutais bronchitu iekaisums
b	Bronchite cronica Bronchiettasia	Kroniskais bronchitu iekaisums
c	Bronchite s.a.i.	Bronchitu iekaisums
107	Bronco-polmonite (acuta) Bronchite capillare	Katarāls plaušu iekaisums Kapilāro bronchitu iekaisums

Lithuanien — Lithuanian	Néerlandais — Dutch	N°
		104
		a
	Hoofdverkoudheid Verkoudheid in't hoofd	*a(a)*
		b
	Voorhoofdsholteontsteking Sinusitis	*b(a)*
	Ontsteking van 't strottenhoofd	**105**
	Keelontsteking Laryngitis	*(a)*
Bronchitas	Bronchitis	**106**
	Acute bronchitis Verkoudheid (v) Acute catarrh der luchtwegen	*a*
	Chronische bronchitis	*b*
Bronchitas	Bronchitis z.n.a.	*c*
	Bronchopneumonie Capillaire bronchitis	**107**

N°	Polonais — Polish	Portugais — Portuguese
104		Molestias das fossas nasais e anexos
a		
a(a)	Katar nosa	Catarrheira (v) Defluxo (v)
b		
b(a)	Zapalenie zatok czołowych Zapalenie zatok szczękowych	Abcesso do seno... Sinusite
105		
(a)	Zapalenie krtani (nieżyt)	Laryngite
106		Bronchite Bronquite
a	Ostry nieżyt oskrzeli	Bronchite aguda Bronquite aguda
b	Przewlekły nieżyt oskrzeli	Bronchite chronica Bronquite chronica
c	Zapalenie oskrzeli	Bronchite
107	Ogniskowe Odoskrzelowe Zapalenie płuc	Bronchopneumonia Bronco-pneumonia Bronquite capilar

Roumain — Roumanian	Russe — Russian	N°
		104
		a
Coriza Guturai	Корыза — Coryza Насморк — Nasmork (v)	*a(a)*
		b
Sinusită	Воспаление лобной пазухи — Vospalenie lobnoï pazukhi Синузит — Sinusit	*b(a)*
		105
Laringită	Ларингит — Laryngit	*(a)*
Bronşită Bronşite	Бронхит — Bronkhit Воспаление дыхательного горла — Vospalenie dykhatelnogo gorla	**106**
Bronşita acută	Острый бронхит — Ostryï bronkhit Трахео-бронхит — Trakheo-bronkhit	*a*
Bronşita cronica Dilatatia bronchiilor	Хронический бронхит — Khronitcheskiï bronkhit Разширение бронхов — Razchirenie bronkhov	*b*
Bronşită	Бронхит — Bronkhit	*c*
Bronho-pneumonie Bronşita capilară	Бронхопневмония — Bronkhopnevmonia Бронхит капилярный — Bronkhit capilliarnyï	**107**

N°	Serbo-croate — Serbo-Croatian	Suédois — Swedish
104		
a		
a(a)	Кијавица Kijavica (Kijavitsa) Хуњавица Hunjavica (Hunjavitsa)	Snuva
b		
b(a)		
105		
(a)		Akut strupinflammation
106	Бронхитис Bronhitis	Luftrörsinflammation
a	Акутни бронхитис Akutni bronhitis	Akut luftrörsinflammation
b	Кронични бронхитис Kronični bronhitis	Kronisk luftrörsinflammation
c	Бронхитис Bronhitis	Luftrörsinflammation
107	Бронхично запаљење плућа Bronhično zapaljenje pluća (Bronhichno zapaljenje plucha)	Bronkopneumoni Kapillärbronkit Akut lunginflammation

Tchèque — Czech	Turc — Turkish	N°
		104
		a
Rýma	Nezle	*a(a)*
		b
Zánět dutiny... celní	Sinüzit	*b(a)*
Nemoci hrtanu		**105**
Zánět hrtanu	Larenjit	*(a)*
Katarrh průduškový	Bronşit	**106**
Zánět průdušek prudký		*a*
Zánět průdušek vleklý		*b*
Zánět průdušek nerozlišený		*c*
Lalokový zánět plic Lalůčkový zánět plic Zánět průdušinek Katarralní zánět plic	Bronkopnömoni	**107**

N°	Nomenclature internationale International Nomenclature	Latin
108	**Pneumonie lobaire** (pneumococcique)	Pneumonia crouposa Pneumonia fibrinosa Pneumonia duplex Pneumonia migrans Pneumonia bilateralis Pneumonia lobaris Pneumonia pneumococcica
109	**Pneumonie non spécifiée**	Pneumonia n.s.
110	**Pleurésie** non spécifiée comme tuberculeuse	Pleuritis n.s.
a	Empyème	Pleuritis purulenta Empyema pleurae
b	Autres formes de pleurésie et pleurésie s.a.i.	Hydrothorax Haemothorax Pleuritis exsudativa
115	**Maladies de la cavité buccale, de ses annexes, du pharynx et des amygdales,** y compris végétations adénoïdes	
a	Affections des dents et des gencives	
a(a)	*Stomatite*	Stomatitis
b	Angine septique, streptococcique	Angina septica Angina streptococcica
c	Autres affections des amygdales et du pharynx	
c(a)	*Angine de Ludwig*	Angina Ludovici Angina maligna

Allemand — German	Anglais — English	N°
Kruppöse Lungenentzündung Echte Lungenentzündung Lobäre Lungenentzündung Pneumokokken Lungenentzündung Eigentliche Lungenentzündung	Lobar pneumonia Pneumococcal pneumonia Croupous pneumonia Primary pneumonia Bilateral pneumonia	**108**
Pneumonie Lungenentzündung	Pneumonia Acute pulmonary congestion Septic pneumonia	**109**
Pleuritis	Pleurisy	**110**
Eiterbrust Empyema	Empyema Purulent pleurisy	*a*
Brustfellentzündung (v) Rippenfellentzündung (v)	Serofibrinous pleurisy Pleural effusion	*b*
		115
		a
Mundentzündung Stomatitis Mundschleimhautentzündung	Stomatitis	*a(a)*
Streptokokken Angina	Septic sore throat Septic angina	*b*
		c
Angina maligna Ludwigsche Halsentzündung Bösartige Halsentzündung	Ludwig's disease	*c(a)*

N°	Bulgare — Bulgarian	Danois et norvégien Danish and Norwegian
108		Krupös Lungebetændelse Lobær Lungebetændelse
109	Пневмония Pneumonia	Lungebetændelse
110		Lungehindbetændelse Pleuritt
a		Empyem
b		Pleuritt
115		
a		
a(a)		Mundbetændelse
b		Septisk Halsbetændelse Streptokokangina
c		
c(a)		

Espagnol — Spanish	Estonien — Estonian	N°
Pneumonia lobar Pneumonia crupal Pneumonia fibrinosa Pleuropneumonia Pneumonia pneumocócica	Lobaraline kopsupõletik	**108**
Pneumonia no especificada Fluxion de pecho (v)	(Eristlemata) Kopsupõletik	**109**
Pleuresia	Kopsukelme põletik Pleuriit	**110**
Empiema Piotorax Pleuresia purulenta		*a*
Derrame toráxico Pleuritis s.o.i.		*b*
		115
		a
Stomatitis	Suuõõne põletik	*a(a)*
Angina de estreptococos		*b*
		c
Angina de Ludwig	Angina Ludowici	*c(a)*

N°	Finnois — Finnish	Français — French
108	Keuhkokuume	Pneumonie lobaire Pneumonie franche Pneumonie double Pneumonie primitive Pleuropneumonie
109		Fluxion de poitrine (v) Pneumonie s.a.i. Congestion pulmonaire aiguë
110	Äkillinen keuhkopussintulehdus	Pleurésie
a		Empyème Pleurésie purulente
b	Keuhkopussintulehdus	Pleurite Pleurésie s.a.i.
115		
a		
a(a)		Stomatite
b		Angine septique Angine streptococcique
c		
c(a)		Angine de Ludwig Amygdalite gangréneuse

Grec — Greek	Hongrois — Hungarian	Islandais — Icelandic	N°
Πνευμονια κατὰ λοβους Pneumonia kata lobous	Lebenyes vagy rostonyás tüdőgyulladás	Taksótt	**108**
Πνευμονία Pneumonia	Tüdőgyulladas		**109**
Πλευρῖτις Pleuritis	Mellhártyagyulladás	Hingsótt	**110**
			a
Πλευρῖτις Pleuritis	Mellhártyagyulladás	Hingsótt	*b*
	A szájüreg, a garat és a mandulák betegsegei		**115**
			a
Στοματιτις Stomatitis	Szájgyulladas	Munnangur	*a(a)*
			b
			c
	Farkastorok		*c(a)*

Nº	Italien — Italian	Letton — Latvian
108	Polmonite lobare Polmonite fibrinosa Polmonite crupale Splenopolmonite	Lobārā pneumonija
109	Polmonite s.a.i.	Pneumonija Plaušu iekaisums
110	Pleurite	Pleurits
a	Empiema Piotorace Pleurite purulenta	
b	Pleurite s.a.i.	Pleurits
115		
a		
a(a)	Stomatite	
b	Angina settica Angina streptococcica	
c		
c(a)	Angina di Ludwig	

Lithuanien — Lithuanian	Néerlandais — Dutch	N°
	Croupeuse pneumonie Lobaire pneumonie Massieve pneumonie Pleuropneumonie	**108**
Plaučiu uždegimas	Pneumonie z.n.a. Longontsteking	**109**
	Borstvliesontsteking	**110**
	Empyeem	*a*
	Droge pleuris Natte pleuris Pleuris Pleuritis	*b*
		115
		a
	Mondontsteking Stomatitis	*a(a)*
		b
		c
	Necrotische angina	*c(a)*

N°	Polonais — Polish	Portugais — Portuguese
108	Płatowe zapalenie płuc	Pneumonia lobar Pneumonia pneumocócica
109	Zapalenie płuc	Pneumonia sem epitheto
110	Zapalenie opłucnej	Pleuriz Pleurite Pleurizia
a	Ropniak opłucnej Ropne zapalenie opłucnej	Pleurite purulenta
b		Empyema Empiema
115		Molestas da cavidade bucal, dos seus anexos, do faring e das amigdalas
a		
a(a)	Zapalenie dziąseł Zapalenie jamy ustnej	
b	Bakteryjne zapalenie migdałków	Angine septica Angina estreptococica
c		
c(a)	Angina Ludovici	Angina de Ludwig

Roumain — Roumanian	Russe — Russian	N°
Pneumonie lobară Pneumonie pneumococică	Пневмония крупозная — Pnevmonia kruposznaïa Пневмония лобарная — Pnevmonia lobarnaïa Пневмония — Pnevmonia Пневмококковая пневмония — Pnevmokokkovaïa pnevmonia Воспаление легких — Vospalenie legkikh (v)	**108**
Pneumonie (nespecificata) Congestia pulmonară acută	Пневмония (без обозначения) — Pnevmonia (bez oboznatchenia)	**109**
Pleurezie	Плеврит — Plevrit	**110**
Empiem Pleurezie purulentă	Воспаление плевры гнойное — Vospalenie plevry gnoïnoe	*a*
Pleurezie	Воспаление плевры эксудативное — Vospalenie plevry eksudativnoe Плеврит б. у. — Plevrit b.u.	*b*
Bolile cavităţii bucale, ale anexelor, faringelui şi ale amigdalelor		**115**
		a
Stomatită	Стоматит — Stomatit Воспаление рта — Vospalenie rta	*a(a)*
Angină streptococică	Септическая ангина — Septitcheskaïa angina Стрептококковая ангина — Streptokokkovaïa angina	*b*
		c
Angină lui Ludwig	Ангина Людовика — Angina Liudovika Жаба Людовика — Jaba Liudovika	*c(a)*

N°	Serbo-croate — Serbo-Croatian	Suédois — Swedish
108		Akut lobär lunginflammation Krupös lunginflammation
109	Запаљење плућа Zapaljenje pluća (Zapaljenje plucha)	Lunginflammation
110	Запаљење плућне маранице Zapaljenje plućne maramice (Zapaljenie pluchne maramitse)	Lungsacksinflammation
a	Гнојно запаљење плућне марамице Gnojno zapaljenje plućne maramice	
b	Запаљење плућне марамице Zapaljenje plućne maramice	Lungsacksinflammation
115		
a		
a(a)		Muninflammation
b	Септична ангина Septična angina (Septichna angina)	Septisk halsfluss
c		
c(a)		

Tchèque — Czech	Turc — Turkish	N°
Kruposní zánět plic		**108**
Zánět plic nerozlišený	Zatürree	**109**
Zánět pohrudnice	Zatuccem	**110**
Zánět pohrudnice hnisavý		*a*
Zánět pohrudnice suchý	Zatuccem	*b*
Nemoci ústni dutiny a jejich adnex		**115**
		a
Zánět úst		*a(a)*
		b
Choroby hltanu anebo mandlí		*c*
		c(a)

N°	Nomenclature internationale International Nomenclature	Latin
115*c(b)*	*Autres angines*	Angina faucium Amygdalitis Angina acuta Angina catarrhalis
d	Localisations autres, et non spécifiées	
119	**Diarrhée et entérite (au-dessous de 2 ans)**	Enteritis Diarrhoea Dyspepsia infantum Gastroenteritis acuta infectiosa Catarrhus gastroenteritis Catarrhus intestinalis Cholera infantum
120*a*	**Diarrhée, entérite (‡) (2 ans et plus)**	Cholera nostras Enterocolitis Cholerina Enteritis catarrhalis
121	**Appendicite**	Appendicitis Appendicitis perforativa Typhlitis Perityphlitis
127(‡)	*(a) Ictère catarrhal*	Icterus catarrhalis
147	**Infection puerpérale**	Sepsis puerperalis Septicemia puerperalis
151	**Furoncle**	Furunculus Furunculosis Furunculi conglomerati
152	**Phlegmon, abcès chaud, cellulite**	Abscessus acutus Phlegmone Abscessus calidus

Allemand — German	Anglais — English	N°
Halsentzündung Angina faucium Mandelentzündung	Quinsy Tonsillitis Naso-pharyngitis Sore throat (v)	115*c*(*b*)
		d
Darmkatarrh Brechdurchfall des Kindes (v) Sommerdurchfall	Diarrhœa, Enteritis Infantile diarrhœa Intestinal catarrh Dyspepsia*, Colitis Cholera infantum Summer diarrhœa (v), Epidemic diarrhœa, Gastroenteritis Green diarrhœa (v) Epidemic gastro-enteritis Zymotic diarrhœa	119
Einheimischer Brechdurchfall (v) Durchfall		120*a*
Blinddarmentzündung Appendizitis	Appendicitis Perityphlitis Typhlitis	121
Katarrhalische Gelbsucht Gelbsucht o.n.B.	Catarrhal jaundice	127
Kindbettfieber	Puerperal sepsis Puerperal septicæmia	147
Furunkel Karbunkel	Carbuncle Boil Furuncle Furunculosis	151
Phlegmone Akuter Abscess Heisser Abscess Zellgewebeentzündung	Cellulitis Acute abscess Phlegmon	152

N°	Bulgare — Bulgarian	Danois et norvégien Danish and Norwegian
115*c*(*b*)		Halsbetændelse Katarrhalsk Angina Follikulær Angina Almindelig Halsbetændelse Mandelbetændelse
d		
119	Диария и ентеритъ Diaria i enterit	Kolerine Akutt infeksjös Gastroenteritt Mavetarmkatarrh Akut Tarmkatar Akutt Diarré
120*a*	Ентеритъ Enterit	Gastroenteritt
121	Апендичитъ Apendicit	Blindtarmsbetændelse
127		Katarrhalsk Gulsot Katarrhalsk Icterus
147	Родилна треска Rodilna treska	Barselfeber
151		Kong Furunkel Blödbyld
152		Absces Bindevævsbetændelse Phlegmone

Espagnol — Spanish	Estonien — Estonian	N°
Amygdalitis s.o.i. Tonsilitis s.o.i. Esquinancia Angina s.o.i.	Igasugused angiinad	**115***c(b)*
		d
Diarrea, Enteritis Cólera de los niños Gastro-enteritis infantil Colera infantil Catarro intestinal Dispepsia de los niños	Kõhulahtisus Soolepõletik	**119**
Cólera nostras Colerina Lientería Enteritis coleriforme	Kõhulahtisus	**120***a*
Apendicitis Inflamación del ciego Tiflitis Peritiflitis	Ussjätkepõletik Appenditsiit	**121**
Ictericia catarral		**127**
Septicemia puerperal Fiebre de leche	Lapsevoodi mädaveresus Lapsevoodi roiskveresus	**147**
Furunculo	Furunkuloos Koeranael-tõbi	**151**
Flegmon Absceso caliente Tumor flegmonoso	Flegmoon Soe mädanik	**152**

N°	Finnois — Finnish	Français — French
115*c*(*b*)	Kitarisatulehdus	Angine s.a.i. Mal de gorge (v) Esquinancie (v) Cellulite du cou
d		
119	Epämääräinen kuume	Diarrhée Entérite des nourrissons Gastro-entérite des nourrissons Choléra infantile
120*a*	Kotimainen kolera Tarttuva mahatulehdus Tarttuva suolitulehdus	Diarrhée s.a.i. Entérite catarrhale Entérite cholériforme Cholérine, Choléra nostras Entérite muco-membraneuse Lientérie
121	Akillinen mahakatarri Akillinen sublikatarri	Appendicite Typhlite Péri-typhlite
127		Ictère catarrhal
147	Lapsivuodekuume	Septicémie puerpérale Fièvre puerpérale Infection puerpérale
151		Furonculose Anthrax (furoncles agglomérés)
152		Phlegmon Abcès chaud

Grec — Greek	Hongrois — Hungarian	Islandais — Icelandic	N°
Φαρυγγιτις, Pharyggitis Αμυγδαλιτις Amygdalitis	Mandulagyulladás Torokgyulladás	Kverkabólga	115*c*(*b*)
			d
Διαρροια Diarroia Εντερῖτις Enteritis	Hasmenés Bélgyulladás Bellob Bélhurut Gyermekek hasmenése	Idrakvef	119
Εντερῖτις Enteritis	Vastagbélhurut Vekonybélhurut		120*a*
Σκωληκοειδῖτις Skolekoeiditis	Féregnyúlvanygyulladás Vakbélgyulladás		121
	Sárgasag		127
Σηψαιμία Sepsaimia Λοιμώξεις ἐπιλόχειοι Loimokseis epilocheioi	Gyermekagyi vermergezés Gyermekagyi fertőzes Gyermekagyi láz	Barnfarasótt	147
Δοθιήν Dothièn	Keleveny		151
Φλεγμων Phlegmon Θερμον αποστημα Thermon apostema	Bőralatti kötőszövet lob Kelések Kötőszövetgyulladás Meleg talyog		152

N°	Italien — Italian	Letton — Latvian
115*c*(*b*)	Angina s.a.i. Tonsillite s.a.i.	
d		
119	Enterite Gastroenterite dei bambini Colera infantile Dispepsia (infantile)	
120*a*	Colerina Colera nostras Colera sporadico* Colite	
121	Appendicite Peritiflite	Aklās zarnas (piedēkla) iekaisums
127	Itterizia catarrale Itterocatarrale	
147	Setticemia puerperale Febbre puerperale Febbre da latte	
151	Foruncolo Foruncolosi	Furunkuloze Augoņi
152	Flemmone Ascesso caldo	

Lithuanien — Lithuanian	Néerlandais — Dutch	N°
	Angina Keelontsteking Katarrhale angina	115*c*(*b*)
		d
Viduriavimas Žarnu uždegimas	Diarrhœ Enteritis Acute gastroenteritis Dyspepsie Decompositie Diarrhee Buikloop	119
	Colitis Dikkedarmontsteking	120*a*
Apendicitas	Appendicitis Typhlitis Blindedarmontsteking	121
	Catarrhalische geelzucht Geelzucht z.n.a.	127
Gimdyv. septicemía	Kraamkoorts Septichæmie in het kraambed Kraamvrouwenkoorts	147
	Furunkel Furunculosis Steenpuist Negenoog	151
	Phlegmone Warm abscess Abscess z.n.a.	152

N°	Polonais — Polish	Portugais — Portuguese
115*c*(*b*)	Zapalenie gardła	Esquinencia Angina s.o.i.
d		
119	Nieżyt kiszek Zołądka Biegunka Nieżyt zołądka Jelit	Diarrhéa Diarreia Enterite Enterite choleriforme
120*a*	Zapalenie kiszek Cholera swojska Katar kiszek Zapalenie jelita grubego	Cholerina Lienteria Colite
121	Zapalenie wyrostka robaczkowego	Appendicite Typhlite Apendicite
127	Żółtaczka nieżytowa	Ictericia catarrhal
147	Gorączka połogowa Posocznica połogowa	Septicemia puerperal Infecção puerperal
151	Czyrak	Furunculo
152	Ropowica Flegmona Absces	Phlegmão Abcésso quente Flemão

Roumain — Roumanian	Russe — Russian	115*c*(*b*)-152 N°
	Жаба б. у. — Jaba b.u. (v)	115*c*(*b*)
Angină		*d*
Diaree Enterită	Диаррея — Diarreia Энтерит — Enterit Холероподобный энтерит — Kholeropodobnyï enterit Холера детская — Kholera detskaïa Понос — Ponos (v) Холерина — Kholerina	119
Diaree Enterită	Диаррея — Diarreia Энтерит — Enterit	120*a*
Apendicită	Аппендицит — Appendicit Воспаление слепой кишки — Vospalenie slepoï kichki	121
Icter cataral	Катарральная желтуха — Catarralnaïa jeltukha	127
Infecţie puerperală	Септицемия родильниц — Septicemia rodilnits Родовая горячка — Rodovaïa goriatchka	147
Furuncul	Фурункул — Furunkul Чирей — Tchireï (v) Веред — Vered (v)	151
Flegmon Absces cald	Флегмон — Flegmon Нарыв — Naryv Язвина — Iazvina (v)	152

№	Serbo-croate — Serbo-Croatian	Suédois — Swedish
115*c*(*b*)	Вратобоља Vratobolja Грлобоља Grlobolja	Akut inflammation i mandlarna i svalget Halsfluss
d		
119	Пролив Proliv Антеритис Anteritis	Barnkolera Inhemsk kolera
120*a*	Пролив Proliv Антеритис Anteritis	Infektiös magtarminflammation
121	Запаљење слепог црева Zapaljenje slepog creva (Zapaljenje slepog tsreva)	Blindtarmsinflammation
127	Жутица Žutica (Jutitsa)	Katarral gulsot
147	Грозница породиља Groznica porodilja (Grosnitsa porodilja)	Barnsängsfeber
151	Чир Čir (Chir)	Böld
152	Флегмон Flegmon	Abscess Bulnad

Tchèque — Czech	Turc — Turkish	N°
Bolení krku Angina	Anjin	115*c*(*b*)
		d
Hnačka Průjem Katar střevní Běhavka (v) Choleroidné zapálení střev Choleroid Zánět střev	Ishal	119
Cholerina Zánět střev		120*a*
Zánět červikovitého přívěsku	Apandicit	121
Žloutenka katarralní		127
Horečka šestinedělek Horečka omladnik	Hummayi nifasî	147
Nežitina Furunculosis	Köpek memesi (v)	151
Zánět podkožního vaziva Flegmona Teplá hlíza		152

N°	Nomenclature internationale International Nomenclature	Latin
153	**Autres maladies de la peau**	
(a)	*Infestation par poux*	Pediculatio Pediculi, Pedicularis morbus Pediculosis capitis, corporis, pubis Phtiriasis capitis, corporis, pubis
(b)	*Gale*	Scabies Acariosis
(c)	*Pemphigus (adultes)*	Pemphigus Pemphigus adultæ ætatis
154(‡)	**Ostéomyélite**	Osteomyelitis infectiosa Osteomyelitis acuta infectiosa
161(‡)(*a*)	*Pemphigus des nouveau-nés*	Pemphigus neonatorum

Allemand — German	Anglais — English	N°
		153
Läuse Verlausung	Louse infestation Pediculosis	*(a)*
Scabies Krätze (v)	Scabies Itch (v)	*(b)*
Pemphigus Blasenausschlag (v)	Pemphigus (not neonatorum)	*(c)*
Knochenmarkentzündung	Osteomyelitis Acute osteomyelitis Infective osteomyelitis Purulent osteomyelitis	**154**
Blasenausschlag der Neugeborenen	Pemphigus neonatorum	**161***(a)*

N°	Bulgare — Bulgarian	Danois et norvégien Danish and Norwegian
153		
(a)	Болести на кожата Bolesti na kojata	Lusesyke Lus Lusesyge
(b)		Skabb Fnat Scabies
(c)		Pemfigus hos Voksne Blæreudslet hos Voksne
154		Akutt Benmarvsbetændelse Osteomyelitt Benmarvsbetændelse
161(*a*)		Pemfigus hos Nyfödte Blæreudslet hos Nyfödte

Espagnol — Spanish	Estonien — Estonian	N°
		153
Piojos Ptiriasis	Phtiriasis	*(a)*
Sarna Roña (v) Acariosis	Scabies	*(b)*
Pénfigo	Pemphigus	*(c)*
Osteomielitis infecciosa aguda Osteitis infecciosa	Äge infektsioosne luuüdi-põletik	**154**
Pénfigo de los recién nacidos		**161**(*a*)

N°	Finnois — Finnish	Français — French
153		
(a)		Poux de tête, du corps, du pubis Pédiculose Phtiriase
(b)		Gale Acariose Sarcoptose
(c)		Pemphigus
154		Ostéomyélite Ostéite Périostite
161(*a*)		Pemphigus des nouveau-nés

Grec — Greek	Hongrois — Hungarian	Islandais — Icelandic	N°
			153
	Tetvesseg		*(a)*
	Rühkór	Kládi	*(b)*
	Bubor		*(c)*
Οστεομυκλιτις μολυσματικὴ οξεια Osteomyelitis molysmatike oxeia	Csontgyulladás Csontvelőgyulladás Csonthártyagyulladás		**154**
	Az ujszülottek bubor		**161**(*a*)

N°	Italien — Italian	Letton — Latvian
153		
(a)	Pidocchi Pediculosi	Utu slimiba Pedikuloze
(b)	Scabbia Rogna (v) Acariosi	
(c)	Pemfigo	
154	Osteomielite infettiva acuta Osteite infettiva	Kaulu smadzenu iekaisums
161(*a*)	Pemfigo dei neonati	

Lithuanien — Lithuanian	Néerlandais — Dutch	N°
	Hoofdluis	**153**
	Kleederluis	
	Platluis, Platje (vv)	
	Luizenziekte	*(a)*
	Pediculosis	
Niežai	Schurft (v)	*(b)*
	Scabies	
	Acariosis	
	Wormgaten	
	Pemphigus	*(c)*
	Osteomyelitis acuta infectiosa	**154**
	Beenontsteking	
	Beencaries	
	Beenmergontsteking	
	Pemphigus neonatorum	**161**(*a*)

N°	Polonais — Polish	Portugais — Portuguese
153		
(a)	Wszawica	Piolharia Piolhos Phthiriasis
(b)	Swierzb	Sarna
(c)	Pęcherzyca	Pemfigo
154	Zapalenie szpiku kostnego	Osteomiélite
161(*a*)	Pęcherzyca noworodków	Pemfigo dos recemnatos

Roumain — Roumanian	Russe — Russian	153-161(a) N°
		153
Păduchi Pediculoza	Вшивость (вошь) — Vchivost (voch)	(a)
Râie (v.) Scabie	Чесотка — Tchesotka	(b)
Pemfigusul	Пемфигус (взрослых) — Pemphigus (vzroslykh)	(c)
Osteomielită Periostită	Остеомиэлит — Osteomielit Воспаление костнаго мозга — Vospalenie kostnago mozga	154
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N°	Serbo-croate — Serbo-Croatian	Suédois — Swedish
153		
(*a*)	Вашљивост Vašljivost Уш = Ваш Uš (ush) = Vaš (vash) = *Pediculus*	Lus Lusig
(*b*)		Skabb
(*c*)		Papillomatös pemfigus
154	Акутно запаљење костне сржи Akutno zapaljenje kostne srži (Akutno zapalenje kostnę srji)	Beninflammation Akut beninflammation
161(*a*)		Pemfigus hos nyfödda

Tchèque — Czech	Turc — Turkish	153-161*(a)* N°
		153
Všivina	Bitlenme	*(a)*
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		(c)
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Troisième Partie

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Les numéros et lettres qui suivent les noms de maladies désignent les rubriques et sous-rubriques de la Nomenclature internationale détaillée de 1938, publiées dans la Deuxième Partie.

Part III

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The numbers and letters which follow the names of diseases indicate the headings and sub-headings of the detailed International List of 1938, given in Part II.

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B

B... en caractères cyrilliques, *cf.* **V...** ou **W...**
B... in Cyrillic characters, *cf.* **V...** or **W...**
б. о. = без обозначения / b. o. = bez oboznatchenia *rus.* = sans précision = without further indication = *sine indicio ultra*
б. у. = без указания / b. u. = bez ykazania *rus.* = sans précision = unqualified = *sine indicio*
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Bacillus dysenteriae Flexner [Infection à —] *fr.* 27*a*
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Builenpest *néer.* 3*a(a)*

Bujakor *hong.* 30

Bukhindetuberkulose *nor.* 15*(b)*

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Bue *bulg.* 38*b*

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C

C... en caractères cyrilliques, *cf.* S... ou Ts...

C... in Cyrillic characters, *cf.* S... or Ts...

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Cachexia *port.* 28*d*

Cachexia malariae *lat.* 28*d*

Cachexie palustre *fr.* 28*d*

Cachexie paralytique *fr.* 30*b*

Calentura intermitente (v) *esp.* 28*d*

Canapa (v) [Febbre da—] *it.* 38*f(b)*

Cancro molle *it. port.* 44*a(a)*

Чангир [Мекани —]
Čangir [Mekani —] *serb.* 44*a(a)*

Cangrena gassosa *it.* 24*c*

Căpuse (Afr. de Sud) [Febra prin inepături de —] *roum.* 39*b(cb)*

Caquexia *esp.* 28*d*

Carbón *esp.* 7*c*

Carbonchio *it.* 7*c*

Carbonchio ematico *it.* 7*b*

Carbuncle *angl.* 151

Carbunculo *port.* 7*c*

Carbunculo pestilencial *port.* 3*a(a)*

Carbunculus anthracis *lat.* 7*a*

Carbunculus malignus *all. lat.* 7*a*

Cărbune *roum.* 7*c*

Cărbune septicemico *roum.* 7*b*

Cărbune viscerale *roum.* 7*b*

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Caries [Vertebral —] *angl.* 16

Carrapatos [Febre recurrente dos —] *port.* 31*b*

Carreau (v) *fr.* 15*(b)*

Carrion [Enfermedad de —] *esp.* 29*(f)*

Carrion [Maladie de—] *fr.* 29*(f)*

Carrion [Morbo di —] *it.* 29*(f)*

Carrion's disease *angl.* 29*(f)*

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Castellani [Treponemosi di —] *it.* 32*b(c)*

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Catarrh der luchtwegen [Acute —] *néer.* 106*a*

Catarrh [Epidemic—] (v) *angl.* 33*b*

Catarrh [Febrile—] (v) *angl.* 33*b*

Catarrh [Intestinal—] *angl.* 119, 120*a*

Catarrh [Nasal —] *angl.* 104*a*

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Catarrhus apicis pulmonis *lat.* 13*b(c)*

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Catarrhus intestinalis *lat.* 119
Catarro* s.o.i. *esp.* 104*a(a)*
Catarro del oido *esp.* 89*a(a)*
Catarro intestinal *esp.* 119
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Cavernas pulmonares* *esp.* 13*b(c)*
Caverne polmonari* *it.* 13*b(c)*
Cavernes pulmonaires s.a.i. *fr.* 13*b(c)*
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Cellulite du cou *fr.* 115*c*
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Červienka *tch.* 27*d*
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Chagas [Doença de —] *port.* 29*(d)*
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Chagas Krankheit *all.* 29*(d)*
Chagas [Maladie de —] *fr.* 29*(d)*
Chagas [Malattia di —] *it.* 29*(d)*
Chagas [Ziekte van —] *néer.* 29*(d)*
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Chancre [Hard —] *angl.* 30*d*
Chancre induré *fr.* 30*d*
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Chancre [Soft —] *angl.* 44*a(a)*
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Chancro blando *esp.* 44*a(a)*
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Encephalitis lethargica acuta *lat.* 37*a*
Encephalitis (Sequelæ) *angl.* 37*b*
Encephalitis tuberculosa *lat. néer.* 14*b*
Encephalitt [Epidemisk —] *nor.* 37*c*
Endocardită bacteriană acută *roum.* 91*a*
Endocardită bacteriană subacută *roum.* 91*b*
Endocardită reumatismală acută *roum.* 58
Endocardite à *Streptococcus viridans* *fr.* 91*b*
Endocardite à streptocoques *fr.* 91*b*
Endocardite acuta (bacillare) *it.* 91*a*
Endocardite aguda (não-rheumatismal) *port.* 91*a*
Endocardite aiguë (non-rhumatismale) *fr.* 91*a*
Endocardite
bacteriana aguda *port.* 91*a*
bacteriana subaguda » 91*b*
Endocardite bactérienne aiguë *fr.* 91*a*
Endocardite bactérienne subaiguë *fr.* 91*b*
Endocardite gonococcique *fr.* 25*(a)*
Endocardite infectieuse aiguë *fr.* 91*a*
Endocardite infectieuse subaiguë *fr.* 91*b*
Endocardite lenta *it.* 91*b*
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Endocarditis [Acute bacterial —] *angl.* 91*a*
Endocarditis [Acute rheumatic —] *angl.* 58
Endocarditis aguda *esp.* 91*a*
Endocarditis [Akute —] (nicht rheumatische, bakterielle) *all.* 91*a*
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bacteriana acuta *lat.* 91*a*
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Endocarditis lenta *all. angl. lat.* 91*b*
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Endokarditt [Akutt bakterisk —] *nor.* 91*a*
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Enfermedad porádenica *esp.* 44*a(b)*
Ενκεφαλιτις επιδημικη / Enkephalitis epidemike *gr.* 37*c*
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Entamoebiasis *hong.* 27*b*
Enteric fever *angl.* 1

N.B. — Ce terme désigne fréquemment l'ensemble des fièvres typhoïdes *et* paratyphoïdes (c.-à-d. 1 & 2).
The term often covers *both* typhoid and paratyphoid fevers (i.e. 1 & 2).

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[Холероподобный —] } *rus.* 119
Enterit
[Kholeropodobnyï —]

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Enterit b.o.

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Entérite s.a.i. *fr. it.* 119, 120*a*
Entérite s.o.i. *port.* 119, 120*a*
Entérite catarrhale *fr.* 120*a*
Entérite cholériforme *fr.* 119, 120*a*
Enterite choleriforme *port.* 119, 120*a*
Entérite muco-membraneuse *fr.* 120*a*
Enterite tubercolare *it.* 15*(a)*
Entérite tuberculeuse *fr.* 15*(a)*
Enterite tuberculosa *port.* 15*(a)*
Enteritis *angl. esp. lat.* 119, 120*a*
Εντεριτις } *gr.* 119, 120*a*
Enteritis
Enteritis catarrhalis *lat.* 120*a*
Enteritis coleriforme *esp.* 120*a*
Enteritis tuberculosa *esp. lat.* 15*(a)*
Enteritis [Tuberculous —] *angl.* 15*(a)*
Enterobiasi *it.* 42*(f)*
Enterocolitis *lat.* 120*a*
Entsefaliit [Epideemiline —] *est.* 37*c*
Entsefaliit [Letargiline —] *est.* 37*c*
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Epaïltiva haava *fin.* 44*a(a)*
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Epid. ajukelme meningiit *est.* 6
Epid. seiles liauku uždegimas *lith.* 44*c*
Epid. smegenu pleves uždegimas *lith.* 6
Epideemiline entsefaliit *est.* 37*c*
Epidemiska(s) *lett.*
= *epidemicus, a, um*
Epidemiskais galvas un muguras smadzenu plēves iekaisums *lett.* 6
Epidemiskais pieausses dziedzera iekaisums *lett.* 44*c*
Epidemiskais smadzenu iekaisums *lett.* 37*c*
Епидемиске болести } *serb.* = *Morbi*
Epidemiske bolesti } *epidemici*
Епидемиченъ енцефалитъ } *bulg.*
Epidemitchen encephalit } 37*c*
Епидемични болести } *bulg.* = *Morbi*
Epidemitchni bolesti } *epidemici*

Epididimite caseosa *it.* 20
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Épididymite caséeuse *fr.* 20
Epididymitis gonorrhoica *lat.* 25*(a)*
Epididymitis tuberculosa *lat.* 20
Epididymitis [Tuberculous —] *angl.* 20
Erbsyphilis *all.* 30*d(a)*
Erisipela *esp. it.* 11
Erizipel *roum.* 11
Erntefieber *all.* 32*b(d)*
Erpete zoster *it.* 38*c*
Ertágulás *hong.* = *aneurysma* 30*c*
Erupción morbilosa *esp.* 35
Erupción rubeólica *esp.* 38*d*
Erüsiipel *est.* 11
Erysipel *all.* 11
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Ερυσιπελας } *gr.* 11
Erysipelas
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Ερύθρωσις } *gr.* 38*d*
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Escarlatina *esp. port.* 8
Escrofula* *esp. port.* 19
Espirochetose icterohemorrhagica *port.* 32*a*
Espiroquetose de Weil *port.* 32*a*
Espiroquetosis icterohemorragica *esp.* 32*a*
Espiroquetosis por *L. hebdomadis* *esp.* 32*b(c)*
Espiroquetosis por mordedura de rata *esp.* 32*b(b)*
Espundia *esp.* 29*(b)*
Esquentamento (v) *port.* 25*a*
Esquinancia* *esp.* 115*c(b)*
Esquinancie* *fr.* 115*c(b)*
Esquinencia* *port.* 115*c(b)*
Esthiomene *angl.* 44*a(b)*
Esthiomène *fr.* 44*a(b)*
Estivo-automnale [Fièvre —] *fr.* 28*c*
Estomatitis *esp.* 115*a(a)*
Εύλογία } *gr.* 34*a*
Eulogia

Εξανθηματικός τυφος / Exanthematikos typhos *gr.* 39*a*
Exanthématique [Fièvre —] *fr.* 39*b(ca)*
Exanthematisches Zeckenfieber von São Paulo *all.* 39*b(cd)*
Exanthematous fever *angl.* 39*b(ca)*

F

f.a.i. = fără altă indicaţié *(roum.)*
= sans autre indication
= unspecified
= *non specificata.*

Faaresyge *dan.* 44*c*
Fadenwurm *all.* 42*(e)*
Farciminium *all.* (obs.), *lat.* 26*a*
Farcin *esp. fr.* 26*a*
Farcino *it.* 26*a*
Farkastorok *hong.* 115*c(a)*
Farsótt(ir) *isl.* = *Morbus(i) contagiosus(i)*
Fascioliase *fr.* 42*(d)*
Faux croup *fr.* 105*(a)*
Favus *fr.* 43*c*
Febbre biliosa emoglobinurica *it.* 28*c(b)*
Febbre bottonosa *it.* 39*b(ca)*
Febbre da bacillo coli *it.* 24*d*
Febbre da canapa (v) *it.* 38*f(b)*
Febbre da latte (v)* *ital.* 147
Febbre da morso di topo *it.* 32*b(b)*
Febbre da papataci *it.* 38*f(b)*
Febbre dei cinque giorni *it.* 39*c(a)*
Febbre dei tre giorni *it.* 38*f(b)*
Febbre dei sette giorni *it.* 38*f(a)*
Febbre delle trincee *it.* 39*c(a)*
Febbre di Brill *it.* 39*a(b)*
Febbre di Malta *it.* 5*a*
Febbre di Marsiglia *it.* 39*b(ca)*
Febbre di Oroja *it.* 29*(f)*
Febbre di Pfeiffer *it.* 44*d(b)*
Febbre di tsutsugamuchi *it.* 39*b(b)*
Febbre emoglobinurica *it.* 28*c(b)*
Febbre estiva *it.** 38*f(b)*
Febbre fluviale del Giappone *it.* 39*b(b)*
Febbre ghiandolare *it.* 44*d(b)*
Febbre gialla *it.* 38*a*
Febbre maculata delle Montagne Rocciose *it.* 39*b(cc)*
Febbre malarica *it.* 28*d*
Febbre Mediterranea *it.* 5*a*
Febbre miliare *it.* 44*d(f)*
Febbre ondulante *it.* 5*a*
Febbre palustre *it.* 28*d*
Febbre puerperale *it.* 147
Febbre purpurea delle Montagne Rocciose *it.* 39*b(cc)*
Febbre « Q » *it.* 39*b(ce)*
Febbre quartana *it.* 28*b*
Febbre quintana *it.* 39*c(a)*
Febbre ricorrente *it.* 31*c*
Febbre ricorrente da pidocchio *it.* 31*a*
Febbre ricorrente da zecche *it.* 31*b*
Febbre ricorrente europea *it.* 31*a*
Febbre ricorrente spagnuola *it.* 31*b*
Febbre rossa *it.* 38*f(a)*
Febbre terzana *it.* 28*a*
Febbre tifoidea *it.* 1
Febbre (Tifo) delle Montagne Rocciose *it.* 39*b(cc)*
Febbre volinica *it.* 39*c(a)*
Febbri paratifiche *it.* 2
Febbri paratifoidi *it.* 2
Febră aftoază *roum.* 44*d(a)*
Febră bilioasa hemoglobinurică *roum.* 28*c(b)*
Febră butonoasă *roum.* 39*b(ca)*
Febră de cinci zite *roum.* 39*c(a)*
Febră de trei zite *roum.* 38*f(b)*
Febră fluviala japonă *roum.* 39*b(b)*
Febră galbenă *roum.* 38*a*
Febră ondulantă *roum.* 5*c*
Febră paratifoidă *roum.* 2
Febră prin intepături de căpuse (Afr. de Sud) *roum.* 39*b(cb)*
Febră purpurie munţilor stăncoşi *roum.* 39*b(cc)*
Febră « Q » (Queensland) *roum.* 39*b(ce)*
Febră recurent *roum.* 31*c*
Febră recurenta cu căpuşe *roum.* 31*b*
Febră recurentă epidemica cu păduchi *roum.* 31*a*

Fever [Gaol —] (v) *angl.* 39*a*
Fever [Glandular —] *angl.* 44*d(b)*
Fever [Hæmoglobinuric bilious —] *angl.* 28*c(b)*
Fever [Intermittent —] (v) *angl.* 28*d*
Fever [Japanese River —] *angl.* 39*b(b)*
Fever [Louse-borne typhus —] *angl.* 39*a*
Fever [Malta —] *angl.* 5*a*
Fever [Marsh —] *angl.* 28
Fever [Mediterranean —] *angl.* 5*a*
Fever [Miliary —] *angl.* 44*d(f)*
Fever [Mite —] *angl.* 39*b(b)*
Fever [Mossman —] *angl.* 39*b(ce)*
Fever [Mud —] *angl.* 32*b(d)*
Fever [Oroya —] *angl.* 29*(f)*
Fever [Pappataci —] *angl.* 38*f(b)*
Fever [Paratyphoid —] *angl.* 2
Fever [Phlebotomus —] *angl.* 38*f(b)*
Fever [Q —] *angl.* 39*b(ce)*
Fever [Quartan —] *angl.* 28*b*
Fever [Queensland —] *angl.* 39*b(ce)*
Fever [Quotidian —] *angl.* 28*c*
Fever [Rat-bite —] *angl.* 32*b(b)*
Fever [Relapsing —] *angl.* 31*c*
Fever [Remittent —] (v) *angl.* 28*d*
Fever [Rheumatic —] *angl.* 58
Fever [Rocky Mountain Spotted —] *angl.* 39*b(cc)*
Fever [Sandfly —] *angl.* 38*f(b)*
Fever [Scarlet —] *angl.* 8
Fever [South African Tick-bite —] *angl.* 39*b(cb)*
Fever [Tick —] [*Sp. duttoni*] *angl.* 31*b*
Fever [Trench —] *angl.* 39*c(a)*
Fever [Tropical malignant tertian —] *angl.* 28*c*
Fever [Typhoid —] *angl.* 1
Fever [Undulant —] *angl.* 5*c*
Fever [Yellow —] *angl.* 38*a*
Fiebre amarilla *esp.* 38*(a)*
Fiebre biliosa hemoglobinurica *esp.* 28*c(b)*
Fiebre botonosa *esp.* 39*b(ca)*
Fiebre cotidiana *esp.* 28*c*
Fiebre cuartana *esp.* 28*b*
Fiebre de cinco dias *esp.* 39*c(a)*
Fiebre de las trincheras *esp.* 39*c(a)*
Fiebre de leche (v) *esp.* 147
Fiebre de los flebótomos *esp.* 38*f(b)*
Fiebre de los marinos de Tolón *esp.* 39*b(a)*
Fiebre de los pappataci *esp.* 38*f(b)*
Fiebre de los tres dias *esp.* 38*f(b)*
Fiebre de Malta *esp.* 5*a*
Fiebre de Mejico *esp.* 39*b(a)*
Fiebre de Oroya *esp.* 29*(f)*
Fiebre de siete dias (del Japon) *esp.* 32*b(c)*
Fiebre dengue *esp.* 38*f(a)*
Fiebre enterica *esp.* 1
Fiebre escarlatinosa *esp.* 8
Fiebre estío-otoñale *esp.* 28*c*
Fiebre exantemática mediterranea (de Olmer) *esp.* 39*b(ca)*
Fiebre fluvial del Japón *esp.* 39*b(b)*
Fiebre hemoglobinurica *esp.* 28*c(b)*
Fiebre intermitente (v) *esp.* 28*d*
Fiebre maculosa de los Montañas Rocosas *esp.* 39*b(d)*
Fiebre maligna *esp.* 28*c*
Fiebre maregmatica* *esp.* 28*d*
Fiebre mediterranea *esp.* 5*a*
Fiebre miliar *esp.* 44*d(f)*
Fiebre mucosa* *esp.* 1
Fiebre ondulante *esp.* 5*c*
Fiebre otoñal del Japon *esp.* 32*b(c)*
Fiebre palustre *esp.* 28*d*
Fiebre perniciosa *esp.* 28*c*
Fiebre petequial *esp.* 39*a*
Fiebre « Q » *esp.* 39*b(ce)*
Fiebre recurrente s.o.i. *esp.* 31*c*
Fiebre recurrente africana *esp.* 31*b*
Fiebre recurrente de garrapatas *esp.* 31*b*
Fiebre recurrente de Obermeyer *esp.* 31*a*
Fiebre recurrente hispanoafricana *esp.* 31*b*
Fiebre sub-terciana *esp.* 28*c*
Fiebre terciana *esp.* 28*a*
Fiebre tifoidea *esp.* 1
Fiebre verrucosa del Guaitara *esp.* 29*(f)*
Fiebres paratifoideas *esp.* 2
Fièvre à pappataci *fr.* 38*f(b)*

Fièvre à phlébotomes *fr.* 38*f(b)*
Fièvre à piqûres de tiques de l'Afrique du Sud *fr.* 39*b (cb)*
Fièvre aphteuse *fr.* 44*d(a)*
Fièvre bilieuse hématurique *fr.* 28*c(b)*
Fièvre bilieuse hémoglobinurique *fr.* 28*c(b)*
Fièvre boutonneuse à *R. conori* *fr.* 39*b(ca)*
Fièvre de cinq jours *fr.* 39*e(a)*
Fièvre de Malte *fr.* 5*a*
Fièvre de Mandchourie *fr.* 39*b(b)*
Fièvre de morsure de rat *fr.* 32*b(b)*
Fièvre de sept jours japonaise *fr.* 32*b(e)*
Fièvre de Sumatra *fr.* 39*b(c)*
Fièvre de trois jours *fr.* 38*f(b)*
Fièvre de Wolhynie *fr.* 39*c(a)*
Fièvre des coupeurs de cannes (du Queensland) *fr.* 39*b(ce)*
Fièvre des lièvres *fr.* 26*b*
Fièvre des marais *fr.* 32*b(d)*
Fièvre des moissons *fr.* 32*b(d)*
Fièvre des tranchées *fr.* 39*c(a)*
Fièvre estivo-automnale *fr.* 28*c*
Fièvre exanthématique *fr.* 39*b(a)*
Fièvre fluviale du Japon (à *R. orientalis) fr.* 39*b(b)*
Fièvre glandulaire de Pfeiffer *fr.* 44*d(b)*
Fièvre intermittente *fr.* 28*d*
Fièvre jaune *fr.* 38*a*
Fièvre méditerranéenne *fr.* 5*a*
Fièvre miliaire* *fr.* 44*d(f)*
Fièvre muqueuse (v) *fr.* 1
Fièvre nautique de Toulon *fr.* 39*b(a)*
Fièvre ondulante *fr.* 5*c*
Fièvre ondulante bovine *fr.* 5*b*
Fièvre ondulante caprine *fr.* 5*b*
Fièvre d'Oroya *fr.* 29*(f)*
Fièvre palustre *fr.* 28*d*
Fièvre pernicieuse* *fr.* 28*d*
Fièvre pourprée de Minas Geraes *fr.* 39*b(cd)*
Fièvre pourprée des Montagnes Rocheuses *(R. Rickettsi) fr.* 39*b(cc)*
Fièvre pourprée de São Paulo *fr.* 39*b(cd)*
Fièvre puerpérale *fr.* 147
Fièvre récurrente *fr.* 31*c*
Fièvre récurrente africaine *fr.* 31*c*
Fièvre récurrente épidémique à poux *(Sp. obermeieri) fr.* 31*a*
Fièvre récurrente à *Ornithodorus* *fr.* 31*b*
Fièvre récurrente à tiques *fr.* 31*b*
Fièvre splénique *fr.* 7*b*
Fièvre tachetée des Montagnes Rocheuses *fr.* 39*b(cc)*
Fièvre typhoïde *fr.* 1
Fièvres paratyphoïdes *fr.* 2
Filaria *all.* 42*(e)*
Filaria sanguinis *lat.* 42*(e)*
Filariasis *all. angl. lat. néer.* 42*(e)*
Filariose *fr.* 42*(e)*
Filariosi *it.* 42*(e)*
Filariosis *esp.* 42*(e)*
Filarioză *roum.* 42*(e)*
Филариозы / Filariozy *rus.* 42*(e)*
Fimatosis* *esp.* 13*c*
Fjärde sjukdom *suéd.* 44*d(e)*
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Fläckfeber *suéd.* 39*a*
Fläcktyfus *suéd.* 39*a*
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Flea-borne typhus *angl.* 39*b(a)*
Fleckfieber *all.* 39*a*
Fleckfieber [Epidemisches —] *all.* 39*a*
Fleckfieber [Rocky-Mountain —] *all.* 39*b(cc)*
Flecktyphus *all.* 39*a*
Flecktyphus (durch Flöhe übertragener) *all.* 39*b(a)*
Flecktyphus [Mexikanischer —] *all.* 39*b(a)*
Flegmon *esp. roum.* 152
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Flekkfeber *nor.* 39*a*
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Flexner dysentery *angl.* 27*a*
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Fluviale du Japon [Fièvre —] *fr.* 39*b(b)*
Fluxion de pecho (v) *esp.* 109
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Fnat *dan.* 153*(b)*
Folie paralytique *fr.* 30*b*
Foot-and-mouth disease *angl.* 44*d(a)*
Foruncolo *it.* 151
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Frambesia *esp.* 32*b(c)*
Framboesia *all.* 32*b(c)*
Frambœsia *angl.* 32*b(c)*
Framboesia tropica *esp. lat. néer.* 32*b(c)*
Framboesia tropicale *it.* 32*b(c)*
Framboesie *all.* 32*b(c)*
Framboezia tropicál *port.* 32*b(c)*
Francis [Maladie de —] *fr.* 26*b*
Frengi *turc* 30
Frieseln *all.* 44*d(f)*
Friguri de baltă *roum.* 28*d*
Friguri palustre *roum.* 28*d*
Frossa *suéd.* = *febris intermittens* 28*d*
Ftizie (v) *roum.* 13*c*
Fülfolyás *hong.* 89*a(a)*
Fülgyulladás *hong.* 89*a(a)*
Füllob *hong.* 89*a(a)*
Fültőmirigylob *hong.* 44*c*
Fültőmirigylob [Járvany —] *hong.* 44*c*
Fünftagefieber *all.* 39*c(a)*
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Furunkuloos *est.* 151
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G

Gaasgangreen *est* 24*c*
Gadijumi *lett.*, Gadijums *lett.* } = *Casus*
Gafeira (v) *port.* 42*(h)*
Gălbinare epidemică *roum.* 44*d(g)*
Gale *fr.* 153*(b)*
Gallico (v) *port.* 30*d*
Galvas smadzenu iekaisums [Akutais —] *lett.* 36
Galvos smegenu uždegimas *lith.* 36
Gangraena aerogenes *lat.* 24*c*
Gangraena emphysematosa *lat.* 24*c*
Gangrena gaseosa *esp.* 24*c*
Гангрена [Гасна], Gangrena [Gasna —] } *serb.* 24*c*
Gangrenă gazoasă *roum.* 24*c*
Gangrena gazosa *port.* 24*c*
Гангрена (Газовая), Gangrena (Gazovaia) } *rus.* 24*c*
Gangrène gaseuse *fr.* 24*c*
Gaol fever (v) *angl.* 39*a*
Garais klepus *lett.* 9
Garrapatas [Fiebre recurrente de —] *esp.* 31*b*
Gas gangrene *angl.* 24*c*
Gasbrand *all. dan. nor. suéd.* 24*c*
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Гасна гангрена, Gasna gangrena } *serb.* 24*c*
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Gastro-entérite *fr.* 120*a*
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Gastro-enteritis [Acute —] *néer.* 119
Gastro-enteritis [Epidemic —] *angl.* 120*a*
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Gastroenteritt [Infektiös —] *nor.* 119, 120*a*
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Geelzucht [Epidemische —] *néer.* 44*d(g)*
Gegebetegségek *hong.* 105
Gehirnentzündung [Influenzische] *all.* 33*b*
Gehirnentzündung [Übertragbare —] *all.* 37*a*
Gehirnerweichung [Fortschreitende—] *all.* 30*b*
Gehirngrippe (v) *all.* 37*c*
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I

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Influensa pneumonie *d. & n.* 33*a*
Influensa uden lungekomplikationer *dan.* 33*b*
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Influenza med lungekomplikationer *d. & n.* 33*a*
Influenzische Broncho-pneumonie *all.* 33*a*
Influenzische Encephalitis *all.* 33*b*
Influenzische Gehirnentzündung *all.* 33*b*
Influenzische Lungenentzündung *all.* 33*a*
Influenzische Pneumonie *all.* 33*a*
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Intermittent fever (v) *angl.* 28*d*
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Ipoemia dei minatori (ant.) *it.* 40
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Ittero catarrale *it.* 127*(a)*
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Ittero infettivo *it.* 32*a*
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J

Жаба б. у. / Jaba (v) b.u. *rus.* 115*c(b)*
Жаба Людовика / Jaba Ludovica *rus.* 115*b*
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Жаба [Сифилитическая —] / Jaba [Sifilititcheskaïa —] *rus.* 30*d*
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Jelit [Niezit —] *pol.* = *Enteritis* 119, 120*a*
Желтая лихорадка / Jeltaia likhoradka *rus.* 38*a*
Желчно-геморрагический спирокетоз / Jeltchno-gemorragitcheski spirokhetoz *rus.* 32*a*
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Jungle (tropical) typhus *angl.* 39*b(b)*
Juodligé *lith.* 7*c*

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Kachexie malarická *tch.* 28*d*
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Κακοήθης φλύκταινα / Kakoedes phlyktaina *gr.* 7
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Kleederluis *néer.* = *Pediculus vestimenti* 153*(a)*
Klem *néer.* 12
Клещевый возвратный тиф / Kleschevyï vozvratnyï tif } *rus.* 31*b*
Klierkoorts *néer.* 44*d(b)*
Klierziekte *néer.* 19
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Knokkelkoorts [Tropische —] *néer.* 38*f(a)*
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Koeranael-tõbi *est.* 151
Kõhukelme tuberkuloos *est.* 15*(b)*
Kõhulahtisus *est.* = *Diarrhoea* 119, 120*a*
Kõhusoetõbi *est.* 1
Kõhutõbi *est.* 1
Kõhutõbi [Verine —] *est.* 27*d*
Kõhutüüfus *est.* 1
Κοιλιακὸς τυφος / Koiliakos typhos } *gr.* 1
Коккцидии / Kokktsidii } *rus.* 29*a*
Κοκκῦτις / Kokkytis } *gr.* 9
Коклюш / Koklioucн } *rus.* 9
Kokliušas *lith.* 9
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Koldfeber *d. & n.* 28*d*
Kolera *d. & n. hong. turc* 4
Колера / Kolera } *serb.* 4
Kolera [Aasialainen —] *fin.* 4
Kolera [Asiatisk —] *suéd.* 4
Kolera [Azsiai —] *hong.* 4
Kolera [Kotimainen —] *fin.* 120*a*
Kolerine *d. & n.* 119, 120*a*
Колибациллоз / Kolibacilloz } *rus.* 24*d*
Kollane palavik *est.* 38*a*
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Koolera *est.* 4
Koorts *néer.* = *Febris*
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Kopsutoru põletik *est.* 106*c*
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Корь / Kor } *rus.* 35
Korduv palavik *est.* 31*a*
Коременъ тифъ / Koremen tif } *bulg.* 1
Kõri põletik *est.* 105*(a)*
Körnerkrankheit *all.* 88*(a)*
Korvanäärmeis [Infektsioon —] *est.* 44*c*
Korvapoletik *est.* 89*a(a)*
Корыза / Koryza } *rus.* 104*a(a)*
Kossageit *isl.* = *Impetigo contagiosa* 153*(d)*
Костне сржи [Акутно запаљење —] / Kostne srži [Akutno zapaljenje —] } *serb.* 154
Костоеда сосцевидного отростка височной кости / Kostoeda sostsevidnogo otrostka visotchnoï kosti } *rus.* 89*b(a)*
Kotayama [Malattia di —] *it.* 42*(b)*
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Kőtőszővetgyulladás *hong.* = *Phlegmon* 152
Kraamkoorts *néer.* 147
Kraamvrouwenkoorts *néer.* 147
Krampji [Sastinguma —] *lett.* 12
Краснуха / Krasnukha } *rus.*

— dans la nomenclature officielle, désigne la « rubéole » *(rubella)* 38*d*
— vulgairement, désigne aussi la « scarlatine » *(scarlatina)* 8
— in the official nomenclature corresponds to "german measles" *(rubella)* 38*d*
— in common language means also "scarlet fever" *(scarlatina)* 8

Краснуха [(карлатинообразная] / Krasnukha [Skarlatinoobraznaia —) *rus.* 44*d(e)*
Krätze (v) *all.* 153*(b)*
Kraujo užkretimas *lith.* 24*a*
Кровохаркание туберкулезное / Krovokharkanie tuberkuloznoïe *rus.* 13*b*
Круп / Krup *rus.* 10
Kruposni zánět plic *tch.* 108
Krupp* *all.* 11
Krupp* [Ansteckender —] *all.* 10
Kruvinoji *lith.* 27*d*
Крысный тиф / Krysnyï tif *rus.* 39*b(b)*
Krztusiec *pol.* 9
Kuduz *turc* 38*b*
Куга / Kuga *serb.* 3*c*
» [Бубонска —] / » [Bubonska —] » 3*a(a)*
» [Плућна —] / » [Plućna —] » 3*b*
Kulhavka [Slintavka a —] (vet) *tch.* 44*d(a)*
Kulkutaudinluontoinen aivokalvontulehdus *fin.* 6
Kuolleita *fin.* = *Mortui*
Kuppa *fin.* 30*d*
Kuppa [Piileva —] *fin.* 30*d*
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Meningite infettiva s.a.i. *it.* 81*b*
Meningite meningococcica *it.* 6
Méningite miliaire *fr.* 14*a*
Meningite miliar *port.* 14*a*
Meningite miliare *it.* 14*a*
Méningite non méningococcique *fr.* 81*a*
Meningite pelo meningococo *port.* 6
Meningite purulenta s.a.i. *it.* 81*b*
Meningite semplice *it.* 81*a*
Méningite simple *fr.* 81*a*
Meningite simples *port.* 81*a*
Meningite tubercolare *it.* 14*a*
Meningite tuberculosa *port.* 14*a*
Meningitis bacilar *esp.* 14*a*
Meningitis cerebro-espinal epidemica *esp.* 6
Meningitis [Cerebro-spinal—] *angl.* 6
Meningitis [Epidemische —] *néer.* 6
Meningitis [Epidemische cerebro-spinal —] *all.* 6
Meningitis infecciosa *esp.* 81*a*
Meningitis [Meningococcal —] *angl.* 6
Meningitis meningococcica *lat.* 6
Meningitis meningococica *esp.* 6
Meningitis miliar *esp.* 14*a*
Meningitis miliaris *lat.* 14*a*

Meningitis no-meningococica *esp.* 81*a*
Meningitis (non-meningococcal) *angl.* 81*a*
Meningitis purulenta s.o.i. *esp.* 81*b*
Meningitis serosa porcinarii *lat.* 38*f*
Meningitis simple *esp.* 81*a*
Meningitis [Simple —] *angl.* 81*a*
Meningitis simplex *lat. néer.* 81*a*
Meningitis tuberculosa *all. esp. lat. néer.* 14*a*
Meningitis [Tuberculous —] *angl.* 14*a*
Meningitt [Tuberkulös —] *nor.* 14*a*
Méningococcémie *fr.* 6
Méningococcie *fr.* 6
Meningocócia *esp.* 6
Menschliche Eingeweidewürmer *all.* 42*(d)*
Mensenpest *néer.* 3*e*
Merevgörcs *hong.* 12
Merevgörcs [Fertőzeses —] *hong.* 12
Meris *lett.* 3*c*
Meslinger *nor.* 35
Mešlungis *lith.* 12
Mešlungis [Stabas —] *lith.* 12
Metritis gonorrhoica *lat.* 25*(a)*
Mexico-palavik *est.* 39*a*, 39*b(a)*
Мягкий шанкр / Miagkiĭ chankr } *rus.* 44*a(a)*
Miasi *it.* 42*(i)*
Micoses *port.* 43
Micosi *it.* 43
Micosis *esp.* 43
Micoze *roum.* 43
Middenoorontsteking *néer.* 89*a(a)*
Mieki [Wrzod —] *pol.* 44*a(a)*
Mighetto *it.* 43*(a)*
Mijtekoorts [Sumatraansche —] *néer.* 39*b(b)*
Микозы / Mikozy } *rus.* 43
Mikrosporie *all.* 43*(b)*
Mikstais Šanker *lett.* 44*a(a)*
Miliærtuberkulose *d. & n.* 22*a*
Miliaire* [Fièvre —] *fr.* 44*d(f)*
Милиарная лихорадка / Miliarnaia likhoradka } *rus.* 44*d(f)*
Miliartuberkulose *all.* 22*c*
Miliartuberkulose [Akute —] *all.* 22*a*
Miliartuberkulose [Chronische —] *all.* 22*b*
Miliary fever *angl.* 44*d(f)*
Miltbrand *d. & n.* 7*c*
Miltvuur *néer.* 7*c*
Miltvuur [Gastrointestinale —] *néer.* 7*b*
Miltvuur [Septische —] *néer.* 7*b*
Miltvuurpuiste *néer.* 7*a*
Milzbrand (v) *all.* 7*c*
Minas Geraes [Fièvre de —] *fr.* 39*b(cd)*
Minkštasis šankeris *lith.* 44*a(a)*
Miocardită reumatismală acută *roum.* 58
Miozită epidemică *roum.* 44*d(c)*
Mire, Mirimai, Mirusieji *lith.* = *Mortuus, i*
Mirigygümő *hong.* 19
Miruši *lett.* = *Mortui*
Mislingar *isl.* 35
Mite fever (Sumatra) *angl.* 39*b(b)*
Mitmesugused seenhaigused *est.* 43*(b)*
Mittelmeerfieber *all.* 5*a*
Mittelohrentzündung *all.* 89*a(a)*
Mjältbrand *suéd.* 7*c*
Moccio (v) *it.* 26*a*
Mocsárláz *hong.* 28*d*
Mocsárláz [Heveny —] *hong.* 28*d*
Mocsárláz senyv *hong.* = *Cachexia palustre* 28*d*
Moeraskoorts *néer.* 28*d*
Molestia *port.* = *Morbus*
Molestia de Buillaud *port.* 58
Molestia hidatica *port.* 41
Molestias infectuosas *port.* = *Morbi infectiosi*
Молочница / Molotchnitsa } *rus.* 43*a*
Mond en klauwzeer (vet) *néer.* 44*d(a)*
Mondontsteking *néer.* 115*a(a)*
Mondzeer *néer.* 44*d(a)*
Moniliase *fr.* 43*a*
Mononucléose infectieuse *fr.* 44*d(b)*
Mononucleosi infettiva *it.* 44*d(b)*
Mononucleosis [Infectious —] *angl.* 44*d(b)*

Mononucleosis infectiosa *all. lat.* 44*d(b)*
Mononucleoza infectiosa *roum.* 44*d(b)*
Mononukleoza zakaźna *pol.* 44*d(b)*
Montagnes Rocheuses [Fièvre tachetée ou pourprée des —] *fr.* 39*b(cc)*
Mor *pol. tch.* 3*c*
Mor [Plicní —] *tch.* 3*b*
Mor [Žlutý —] *tch.* 38*a*
Morbi gonorrhoici *lat. néer.* 25*(c)*
Морбили / Morbili } *serb.* 35
Morbilli *all. angl. lat.* 35
Morbillo *it.* 35
Morbus addisonii tuberculosus *lat. néer.* 21*a*
Morbus Bangii *lat.* 5*b*
Morbus Boringiæ *lat.* 44*d(c)*
Morbus Brillii *lat.* 39*a(b)*
Morbus coxae *angl. lat.* 17*b*
Morbus quartus *lat.* 44*d(e)*
Morbus Weilii *lat.* 32*a*
Морбус Веили / Morbus Weyli } *serb.* 32*a*
Mordedura de animaes raivoso *port.* 38*b*
Mormo *port.* 26*a*
Morová nákaza *tch.* 3*a(b)*
Morová rána *tch.* 3*a(a)*
Morpions (vv) *fr.* = *Pediculi pubis* 153*(a)*
Morsicature di animali rabbici o sospetti *it.* 38*b(b)*
Morsure d'animal enragé *fr.* 38*b(b)*
Morsure de rat [Fièvre par —] *fr.* 32*b(b)*
Morva *it. roum.* 26*a*
Morve *fr.* 26*a*
Mossman fever *angl.* 39*b(c)*
Moučník *tch.* 43*(a)*
Мозга [Туберкулозно запаљење —] / Mozga [Tuberkulozno zapaljenje—] } *serb.* 14*a*
Мозга [Запаљење —] / Mozga [Zapaljenje —] } *serb.* 6
Mozgowych nagminne [Epidemiczne zapalenie opon —] *pol.* 6
Mozkových a mišnich [Epidemicky zanet plen —] *tch.* 6
Mpace / Mraze } *serb.* 35
Mud-fever *angl.* 32*b(d)*
Mud field fever *angl.* 32*b(d)*
Muermo *esp.* 26*a*
Mughetto *it.* 43*(a)*
Muguet *esp. fr.* 43*(a)*
Muguras smadzenu iekaisums [Akutais —] *lett.* 36
Mugurkaula smadzenu degeneracija *lett.* 30*a*
Muhk [Medina või Orientaal —] *est.* 29*(bb)*
Mükoos *est.* 43
Mumps *all. angl.* 44*c*
Mundbetændelse *dan.* 115*a(a)*
Mundentzündung *all.* 115*a(a)*
Mundschleimhautentzündung *all.* 115*a(a)*
Mundschwämmchen *all.* 43*(a)*
Muninflammation *suéd.* 115*a(a)*
Munnangur *isl.* = *Stomatitis aphtosa* 115*a(a)*
Munţilor stăncoşi [Febra purpurie —] *roum.* 39*b(cc)*
Muriner Flecktyphus *all.* 39*b(a)*
Myalgi [Epidemisk —] *dan.* 44*d(c)*
Myalgia epidemica *lat.* 44*d(c)*
Myasis *it.* 42*(i)*
Mycétome *fr.* 43*c*
Mycosen *néer.* 43*c*
Mycoses *angl. fr. lat. port.* 43*c*
Myiase *fr.* 42*(i)*
Myiasis *angl.* 42*(i)*
Μυκωσεις / Mykoseis } *gr.* 43
Mykoser *d. & n.* 43
Mykosis *all.* 43*c*
Mykosy *tch.* 43*c*
Myocardite rhumatismale aiguë *fr.* 58
Myocardite rhumatismale subaiguë *fr.* 58
Myosite épidémique *fr.* 44*d(c)*
Myositis [Epidemic —] *angl.* 44*d(c)*
Myositis epidemica *lat.* 44*d(c)*

N

N. s. = *non-specificatus, a, um* = non spécifiée ; unspecified
Näärmete katk *est.* 3*a(a)*
Naha tuberkuloos *est.* 18
Nahaaluse koe tuberkuloos *est.* 18
Nákaza morová *tch.* 3*a(b)*
Nanukayami *japon.* 32*b(c)*
Нарыв / Naryv *rus.* 152
Нарыв или язва туберкулезная / Naryv ili iazva tuberkuleznaïa *rus.* 18
Нарыв [Холодный —] / Naryv [Kholodnyï —] *rus.* 19
Нарыв [Натечный —] / Naryv [Natechnyï —] *rus.* 19
Насморк / Nasmork (v) *rus.* 104*(a)*
Naso-pharyngitis *angl.* 115*c(b)*
Nátha *hong.* 33
Nautique de Toulon [Fièvre —] *fr.* 39*b(a)*
Nebenhöhlenentzündung *all.* 104*b*
Nefritis tuberculosa *esp.* 20
Negenoog *néer.* = *Furunculi conglomerati* 151
Nekkramp (v) *néer.* 6
Nemoc Hodkinova *tch.* 44*b*
Nemoc [Medinova —] *tch.* 36
Nemoci epidemické *tch.* = *Morbi epidemici*
Nemoci infekčni *tch.* = *Morbi infectiosi*
Neonatorum blennorrhoea gonorrhoica *all. lat.* 25*(b)*
Néphrite aiguë scarlatineuse *fr.* 8
Nephritis tuberculosa *lat. port.* 20
Nervenfieber (v) *all.* (obs.) 1
Nervensyphilis *all.** 30*d*
Nervfeber (v) *suéd.* 1
Neštovice *tch.* 34*c*
Neštovice plané *tch.* 38*e*
Neštovice pravé *tch.* 34*a*
Neštovice vodní *tch.* 38*e*
Neurolues* *all.* 30*d*
Nežitina *tch.* 151
Nezle *turc* 104*a(a)*
Nicolas-Favre [Maladie de —] *fr.* 44*a(b)*
Niežai *lith.* 153*(b)*
Nieżyt jelit *pol.* = *Enteritis* 119, 120*a*
Nieżyt kiszek *pol.* = *Enteritis* 119, 120*a*
Nieżyt kiszek i zołądka *pol.* = *Gastro-enteritis* 119, 120*a*
Nieżyt krtani *pol.* 105*(a)*
Nieżyt zołądka i jelit *pol.* = *Gastro-enteritis* 119, 120*a*
Нитевидная глиста / Nitevidnaia glista *rus.* 41*(f)*
Nivelreumatismi [Akillinen —] *fin.* 58
Nosacizna *pol.* 26*a*
Νοσοι / Nosoi *gr.* = *Morbi*
Νοσοι λοιμώδεις / Nosoi loimodeis *gr.* = *Morbi infectiosi*

O

Оболело / Obolelo *serb.* = *Casus*
Obrna [Detská —] *slovaque* 36
Obrna povšechná [Postupná —] *tch.* 30*b*
Obs. = *obsoletus*, suranné, obsolete
Общий прогрессивный паралич / Obstchiï progressivnyï paralitch *rus.* 30*b*
Odoskrzelowe *pol.* 107
Odra *pol.* 35
Œdem [Bösartiges —] (obs.) *all.* 7
Œdema [Malignant —] (v) *angl.* 24*c*
Œdema malignum (obs.) *all.* 7
Œdème gazeux *fr.* 24*c*
Œdème malin *fr.* 24*c*
Oftalmi [Gonorroisk —] *nor.* 25*(b)*
Oftalmi [Spædebarn —] *dan.* 25*(b)*
Oftalmia blenorragica *it.* 25*(b)*
Oftalmía blenorrágica *esp.* 25*(b)*
Oftalmia noilor-născuti *roum.* 25*(b)*
Oftică *roum.* 13*a*

Ogniskowe *pol.* 107
Ögonsjukdom [Smittsam —] *suéd.* 88
Ohrenentzündung *all.* 89*a(a)*
Oidium albicans *fr.* 43*a*
Оксириозис / Oksiriosis } *rus.* 42*(f)*
Ondulante [Febbre —] *it.* 5*a*
Ondulante [Fièvre —] *fr.* 5*c*
Ondulante bovine [Fièvre —] *fr.* 5*b*
Ondulante caprine [Fièvre —] *fr.* 5*a*
Ontsteking van 't strottenhoofd *néer.* 105*(a)*
Onemocnelých [Počet —] *slovaque* = *Numerus casuum*
Oorontsteking *néer.* 89*a(a)*
Ophtalmia blenorrhagica dos recemnatos *port.* 25*(b)*
Ophtalmie des nouveau-nés *fr.* 25*(b)*
Ophthalmia neonatorum *angl. lat.* 25*(b)*
Опоясывающий лишай / Opoiasyvaiustchiĭ lichaĭ } *rus.* 38*c*
Општа парализа / Opšta paraliza } *serb.* 30*b*
Orbáncz *hong.* 11
Orchite *fr.* 25*(a)*
Orchite ourlienne *fr.* 44*c*
Orchitis gonorrhoica *lat.* 25*(a)*
Orchitis tuberculosa *lat.* 20
Orchitt [Gonorroisk —] *nor.* 25*a*
Ørebetændelse *dan.* 89*a(a)*
Örebetennelse *nor.* 89*a(a)*
Orecchioni (v) *it.* 44*c*
Oreillons *fr.* 44*c*
Oreioane *roum.* 44*c*
Oreion *roum.* 44*c*
Orejones *esp.* 44*c*
Orientaal muhk *est.* 29*b(b)*
Oriental sore *angl.* 29*b(b)*
Orientbeule *all.* 29*b(b)*
Ormesygdomme *dan.* 42*(j)*
Oroya fever *angl.* 29*(f)*
Oroya [Fièvre d' —] *fr.* 29*(f)*
Orr betegségei [Az —] *hong.* 104*a*
Oskrzeli [Ostry niezyt —] *pol.* 106*a*
Oskrzeli [Przewlekly niezyt —] *pol.* 106*b*
Oskrzeli [Zapalenie —] *pol.* 106*c*
Ospa *pol.* 34*a*
Оспа / Ospa } *rus.* 34*a*
Оспа натуральная / Ospa naturalnaïa } *rus.* 34*a*
Оспа ветряная / Ospa vetrianaïa } *rus.* 38*e*
Ospa wietrzna *pol.* 38*e*
Оспице [Велике —] / Ospice [Velike —] } *serb.* 34*a*
Ossiuriosi *it.* 42*(f)*
Остатки летаргического энцефалита / Ostatki letargitcheskago encefalita } *rus.* 37*b*
Ostéite aiguë *fr.* 154
Ostéite chronique *fr.* 154
Osteite infettiva *it.* 154
Osteitis infecciosa *esp.* 154
Остеомиэлит / Osteomielit } *rus.* 154
Osteomielită *roum.* 154
Osteomielite *port.* 154
Osteomielite infettiva acuta *it.* 154
Osteomielitis infecciosa aguda *esp.* 154
Ostéomyélite *fr.* 154
Ostéomyélite tuberculeuse *fr.* 17*a*
Osteomyelitis *angl.* 154
Osteomyelitis acuta infectiosa *lat. néer.* 154
Osteomyelitis [Acute —] *angl.* 154
Osteomyelitis [Infective —] » 154
Osteomyelitis [Purulent —] » 154
Osteomyelitis infectiosa *all. lat.* 154
Οστεομυελιτις μολυσματικη οξεια / Osteomyelitis molysmatike oxeia } *gr.* 154
Osteomyelitis tuberculosa *lat.* 17*a*
Osteomyelitis [Tuberculous —] *angl.* 17*a*
Osteomyelitt *nor.* 154
Остеръ ставенъ ревматизъмъ / Oster staven revmatizem } *bulg.* 58

Ostitis tuberculosa *lat.* 17*b*

Οστρακια / Ostrakia — *gr.* 8

Острое воспаление эндокардия (не-ревматический эндокардит) / Ostroe vospalenie endocardia (ne-revmatitcheskiĭ endokardit) — *rus.* 91*c*

Ostry nieżyt oskrzeli *pol.* 106*a*

Острый бронхит / Ostryĭ bronkhit — *rus.* 106*a*

Острый эндокардит (не-ревматический эндокардит) / Ostryĭ endokardit (ne-revmatitcheskiĭ endokardit) — *rus.* 91

Острый эпидемический энцефалит / Ostryĭ epidemitcheskiĭ encefalit — *rus.* 37*a*

Острый летаргический энцефалит / Ostryĭ letargitcheskiĭ encefalit — *rus.* 37*a*

Острый не-менингококковый менингит / Ostryĭ ne-meningokokkovyĭ meningit — *rus.* 81*b*

Острый полиомиэлит / Ostryĭ poliomielit — *rus.* 36

Острый сочленовный ревматизм / Ostryĭ sotchlenovnyĭ revmatizm — *rus.* 58

Osýpky *slovaque tch.* 35

Otit *turc* 89*a(a)*

Отит / Otit — *rus.* 89*a(a)*

Otita *roum.* 89*a(a)*

Otite *fr. it. port.* 89*a(a)*

Otite grippale *fr.* 33*b*

Ωτιτις / Otitis — *gr.* 89*a(a)*

Otitis *all. angl. esp. néer.* 89*a(a)*

Otitis media *all. angl. lat. néer.* 89*a(a)*

Otitis media acuta *lat.* 89*a(a)*

Otitis media purulenta *lat. néer.* 89*a(a)*

Otrava krve *tch.* 24*a*

Otravenie krvi *tch.* 24*a*

Ourles *esp. fr.* 44*c*

Oxiurosis *esp.* 42*(f)*

Oxyurasis *all. hong. lat.* 42*(f)*

Oxyuris vermicularis *angl.* 42*(f)*

Oxyurose *fr.* 42*(f)*

Oxyurosis *néer.* 42*(f)*

Oxyuroză *roum.* 42*(f)*

Oxyurus vermicularis *all.* 42*(f)*

Ozhřivka *tch.* 26*a*

P

P... en caractères cyrilliques, *cf.* R...

P... in Cyrillic characters, *cf.* R...

Pachyméningite *fr.* 81*b*

Pachymeningitis *angl. lat.* 81*b*

Păduchi *roum.* 153*(a)*

Paerae (Sumatra) 32*b(c)*

Palavik [Unduleeruv —], Bang *est.* 5*b*

Paludéenne [Fièvre —] *fr.* 28*d*

Paludica *esp.* 28*d*

Paludism *roum.* 28*d*

Paludisme *fr.* 28*d*

Paludisme aigu *fr.* 28*d*

Paludisme chronique *fr.* 28*d*

Paludismo *esp. port.* 28*d*

Paludismo maligno *esp.* 28*c*

Palustre [Febre —] *port.* 28*d*

Palustre [Friguri —] *roum.* 28*d*

Panglică *roum.* 42*(g)*

Πανώλης / Panoles — *gr.* 3

Пантљичара / Pantljičara — *serb.* 42*(g)*

Papagaaienziekte *néer.* 44*d(d)*

Papageikrankheit *all.* 44*d(d)*

Papagöje Sygdom *dan.* 44*d(d)*

Papeira *port.* 44*c*

Pappataci fever *angl.* 38*f(b)*

Pappatacifieber *all.* 38*f(b)*

Pappataci [Fièvre à —] *fr.* 38*f(b)*

Pappatacifeber *d. & n.* 38*f(b)*

Paradysenteria *dan.* 27*a*
Paragonimiase *fr.* 42*(d)*
Paragonimiasi polmonare *it.* 42*(d)*
Paragonimiasis *angl.* 42*(d)*
Paralisi infantile *it.* 36
Paralisi progressiva (degli alienati) *it.* 30*b*
Paralisia geral *port.* 30*b*
Parálisis esencial de la infancia *esp.* 36
Parálisis espinal aguda *esp.* 36
Parálisis general *esp.* 30*b*
Параличъ [Детски —] / Paralitch [Detski —] *bulg.* 36
Паралич [Детский —] / Paralitch [Detskiĭ —] *rus.* 36
Паралич [Прогрессивный помешанных —] / Paralitch [Progressivnyĭ pomechannykh —] *rus.* 30*b*
Paraliż dziecięcy *pol.* 36
Paraliż postępowy *pol.* 30*b*
Парализа [Дечија —] / Paraliza [Dečija —] *serb.* 36
Парализа [Општа —] / Paraliza [Opšta —] *serb.* 30*b*
Paralize [Vispārējā —] *lett.* 30*a*
Paralizie generală *roum.* 30*b*
Paralizie infantilă *roum.* 36
Paralüüs [Üldine —] *est.* 30*b*
Paralyse [Generel —] *dan.* 30*b*
Paralyse [Progressive —] *all. néer.* 30*b*
Paralysi [Allmän —] *suéd.* 30*b*
Παράλυσια γενική / Paralysia genike *gr.* 30*b*
Paralysia geral *port.* 30*b*
Paralysia infantil *port.* 36
Paralysie générale *fr.* 30*b*
Paralysie infantile *fr.* 36
Paralysis agitans *all. lat. néer.* 37*b*
Paralysis generalis progressiva alienorum *lat.* 30*b*
Paralysis [Infantile —] *angl.* 36
Paralysis of the insane [General —] *angl.* 30*b*
Parasites intestinaux *fr.* 42*(j)*
Паратиф / Paratif *rus.* 2
Paratifiche [Febbri —] *it.* 2
Paratifiche [Infezioni —] *it.* 2
Paratifo *it.* 2
Paratifoide *port.* 2
Paratifoidi (Febbri) *it.* 2
Paratifs *lett.* 2
Paratifus *esp. néer. roum. turc* 2
Paratifusz *hong.* 2
Paratüüfus *est.* 2
Paratyfas *lith.* 2
Paratyfoid *suéd.* 2
Paratyfus *d. & n. fin. pol.* 2
Paratyfy *tch.* 2
Παρατυφοειδεις πυρετοι / Paratyphoeideis pyretoi *gr.* 2
Paratyphoid fevers *angl.* 2
Paratyphoïde(s) [Fièvre(s) —] *fr.* 2
Paratyphoïde koortsen *néer.* 2
Παρατυφος / Paratyphos *gr.* 2
Paratyphus *all. lat.* 2
Парша / Parcha *rus.* 43*(b)*
Paresis [General —] *angl.* 30*b*
Parkinsonism *angl. roum.* 37*b*
Parkinson [Sindromul lui —] *roum.* 37*b*
Parkinsonisme *fr.* 37*b*
Parkinsonsche Krankheit *all.* 37*b*
Parotidite epidemica *port.* 44*c*
Parotidite épidémique *fr.* 44*c*
Parotidite infecciosa *port.* 44*c*
Parotis *lat.* 44*c*
Паротит [Эпидемический —] / Parotit [Epidemicheskiĭ —] *rus.* 44*c*
Parotite epidemica *it.* 44*c*
Parotitis [Epidemic —] *angl.* 44*c*
Parotitis epidemica *all. lat.* 41*c*
Παροτιτις επιδημικη / Parotitis epidemike *gr.* 44*c*
Pasiutimas *lith.* 38*b*
Påssjuka *suéd.* 44*c*
Pateh [Java —] *néer.* 32*b(c)*
Patra [Maladia a —] *roum.* 44*d(e)*
Pęcherzyca *pol.* 153*(c)*
Pęcherzyca noworodków *pol.* 161*(a)*
Pedicularis morbus *lat.* 153*(a)*
Pediculatio capitis, corporis, pubis *lat.* 153*(a)*

Pediculi *all. lat.* 153*(a)*
Pédiculose *fr.* 153*(a)*
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Rickettsioses *angl. fr.* 39*c*
Rickettsiosi *it.* 39*c*
Rickettsiosis quintana *lat.* 39*c(a)*
Rickettsioze *roum.* 39*c*
Рикетциозы } *rus.* 39*c*
Riketsiosi }
Ringorm *d. & n.* 43*(a)*
Ringworm *angl.* 43*(b)*
Rinitis *esp.* 104*a*
Rippenfellentzündung (v) *all.* 110*b*
Riquetsiose maculosa neotropica *port.* 39*b(cd)*
Riquetsiose murina *port.* 39*b(a)*
Risatauti *fin.* 19
Risipola (v) *it.* 11
Ristill *isl.* 38*c*
Rocky Mountains spotted fever *angl.* 39*b(cc)*
Röda hund *suéd.* 38*d*
Röde Hunde *d. & n.* 38*d*
Родилна треска } *bulg.* 147
Rodilna treska }
Родильниц [Септицемия —] } *rus.* 147
Rodilnits [Septicemia —] }
Rödlinger *dan.* 38*d*
Родовая горячка } *rus.* 147
Rodovaïa goriatchka }
Rödsot *suéd.* 27*d*
Rödsot [Epidemisk —] *suéd.* 27*a*
Rogna (v) *it.* 153*(b)*
Roiskveresus *est.* 24*a*
Roiskveresus [Lapsevoodi —] *est.* 147
Рожа } *rus.* 11
Roja }
Рожа [Поясовидная —] } (v) *rus.* 38*c*
Roja [Poiasovidnaïa —] }
Romadizo *esp.* 104*a*
Roña (v) *esp.* 153*(b)*
Roncsoló toroklob *hong.* 10
Roode hond *néer.* 38*d*
Roodvonk *néer.* 8
Roos *est. néer.* 11
Ropne zapalenie opłucnej *pol.* 110*a*
Ropniak opłucnej *pol.* 110*a*
Ropnica *pol.* 24*b*
Ropowica *pol.* 152
Ros *suéd.* 11
Rose (v) *all.* 11
Rosen *d. & n.* 11

Roseola *angl. esp.* 38*d*
Roseola syphilitica *lat.* 30*d*
Rose rash [Epidemic —] (v) *angl.* 38*d*
Rosolia *it.* 38*d*
Röteln *all.* 38*d*
Rotlauf *all.* 11
Rots *suéd.* 26*a*
Rottebidsygdom *dan.* 32*b(b)*
Rotz *all.* 26*a*
Rõuged *est.* 34*a*
Rougeole *fr.* 35
Róża *pol.* 11.
Roze *lett.* 11
Rože *lith.* 11
Rozklad krve *tch.* 24*b*
Rozyczka *pol.* 38*d*
Ruam *turc* 26*a*
Rubella *angl. lat.* 38*d*
Rubella scarlatinosa *angl. lat.* 44*d(e)*
Rubeola *all. esp. néer. port. roum. serb.* 38*d*
Rubeola *angl.* 35

— dans les pays de langue anglaise, équivaut à « rougeole » *(morbilli)* et non à « rubéole » *(rubella)*, comme c'est le cas dans la plupart des pays.

— In English-speaking countries, "*rubeola*" means "*measles*" *(morbilli)* and not "*german measles*" *(rubella)*, as is the case in most countries.

Рубеола / Rubeola *serb.* 38*d*
Rubeolă scarlatiniformă *roum.* 44*d(e)*
Rubeola scarlatinosa *all. lat.* 44*d(e)*
Rubeolæ *dan. néer. nor.* 38*d*
Rubéole *fr.* 38*d*
Rückenmarklähmung der Kinder *all.* 36
Rückenmarkschwindsucht *all.* 30*a*
Rückfallfieber *all.* 31*c*
Rugemergstering (v) *néer.* 30*a*
Rugeola *esp. roum.* 35
Rugueola* *esp.* 35
Rühkor *hong.* 153*(b)*
Ruhr *all.* 27*d*
Ruhr [Amoeben —] *all.* 27*b*
Ruhr [Bazilläre —] *all.* 27*a*
Rupia (v) *angl.* 30*d*
Ruusu *fin.* 11
Růže *tch.* 11
Ryggmargstœring *nor.* 30*a*
Ryggmärgstvinsot *suéd.* 30*a*
Ryggmarvstuberkulose *nor.* 16
Rygmarvstæring *dan.* 30*a*
Rygmarvstuberkulose *dan.* 16
Rýma *tch.* 104*a(a)*
Rzeżączka *pol.* 25*(a)*

S

s.a.i. = sans autre indication *fr.*
» = senz'altra indicazione *it.*
» = *non specificatus*
» = unspecified
s.o.i. = sin otra indicación *esp.*
» = sem outra indicação *port.*
» = sans autre indication
» = unspecified
» = *non specificatus*
Saar [Veneriske —] *dan.* 44*a(a)*
Сакагија / Sakagija *serb.* 26*a*
Salpingite tuberculeuse *fr.* 20
Sancăr moale *roum.* 44*a(a)*
Sandfly fever *angl.* 38*f(b)*
Šankeŗ [Mikstais —] *lett.* 44*a(a)*
Šankeris [Minkštasis —] *lith.* 44*a(a)*
São Paulo [Fièvre pourprée de —] *fr.* 39*b(cd)*
São Paulo tick-typhus *angl.* 39*b(cd)*
Сап / Sap *bulg. rus.* 26*a*
Sår [Veneriske —] *dan.* 44*a(a)*
Sarampão *port.* 35
Sarampello *port.* 35
Sarampión *esp.* 35
Sarampo (v) *port.* 35
Şarbon *turc* 7*c*
Sarcoptose *fr.* 153*(b)*
Sárgaláz *hong.* 38*a*
Sárgasag *hong.* 127
Sari humma *turc* 38*a*
Sarî sehaya iltihabi *turc* 6
Шарлах / Šarlah, Sharlah *serb.* 8
Sarlakid *est.* 8
Šarlaks *lett.* 8
Sarna *esp. port.* 153*(b)*
Saslimšana *lett.* = *aegrotare*

Sastinguma krampji *lett.* 12
Scabbia *it.* 153*(b)*
Scabie *roum.* 153*(b)*
Scabies *all. angl. fr. lat. néer.* 153*(b)*
Scarlatina *all. angl. néer. lat. roum.* 8
Scarlatine *fr.* 8
Scarlattina *it.* 8
Scarlet fever *angl.* 8
Schafblattern *all.* 38*e*
Schafpocken *all.* 38*e*
Schanker *suéd.* 44*a(a)*
Schanker [Harde —] *néer.* 30*d*
Schanker [Weicher —] *all.* 44*a(a)*
Schanker [Weke —] *néer.* 44*a(a)*
Schanker [Zachte —] *néer.* 44*a(a)*
Scharlach *all.* 8
Scharlakansfeber *suéd.* 8
Schistosomiase *fr.* 42*(b)*
Schistosomiasi *it.* 42*(b)*
Schistosomiasis *angl.* 42*(b)*
Schizotripanosomiasis *it.* 29*(d)*
Schlafkrankheit *all.* 29*(e)*
Schlafsuchtkrankheit *all.* 37*c*
Schlagadererweiterung *all.* 30*c*
Schlammfieber *all.* 32*b(d)*
Schnupfen *all.* 104*a*
Schurft (v) *néer.* 153*b*, 43*(b)*
Schützengrabenfieber *all.* 39*c(a)*
Schwarze Pocken *all.* 34*a*
Schwarzwasserfieber *all.* 28*c(b)*
Schweinehüterkrankheit *all.* = *Meningitis serosa porcinarii* 38*f*
Schweissfieber *all.* 44*d(f)*
Schweissfrieseln *all.* 44*d(f)*
Schwindsucht (v) *all.* 13*c*
Schwindsucht der Gekrösedrüsen *all.* 15*(b)*
Schyzotrypanum cruzi [Infection à —] *fr.* 29*(d)*
Scroffer *suéd.* 19
Scrofola (v) *it.* 19
Scrofula* *angl.* 19
Scrofuloderma *angl.* 18
Scrofulodermie *fr.* 18
Scrofulose *all.* 19
Scrofulosis *all. lat.* 19
Scrophulose *néer.* 19
Seentõbi *est.* 43
Seiles liauku uždegimas [Epid —] *lith.* 44*c*
Sekundaar-nähtused *est.* 30*d*
Seljataabes *est.* 30*a*
Senyv [Mocsarlazas —] *hong.* = *Cachexia palustre* 28*d*
Σηψαιμια / Sepsaimia } 24*a*
Σηψαιμια επιλοχειοι / Sepsaimia epilocheioi } *gr.* 147
Sepsi generalizzata *it.* 24*a*
Sepsis* *angl. hong. néer.* 24*a*
Sepsis (behalve puerperale) *néer.* 24*a*
Sepsis (ohne Kindbettfieber) *all.* 24*a*
Sepsis [Puerperal —] *angl.* 147
Sepsis puerperalis *lat.* 147
Septicæmia* [Non-puerperal —] *angl.* 24
Septicemia *esp. port.* 24*a*
Septicemia puerperal *esp. port.* 147
Септицемия родильниц / Septicemia rodil'nits } *rus.* 147
Septicémie charbonneuse *fr.* 7*b*
Septicémie (non puerpérale) *fr.* 24*a*
Septicémie pesteuse *fr.* 3*a(b)*
Septicémie puerpérale *fr.* 147
Septicemija *lith.* 24*a*
Septichaemia *lat. néer.* 24*a*
Septichaemie in het kraambet *néer.* 147
Септична ангина / Septična angina } *serb.* 115*b*
Септично заражење / Septično zaraženje } *serb.* 24*a*
Septikaemi *d. & n.* 24*a*
Septikaemie *all.* 24*a*
Septikæmisk Pest *d. & n.* 3*a(b)*
Septisemi *turc* 24
Септическая ангина / Septitcheskaïa angina } *rus.* 115*b*
Септицемия / Septitsemia } *rus.* 24*a*
Sequelæ encephalitis lethargicæ *lat.* 37*b*
Séquelles d'encéphalite léthargique *fr.* 37*b*
Sērgas bacillis [Liesas —] *lett.* 7*c*
Setticemia s.a.i. *it.* 24*a*

Setticemia (non puerperale) *it.* 24*a*
Setticemia carbonchiosa *it.* 7*b*
Setticemia pestosa *it.* 3*a(b)*
Setticemia puerperale *it.* 147
Seuchenhaftes Verkalben (v) *all.* 5*b*
Sezonismo *port.* 28*d*
Shashitsu *japon.* 39*b(b)*
Shimamushi *japon.* 39*b(b)*
Shingles (v) *angl.* 38*c*
Shiga dysentery *angl.* 27*a*
Shop (tropical) typhus *angl.* 39*b(bc)*
Sibiro maras *lith.* 7*c*
Сибирская язва / Sibirskaïa iazva } *rus.* 7*(a)*
Siebentagefieber *all.* 32*b(e)*
Sifilide *it.* 30
Sifilide congenita *it.* 30*d(a)*
Sifilis *esp. roum.* 30
Сифилис / Sifilis } *bulg. rus. serb.* 30
Sifilis congénita *esp.* 30*d(a)*
Sifilis congenital *roum.* 30*d(a)*
Сифилис [Наследни —] / Sifilis [Nasledni —] } *serb.* 30*d(a)*
Сифилис наследственный / Sifilis nasledstvennyï } *rus.* 30*d(a)*
Сифилис первичный / Sifilis pervitchnyï } *rus.* 30*d*
Сифилис третичный / Sifilis tretitchnyï } *rus.* 30*d*
Сифилис врожденный / Sifilis vrojdennyï } *rus.* 30*d(a)*
Сифилис вторичный / Sifilis vtoritchnyï } *rus.* 30*d*
Sifiliss *lett.* 30
Sifiliss [Iedzimts —] *lett.* 30*d(a)*
Сифилитическая жаба / Sifilititcheskaïa jaba } *rus.* 30*d*
Sikotauti *fin.* 44*c*
Silmamarjad *est.* 88
Šiltine [Atokrytine —] *lith.* 31*a*
Šiltine [Beriamoji —] *lith.* 39*a*
Šiltine [Viduriu —] *lith.* 1
Simbiosis fuso-espirilar *esp.* 29*(c)*
Singhiozzo epidemico *it.* 44*d(h)*
Singultus epidemica *lat.* 44*d(h)*
Sinus mastoideuse poletik *est.* 89*b(a)*
Sinus põletik *est.* 104*b(a)*
Sinusit *dan.* 104*b(a)*
Синузит / Sinusit } *rus.* 104*b(a)*
Sinusită *roum.* 104*b(a)*
Sinusită mastoidiană *roum.* 89*b(a)*
Sinusite *it. fr. port.* 104*b(a)*
Sinusitis *all. angl. esp. lat.* 104*b(a)*
Sinüzit *turc* 104*b(a)*
Синята пжпка / Sinyata pjepka } *bulg.* 7*a*
Sirds iekšejas pleves iekaisums [Akutais —] (nereimatiskais) *lett.* 91*a*
Şirit *turc* 42*(g)*
Siseelundkonna parasiidid *est.* 42*(g)*
Систеркозы / Sisterkosy } *rus.* 42*(c)*
Sitma *turc* 28*d*
Sjukdom, ar *suéd.* = *Morbus, i*
Sjukdomsfall *suéd.* = *Casus morbi*
Sjukligheten *suéd.* = *Casus morbi*
Skabb *nor. suéd.* 153*(b)*
Skarlagensfeber *d. & n.* 8
Skarlakansfeber *suéd.* 8
Skarlát *hong.* 8
Скарлатина / Skarlatina } *bulg. rus.* 8
Skarlatina *lith.* 8
Скарлатинообразная краснуха / Skarlatinoobraznaïa krasnukha } *rus.* 44*d(e)*
Skarlatsótt *isl.* 8
Skoldkopper *dan.* 38*e*
Σκωληκοειδιτις / Skolekoeiditis } *gr.* 121
Skorv *suéd.* 43*(b)*
Škrkavka *tch.* 42*(f)*
Скрлет / Skrlet } *serb.* 8
Skrofeln *all.* 19
Skrofulose *d. & n.* 19
Скрофулез / Skrofuloz } (v) *rus.* 19
Скрофулоза / Skrofuloza } *serb.* 19
Skvrnitý tyf *tch.* 39*a*
Skvrnivka *tch.* 39*a*

Slaapziekte [Afrikansche —] *néer.* 29*(e)*
Slaapziekte [Europeesche —] *néer.* 37*c*
Slaapzucht *néer.* 37*c*
Slăbire (v) *roum.* 13*c*
Sleeping sickness *angl.* 29*(e)*
Sleepy sickness *angl.* 37*c*
Слязетечение / Sliazetetchenie (v) *rus.* 25*(a)*
Slimiba(s) *lett.* = *Morbus, i*
Slintavka a kulhavka (vet.) *tch.* 44*d(a)*
Случай / Slutchai *bulg. rus.* = *Casus*
Smadzenu iekaisums [Akutais galvas —] *lett.* 36
Smadzenu iekaisums [Akutais muguras —] *lett.* 36
Smadzenu iekaisums [Letargiskais —] *lett.* 37
Smadzenu iekaisums [Epidemiskais —] *lett.* 37
Smallpox *angl.* 34*c*
Smallpox [Mild —] *angl.* 34*b*
Smallpox [Severe —] *angl.* 34*a*
Smegenu [Galvos —] uždegimas *lith.* 36
Smegenu [Letargiskas —] uždegimas *lith.* 37*c*
Smegenu pleves uždegimas [Epid. —] *lith.* 6
Смерть / Smert *bulg. rus.* = *Mortuus*
Smittelförende tuberculose *dan.* = *Tuberculosis contagiosa* 13*b(c)*
Smittkoppor *suéd.* 34
Smittsam gulsot *suéd.* 44*d(g)*
Smittsam ögonsjukdom *suéd.* 88
Smittsam, ma *suéd.* = *Contagiosus, a, um*
Смрти / Smrti *serb.* = *Mortuus, i*
Snět plynová *tch.* 24*c*
Snět slezinná *tch.* 7*b*
Snive *d. & n.* 26*a*
Snue *d. & n.* 104*a(a)*
Snuffles (v) *angl.* 30*d*
Snukio ir nagu liga *lith.* 44*d(a)*
Snuva *suéd.* 104*a(a)*
Sodoka *it.* 32*b(b)*
Sodoku *ang. esp. fr. it. port. roum.* 32*b(b)*
Содоку / Sodoku *rus.* 32*b(b)*
Soe mädanik *est.* = *Abscessus calidus* 152
Soemail *est.* 29*(c)*
Soft sore *angl.* 44*a(a)*
Soğukluğu [Bel —] *turc* 25*(a)*
Sokizületű csúz *hong.* 58
Sokizületű csúz [Heveny —] *hong.* 58
Sokizületű csúz [Heveny lázas —] *hong.* 58
Sokizületű csúz [Lázas —] *hong.* 58
Sokizületű [Heveny lázas —] csuz *hong.* 58
Солитер / Soliter *rus.* 41*(g)*
Sommeil [Maladie du —] *fr.* 29*(e)*
Sommerdurchfall *all.* 119, 120*a*
Sömnsjuka *suéd.* 27*d*
Somnului [Boala —] *roum.* 29*(e)*
Сонная болезнь / Sonnaïa bolezn *rus.* 29*(e)*
Sonne dysentery *angl.* 27*a*
Sonno [Malattia del —] *it.* 29*(e)*
Sono [Doença do —] *port.* 29*(e)*
Soojail mail *est.* 29*(c)*
Soolepõletik *est* = *Enteritis* 119, 120*a*
Soolte tuberkuloos *est.* 15*(a)*
Soopalavik *est.* 28
Sore throat (v) *angl.* 115*c(b)*
Sore throat [Septic —] *angl.* 115*b*
Souchotiny *tch.* 13*c*
Sovesyge *dan.* 29*(e)*
Sovesyke *nor.* 29*(e)*
Spála *tch.* 8
Spædebarn Oftalmi *dan.* 25*(b)*
Spalničky *tch.* 35
Spanskesyke (v) *nor.* 33
Spebarn-oftalmi *nor.* 25*(b)*
Specifiche [Manifestazioni —] *it.* 30*d*
Spécificité* *fr.* 30*d*
Spécificité congénitale *fr.* 30*d(a)*
Spedalskhed *dan.* 23

Spedalskhet *nor.* 23
Spetälska *suéd.* 23
Spiączka *pol.* 37*c*
Spirillosis *angl.* 31*a*
Spirochaetose [Recurrens —] *néer.* 31*c*
Spirochaetosis bronchialis *néer.* 32*b*
Spirochætosis ictero-hemorrhagica *angl. lat.* 32*a*
Spirochétose africaine *fr.* 31*c*
Spirochétose ictéro-hémorragique *fr.* 32*a*
Spirochetosi ittero-emorragica *it.* 32*a*
Spirochetosis Duttoni *lat.* 31*b*
Spirochetosis Obermeieri *lat.* 31*a*
Spirochetoza ictero-hemoragică *roum.* 32*a*
Spirochæta duttoni [Infection à —] *fr.* 31*b*
Spirochæta obermeieri [Infection à —] *fr.* 31*a*
Спирохетоз [Желчно-геморагический —] / Spirokhetoz [Jeltchno-gemoragitcheskiĭ —] *rus.* 32*a*
Spitaa'itauti *finn.* 23
Spitālība *lett.* 23
Spitzpocken *all.* 38*e*
Splénomégalie palustre *fr.* 28*d*
Splenopolmonite (obs.) *it.* 108
Spondylite tuberculeuse *fr.* 16
Spondylitis [Tuberculous —] *angl.* 16
Springwurm (v) *all.* 42*(f)*
Spruw *néer.* 43(*a*)
Срдобоља / Srdobolja *serb.* 27*d*
Stabas (mešlungis) *lith.* 12
Stafilococcemia *it.* 24*b*
Staphylococcus infection *angl.* 24*b*
Starrkrampf *all.* 12
Steenpuist *néer.* 151
Stelkramp *suéd.* 12
Stijfkramp *néer.* 12
Stingsótt *isl.* = *Pleuritis epidemica* 110*b*
Stivkrampe *d. & n.* 12
Stivkrampe hos nyfödte *d. & n.* = *Tetanus neonatorum* 12
Столбняк / Stolbniak *rus.* 12
Стоматит / Stomatit *rus.* 115*a(a)*
Stomatită *roum.* 115*a(a)*
Stomatite *fr. it.* 115*a(a)*
Στοματιτις / Stomatitis *gr.* 115*a(a)*
Stomatitis *all. angl. lat. néer.* 115*a(a)*
Stomatitis aphthosa epidemica *lat.* 44*d(a)*
Stomatitis [Spirochætal —] *angl.* 32*b(a)*
Stomatomycosis *all. angl. lat.* 43*(a)*
Streptococcemia *it.* 24*a*
Streptococcus infection *angl.* 24*a*
Streptokokangina *dan.* 115*b*
Стрептококкцемия / Streptokokkcemia *rus.* 24*a*
Streptokokken Angina *all.* 115*b*
Стрептококковая ангина / Streptokokkovaïa angina *rus.* 115*b*
Стригучий лишай / Strigutchiĭ lichaï *rus.* 43*(b)*
Strubehoste *dan.* 10
Strubekatarrh [Akut —] *dan.* 105*(a)*
Strubetuberkulose *dan.* 13*b(a)*
Strupetuberkulose *nor.* 13*b(a)*
Struma *angl.** 19
Strupekatarrh [Akutt —] *nor.* 105*(a)*
Strupinflammation [Akut —] *suéd.* 105*(a)*
Strupovitost *tch.* 43*(b)*
Struptuberkulos *suéd.* 13*b(a)*
Subtertian malaria *angl.* 28*c*
Süd-afrikanisches exanthematisches Zeckenfieber *all.* 39*b(cb)*
Suchoty *pol.* 13*c*
Südame sisekesta-põletik [Äge batsillaarne —] *est.* 91*a*
Südame sisekesta-põletik [*Str. viridans* —] *est.* 91*b*
Sudor anglicus *lat.* 44*d(f)*
Sudor miliar *esp.* 44*d(f)*
Sudor miliaris *lat.* 44*d(f)*
Sueño [Enfermedad del —] *esp.* 29*(e)*

Suette *fr.* 44*d(f)*
Suette miliaire *fr.* 44*d(f)*
Шуга, Šuga } *serb.* 23
Sugár gombabetegs *hong.* = *Actinomycosis* 43*c*
Сухотка, Sukhotka } (v) *rus.* 13*c*
Сухотка спинного мозга, Sukhotka spinnogo mozga } *rus.* 30*a*
Sullaveiki *isl.* 41*b*
Sumpffieber *all.* 32*b(d)*
Suolikatarri (Akillinen —) *fin.* 120*a*
Suolitulehdus (Tarttuva) *fin.* 120*a*
Suor maligno miliar *port.* 44*d(f)*
Сушење кичме, Sušenje kičme } *serb.* 30*a*
Сушица, Sušica } *serb.* 13*c*
Суставной ревматизм, Sustavnoï revmatizm } *rus.* 58
Süüfilis *est.* 30
Süüfilis [Omandatud — = *Syphilis acquisita est.* 30*d*
Süüfilis [Kaasasündinud —] *est.* 30*d(a)*
Suuõõne põletik *est.* 115*a(a)*
Svalget [Akut inflammation i —] *suéd.* 115*c(b)*
Svefnsyki *isl.* 37*c*
Svindsot *d. & n.* 13*c*
Svingefeber *d. & n.* 5*b*
Svrab *tch.* 153*(b)*
Sweating sickness *angl.* 44*d(f)*
Swierzb *pol.* 153*(b)*
Swinka *pol.* 44*c*
Syfilis *d. & n. lith. pol. suéd.* 30
Syfilis [Ärvd —] *suéd.* 30*d(a)*
Syfilis [Erhvervet —] = *Syphilis acquisita d. & n.* 30*d*
Syfilis [Förvärvad —] = *Syphilis acquisita* 30*d*
Syfilis [Medfödt —] *d. & n.* 30*d(a)*
Synnynnäinen kuppatauti *fin.* 30*d(a)*
Σύφιλις, Syphilis } *gr.* 30
Syphilis *all. angl. fr. lat. néer. port.* 30
Syphilis (Aangeborene) *néer.* 30*d(a)*
Syphilis (Angeborene) *all.* 30*d(a)*
Syphilis congenita *all. lat. port.* 30*d(a)*
Syphilis (Congenital) *angl.* 30*d(a)*
Syphilis congénitale *fr.* 30*d(a)*
Syphilis (Ererbte) *all.* 30*d(a)*
Syphilis hereditaria *all. lat.* 30*d(a)*
Syphilis [Hereditary —] *angl.* 30*d(a)*
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Syphilis secundaria » 30*d*
Syphilis tertiaria » 30*d*
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Syphilis (Verkregen) *néer.* 30*d*
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Szájpenész *hong.* 43*a*
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Szifilisz [Szerzett —] *hong.* = *Syphilis acquisita* 30*d*
Szifilisz [Veleszületett —] *hong.* 30*d(a)*
Szivbelhártyagyulladás [Heveny —] *hong.* = *Endocarditis acuta* 91*a*
[Heveny bacillusos —] *hong.* 91*a*
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T

T. B. *angl.* 13*c*
T. B. C. *néer.* 13*c*
Tbc. *angl.* 13*c*
Tbk. *all.* 13*c*
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Tabe meseraica *it.* 15*(b)*
Tabes dorsal *esp. port.* 30*a*
Tabes dorsalis *all. angl. fr. lat. néer. roum.* 30*a*

Tabes mesaraica *all. lat.* 15*(b)*
Tabes mesenterica *all. angl. lat.* 15*(b)*
Taboparalyse *néer.* 30*b*
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Тэния / Taenia } *rus.* 42*(g)*
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Taenia echinococcus hepatis *lat.* 41*a*
Taenia saginata *lat.* 42*(g)*
Taenia solium *all. lat.* 42*(g)*
Taeniasias *est.* 42*(g)*
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Tähniline tüüfus *est.* 39*a*
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Taksótt *isl.* 108
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Tarmtuberkulos *suéd.* 15*(a)*
Tarmtuberkulose *dan.* 15*(a)*
Tarmtyfus *suéd.* 1
Tarttuva mahatulehdus *fin.* 120*a*
Tarttuva suolitulehdus *fin.* 120*a*
Tasemnice *tch.* 42*(g)*
Tasiemiec *pol.* 42*(g)*
Tatitõbi *est.* 26*a*
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Чахотка / Tchakhotka } (v) *rus.* 13*c*
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Чесотка / Tchesotka } *rus.* 153*(b)*
Четвертая болезнь / Tchetvertaïa bolèzn } *rus.* 44*d(e)*
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Чума легочная / Tchuma liogotchnaïa } *rus.* 3*b*
Чума септическая / Tchuma septitcheskaïa } *rus.* 3*a(b)*
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Teniasi *it.* 42*(g)*
Teniasis *esp.* 42*(g)*
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Тиф [Эпидемический возвратный —] / Tif [Epidemitcheskiĭ vozvratnyï —] *rus.* 31*a*
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Tifo endemico benigno *esp. it.* 39*b(a)*
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Tifo murino *it.* 39*b(a)*
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Tuberculosis systematis nervorum centralis et meningum *lat.* 14
Tuberculosis systematis urogenitalis *lat. néer.* 20
Tuberculosis telae cellulosae *lat. néer.* 18
Tuberculosis [Urinary —] *angl.* 20
Tuberculosis urogenitalis *lat.* 20
Tuberculosis [Vertebral —] *angl.* 16
Tuberculosis vertebralis *lat.* 16
Tuberculosis viscerum abdominalium *lat.* 15
Tuberculoso (Enfermedad de Addison de origen —) *esp.* 21*a*
Tuberculous Addison's disease *angl.* 21*a*

Tuberculoza abdominal *roum.* 15*(c)*
Tuberculoza aparatului genito-urinar *roum.* 20
Tuberculoza aparatului respirator *roum.* 13*c*
Tuberculoza a articulaţiilor *roum.* 17*b*
Tuberculoza colvanei vertebrale *roum.* 16
Tuberculoza diseminată *roum.* 22*c*
Tuberculoza diseminată acuta *roum.* 22*a*
Tuberculoza diseminată cronică *roum.* 22*b*
Tuberculoza intestinelor *roum.* 15*(a)*
Tuberculoza meningelor *roum.* 14*a*
Tuberculoza miliară *roum.* 22*a*
Tuberculoza oaselor *roum.* 17*a*
Tuberculoza a peritoneului *roum.* 15*(b)*
Tuberculoza pielei *roum.* 18
Tuberculoza pulmonara *roum.* 13*b(c)*
Tuberculoza sistemului limfatic *roum.* 19
Tuberculoza sistemului nervos central *roum.* 14*a*
Tuberculoza a ţesutului celular subcutanat *roum.* 18
Туберкулезный менингит / Tuberkuleznyï méningit *rus.* 14*a*
Туберкулезный нарыв / Tuberkuleznyï naryv *rus.* 17*a*
Туберкулезный перетонит / Tuberkuleznyï peritonit *rus.* 15*(b)*
Tuberkuloos *est.* 13*c*
Tuberkuloos [Hingamis organite —] *est.* 13
Tuberkuloosi [Muus —] *fin.* 21
Tuberkuloosne mahlanäärmete *est.* 19
Tuberkuloottinen aivokalvontulehdus *fin.* 14*a*
Tuberkulos *suèd.* 13*c*
Tuberkulos [Allmän —] *suéd.* 22*c*
Tuberkulos [Annan —] *suèd.* 21
Tuberkulos i andra organen *suèd.* 21

Tuberkulos i könsorganen *suéd.* 20*(a)*
Tuberkulös meningitt *nor.* 14*a*
Tuberkulos i urinvägarna *suéd.* 20*(b)*
Tuberkulosa *tch.* 13*c*
Tuberkulosa jiných míst *tch.* 21
Tuberkulosa kloubů *tch.* 17*b*
Tuberkulosa kostí *tch.* 17*a*
Tuberkulosa kůže 18
Tuberkulosa miliární *tch.* 22*c*
Tuberkulosa miliární prudká *tch.* 22*a*
Tuberkulosa miliární vleklá *tch.* 22*b*
Tuberkulosa obratlů *tch.* 16
Tuberkulosa páteře *tch.* 16
Tuberkulosa plic *tch.* 13*b(c)*
Tuberkulosa pobřišnice *tch.* 15*(b)*
Tuberkulosa podkožního vaziva *tch.* 18
Tuberkulosa roztroušená *tch.* 22*c*
Tuberkulosa soustavy mízní *tch.* 19
Tuberkulosa střev *tch.* 15*(a)*
Tuberkulosa urogenitalního *tch.* 20
Tuberkulosa ústrojí dychacího *tch.* 13*b*
Tuberkulosa ústrojí močového *tch.* = *Tuberculosis organorum uropoëticorum* 20
Tuberkulosa ústrojí pohlavního *tch.* = *Tuberculosis genitalis* 20
Tuberkulosa žlaz *tch.* 19
Tuberkulöse addisonsche Krankheit *all.* 21*a*
Tuberkulöse Bronzekrankheit *all.* 21*a*
Tuberkulose der Atmungsorgane *all.* 13*c*
Tuberkulose der Drüsen *all.* 19
Tuberkulose der Geschlechtsorgane *all.* 20
Tuberkulose der Harnorgane *all.* 20
Tuberkulose der Haut *all.* 18
Tuberkulose der Hirnhaut *all.* 14
Tuberkulose der Knochen *all.* 17*a*
Tuberkulose der Unterleibsorgane *all.* 15*(c)*
Tuberkulose der Wirbelsäule *all.* 16
Tuberkulose des Bauchfells *all.* 15*(c)*
Tuberkulose des Gehirns *all.* 14*b*
Tuberkulose des Peritoneums *all.* 15*(b)*
Tuberkulose des Unterhautzellgewebes *all.* 18
Tuberkulose det subkutant Valev *d. & n.* 18
Tuberkulöse Hirnhautentzündung *all.* 14*a*
Tuberkulose i Aandedrætsorganerne *d. & n.* 13
Tuberkulose i hjærnehinder *d. & n.* 14*a*
Tuberkulose i Hud *d. & n.* 18
Tuberkulose i Knogler *dan.* 17*a*
Tuberkulose i Kønsorganerne *dan.* 20
Tuberkulose i Led *dan.* 17*b*
Tuberkulose i nervesystemet *d. & n.* 14
Tuberkulose i Urinorganerne *d. & n.* 20
Tuberkulöser Knochenfrass *all.* 17*a*
Туберкулоза / Tuberkuloza *serb.* 13*c*
Туберкулоза црева / Tuberkuloza creva *serb.* 15*(a)*
Туберкулоза на дихателнитѣ органи / Tuberkuloza na dikhatelnite organi *bulg.* 13*b*
Туберкулоза [Галопирајућа —] / Tuberkuloza [Galopirajuća —] *serb.* 22*a*
Туберкулоза хрптењаче / Tuberkuloza hrptenjače *serb.* 16
Туберкулоза кичме / Tuberkuloza kičme *serb.* 16
Туберкулоза костију / Tuberkuloza kostiju *serb.* 17*a*
Туберкулоза коже / Tuberkuloza kože *serb.* 18
Туберкулоза мокраћних органа / Tuberkuloza mokraćnih organa (Tbk. mokrachnich organa) *serb.* 20

Туберкулоза плућа / Tuberkuloza pluća } *serb.* 13*b(c)*
Туберкулоза зглобова / Tuberkuloza zglobova } *serb.* 17*b*
Туберкулоза жљезда / Tuberkuloza žljezda } *serb.* 19
Tuberkuloze [Elpošanas organu —] *lett.* = *Tuberculosis organum respirationis* 13
Tuberkuloze [Kvepuoj. org. —] *lith.* 13
Tuberkuloze [Plaušu —] *lett.* = *Tuberculosis pulmonum* 13*b(c)*
Tüdocsúcshurut *hong.* 13*b(c)*
Tüdőgümőkór *hong.* 13*b(c)*
Tüdőgyulladás *hong.* 109
Tüdőgyulladás [Hurutos —] *hong.* 107
Tüdőgyulladás [Lebenyes vagy rostonyás —] *hong.* 108
Tüdőhurut *hong.* 107
Tüdővész *hong.* 13*b(c)*
Tuhkarokko *fin.* 35
Tularæmi *d. & n.* 26*b*
Tularæmia *angl. lat.* 26*b*
Tularaemie *néer.* 26*b*
Tularämie *all.* 26*b*
Tularemi *suéd.* 26*b*
Tularemia *esp. it. pol. port.* 26*b*
Туларемия / Tularemia } *rus.* 26*b*
Tularémie *fr. roum.* 26*b*
Tulirokko *fin.* 8
Tumeur blanche *fr.* 17*b*
Tumór branco *port.* 17*b*
Tumor flegmonoso *esp.* 152
Tumor hydatico do figado *port.* 41*a*
Tumore blanco *esp.* 17*b*
Tumori bianchi *it.* 17*b*
Turbare *roum.* 38*b*
Tuse convulsivă *roum.* 9
Tussis convulsiva *all. angl. lat. néer.* 9
Tüüfus [Tähniline —] *est.* 39*a*
Tuulerõuged *est.* 34*b*
Tyf břisni *tch.* 1
Tyf skrvrnitý *tch.* 39*a*
Tyf střevní *tch.* 1
Tyf [Vracející se —] *tch.* 31*a*
Tyf vratný *tch.* 31*a*
Tyfoid *suéd.* 1
Tyfoid-feber *d. & n.* 1
Tyfus brzusny *pol.* 1
Tyfus [Eksantematisk —] *dan.* 39*a*
Tyfus endemic benin (murin) *roum.* 39*b(a)*
Tyfus osutkowy *pol.* 39*a*
Tyfus plamisty *pol.* 39*a*
Tyfus powrotny *pol.* 31*a*
Tymai *lith.* 35
Typheuze koorts *néer.* 1
Typhlite *fr. port.* 121
Typhlitis *angl. lat. néer.* 121
Typho endemico de São Paulo *port.* 39*b(cd)*
Typho exanthematico *port.* 39*a*
Typho exanthematico de São Paulo *port.* 39*b(cd)*
Typho recurrente *port.* 31*a*
Typho de S. Paulo (por carrapatas) *esp.* 39*b(cd)*
Τυφοειδὴς πυρετὸς / Typhoeides pyretos } *gr.* 1
Typhoid *néer.* 1
Typhoid fever *angl.* 1
Typhoïde [Fièvre —] *fr.* 1
Τυφος [Κοιλιακος —] / Typhos [Koiliakos —] } *gr.* 1
Typhus* *all. lat. néer.* 1
Typhus [Abdominal —]* *angl.* 1
Typhus abdominal* *fr.* 1
Typhus abdominalis *all. néer.* 1
Typhus à acariens (à *R. orientalis*) *fr.* 39*b(b)*
Typhus amaril *fr.* 38*a*
Typhus [Benign endemic —] *angl.* 39*b(a)*
Typhus classique *fr.* 39*a*
Typhus endemicus benignus *lat.* 39*b(a)*
Typhus endemicus murinus *lat.* 39*b(a)*
Typhus endémique bénin *fr.* 39*b(a)*
Typhus endémique bénin à puces (murin) à *R. mooseri* *fr.* 39*b(a)*
Typhus [Epidemic —] *angl.* 39*a*

Typhus épidémique *fr.* 39*a*
Typhus européen *fr.* 39*a*
Typhus [Exanthematic —] *angl.* 39*a*
Typhus exanthematicus *all. lat. néer.* 39*a*
Typhus exanthématique (à poux) *fr.* 39*a*
Typhus [Flea-borne —] *angl.* 39*b(a)*
Typhus fever [Louse-borne —] *angl.* 39*a*
Typhus historique *fr.* 39*a*
Typhus mexicain *fr.* 39*b(a)*
Typhus [Mexican —] *angl.* 39*b(a)*
Typhus murin *fr.* 39*b(a*
Typhus [Murine —] *angl.* 39*b(a)*
Typhus [Muriner —] *all.* 39*b(a)*
Typhus pétéchial *fr.* 39*a*
Typhus petechialis *all. lat.* 39*a*
Typhus [Rat-flea —] *angl.* 39*b(a)*
Typhus recurrens *all. lat.* 31*a*
Typhus recurrens obermeieri *lat.* 31*a*
Typhus récurrent *fr.* 31*a*
Typhus Sancti Pauli *lat.* 39*b(cd)*
Typhus [São Paulo tick —] *angl.* 39*b(cd)*
Typhus [Shop —] (Malaya) *angl. néer.* 39*b(a)*
Typhus [Scrub —] (Malaya) *angl.* 39*b(b)*
Typhus [Scrub XK —] *néer.* 39*b(b)*
Typhus à tiques de São Paulo *fr.* 39*b(cd)*
Typhus à tiques sud-africain *fr.* 39*b(cb)*
Typhus [Tropical —] *néer.* 39*b(b)*
Typhus tropical de brousse *fr.* 39*b(b)*
Typhus [Tropical jungle —] *angl.* 39*b(b)*
Typhus tropical urbain *fr.* 39*b(a)*
Typhus [Tropische —] (Sumatra) *néer.* 39*b(b)*

U

U. *(angl.)* = unqualified, unspecified
= non spécifié
= *non specificatus*

u.o. *(dan.)* = uden oplysning
u.o. *(nor.)* = uten oplysning
= sans explication
= without precision
= *non specificatus*
Übertragbare Gelbsucht *all.* 32*a*
Úbyt míchy *tch.* 30*a*
Úbytě *tch.* 13*c*
Uhlák *tch.* 7*c*
Ujszülottek bubor [Az —] *hong.* 161
Ulcer fagedenic *roum.* 29*(c)*
Ulcer [Phagedenic —] *angl.* 29*(c)*
Ulcera bacilar *esp.* 18
Ulcera de Bauru *port.* 29*(bc)*
Ulcera dura *it.* 30*d*
Ulcera fagedenica *esp. it.* 29*(c)*
Ulcera infettante *it.* 30*d*
Ulcera molle *it.* 44*a(a)*
Ulcera phagedenica *port.* 29*(c)*
Ulcera semplice *it.* 44*a(a)*
Ulcera tubercolare *it.* 18
Ulcera tuberculosa *esp.* 18
Ulcera venerea *it.* 44*a(a)*
Ulcère bacillaire *fr.* 18
Ulcère des pays chauds *fr.* 29*(c)*
Ulcère phagédénique *fr.* 29*(c)*
Ulcus durum *lat.* 30*d*
Ulcus molle *all. lat. néer.* 41*a(a)*
Ulcus phagedænicum *lat.* 29*(c)*
Üldine halvatus *est.* 30*b*
Üldine paralüüs *est.* 30*b*
Umferdargula *isl.* 44*d(g)*
Uminis nugaros *lith.* 36
Умирания / Umirania *bulg.* = *Mortui*
Умрели / Umreli *bulg.* = *Mortui*
Умрли(о) / Umrli(o) *serb.* = *Mortuus, i*
Uncinariasis *angl. esp.* 40
Uncinariose *fr.* 40
Uncinariosi *it.* 40
Undulantfeber *suéd.* 5*b*
Undulant fever *angl.* 5*c*
Unduleeruv palavik (Bang) *est.* 5*b*
Unehaigus *est.* 37*c*
Unitauti *fin.* 37*c*
Unterleibstyphus *all.* 1

Uplavice *tch.* 27*c*
Uplavice střevní bacilarní *tch.* 27*a*
Uplavice střevní amœbová *tch.* 27*b*
Ὑπόστροφοι πυρετοι / Upostrophoi pyretoi *gr.* 31*a*
Uréthrite *fr.* 25*(a)*
Urethritis gonorrhoica *lat. néer.* 25*(a)*
Urethritis venerea *néer.* 25*(a)*
Uretrite blenorragica *it.* 25*(a)*
Uretrite gonococcica *it.* 25*(a)*
Uretritt u.o. *nor.* 25*(a)*
Uro-genìtaalne tuberkuloos *est.* 20
Urogenitaltuberkulose *d. & n.* 20
Уш / Uš *serb.* = *Pediculus* 153*(a)*
Uscare *roum.* 13*c*
Ussjätkepõletik *est.* 121
Uta *esp.* 29*(b)*
Utu slimiba *lett.* 153*(a)*
Uyuz *turc* 153*(b)*
Užkretimas [Kraujo —] *lith.* 24*a*

V

Vaginite s.a.i. *fr. it.* 25*(a)*
Vaginite s.o.i. *esp.* 25*(a)*
Vaginitis gonorrhoica *lat.* 25*(a)*
Vahamere-palavìk *est.* 5*a*
Vaïke-aasias *est.* 42*(e)*
Vaiolo *it.* 34*a*
Vaioloide (v) *it.* 34*b*
Vaiuolo *it.* 34*a*
Vaiuoloide *it.* 34*b*
Vakbélgyulladás *hong.* 121
Váltóláz *hong.* = *Febris intermittens* 28*d*
Vannkopper *d. & n.* 38*e*
Вардарска грозница / Vardarska groznica *serb.* 38*f(b)*
Varfeber *suéd.* 24*a*
Varicela *esp. port. roum.* 38*e*
Varicella *all. angl. hong. it. lat. néer. port.* 38*e*
Varicelle *fr.* 38*e*
Variceller *dan.* 38*e*
Variola *lat. port. roum.* 34*c*
Variola hemorrhagica *lat.* 34*a*
Variola major *angl. fr. lat. néer. roum.* 34*a*
Variola minor *all. angl. fr. lat. néer. roum.* 34*b*
Variola vera *all. lat.* 34*a*
Variole *fr.* 34*c*
Вариолоид / Varioloïd *rus.* 34*b*
Varioloide *esp.* 34*b*
Variolois *all. néer.* 34*b*
Varizellen *all.* 38*e*
Vărsat *roum.* 34*c*
Ваш / Vaš *serb.* = *Pediculus* 153*(a)*
Вашљивост / Vašljivost *serb.* 153*(a)*
Vastagbélhurut *hong.* = *Colitis* 119, 120*a*
Vattenkoppor *suéd.* 38*e*
Vattuskräck *suéd.* 38*b*
Вшивость (вошь) / Vchivost (voch) *rus.* 153*(a)*
Veba *turc* 3*c*
Vēdera tifs *lett.* 1
Vejaraupiai *lith.* 38*e*
Vekonybélhurut *hong.* = *Enteritis* 119, 120*a*
Велике богиње / Velike boginje *serb.* 34
Велике оспице / Velike ospice *serb.* 34*a*
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Венерический лимфогранулом / Veneritcheskiï limfogranulom *rus.* 44*a(b)*
Ver de Guinée *fr.* 42*(e)*
Ver de Médine *fr.* 42*(e)*
Ver solitaire *fr.* 42*(g)*
Verbaj *hong.* 30*d*
Vere katk *est.* 3*a(b)*
Вред / Vered (v) *rus.* 151
Verem *turc* 13*b(c)*
Veremi [Amudi fukari —] *turc* 16
Veremi [Barsak —] *turc* 15*(a)*
Veremi [Cilt —] *turc* 18

Veremi [Kemik —] *turc* 17*a*
Verenmyrkytys *fin.* 24*a*
Vérhas *hong.* 27*d*
Vérhas [Amœbas —] *hong.* 27*b*
Vérhas [Bacillusos —] *hong.* 27*a*
Verine kõhutõbi *est.* 27*d*
Верхушечная пневмония / Verkhuchetchnaïa pnevmonia *rus.* 13*b(c)*
Verkoudheid (v) *néer.* 106*a*
Verkoudheid in 't hoofd *néer.* 104*a*
Verlausung *all.* 153*(a)*
Verme solitario (v) *it.* 42*(g)*
Vérmérgezés *hong.* 24*a*
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Vermergezes [Gyermekagyi —] *hong.* 147
Vermes n. s. *lat.* 42*(j)*
Vermi intestinali *it.* 42*(j)*
Verminatio n. s. *lat.* 42*(j)*
Verőértagulás *hong.* = *Aneurysma* 30*c*
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Verruga peruana *esp. port.* 29*(f)*
Verruga peruviana *all. fr.* 29*(f)*
Vers intestinaux* *fr.* 42*(j)*
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Vertebral caries *angl.* 16
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Ветар [Црвени —] / Vetar [Crveni —] *serb.* 11
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Ветряная оспа / Vetrianaïa ospa *rus.* 38*e*
Везвратенъ тифъ / Vezvraten tif *bulg.* 31*a*
Vibrion septique [Infection à —] *fr.* 24*c*
Viduriavimas *lith.* = *Diarrhœa* 119, 120*a*
Viduriu šiltiné *lith.* 1
Vierde ziekte *néer.* 44*d(e)*
Viertagefieber *all.* 28*b*
Vierte (exanthematische) Krankheit *all.* 44*d(e)*
Vihurirokko *fin.* 38*d*
Vijfdaagsche koorts *néer.* 39*c(a)*
Vilutauti *fin.* 28*d*
Vincent Angina *d. & n.* 32*b(a)*
Vincent [Angina de —] *port.* 32*b(a)*
Винцента [Жаба —] / Vintsenta [Jaba —] *rus.* 32*b(a)*
Viruela *esp.* 34*c*
Viruela mayor *esp.* 34*a*
Viruela menor *esp.* 34*b*
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Vlektyphus [Tropische —] (Sumatra) *néer.* 39*b(b)*
Вошь / Voch *rus.* 153*a*
Vodnatky *tch.* 38*e*
Vodní [Neštovice —] *tch.* 38*e*
Водобоязнь / Vodoboïazn *rus.* 38*b*
Волчанка / Voltchanka *rus.* 18
Vomito negro (v) *esp. fr.* 38*(a)*
Voorhoofdsholteontsteking *néer.* = *Sinusitis frontalis* 104*b(a)*
Vörheny *hong.* 8
Воспаление дыхательного горла / Vospalenié dykhatelnogo gorla *rus.* 106
Воспаление [Эпидемическое] / Vospalenié [Epidemitcheskoié —] *rus.* 44*d(c)*
Воспаление глаза [Гнойное —] / Vospalenié glasa [Gnoïnoe —] *rus.* 25*b*
Воспаление костного мозга / Vospalenié kostnogo mozga *rus.* 154
Воспаление легких / Vospalenié lekhkikh (v) *rus.* 108

Воспаление лобной пазухи / Vospalenié lobnoï pazukhi } *rus.* 104*b*
Воспаление мозговых оболочек / Vospalenié mozgovykh obolotchek } *rus.* 81
Воспаление плевры [Эксудативное —] / Vospalenié plevry [Eksudativnoe—] } *rus.* 110
Воспаление плевры [Гнойное —] / Vospalenié plevry [Gnoïnoe —] } *rus.* 110*a*
Воспаление рта / Vospalenié rta } *rus.* 115*a(a)*
Воспаление слепой кишки / Vospalenié slepoï kichki } *rus.* 121
Воспаление уха / Vospalenié ukha } *rus.* 89*(a)*
Возвратныя лихорадки / Vozvratnyia likhoradki } *rus.* 31
Возвратный тиф б. у. / Vozvratnyï tif b.u. } *rus.* 31*c*
Возвратный тиф [Эпидемический —] / Vozvratnyï tif [Epidemitcheskiï —] } *rus.* 31*a*
Возвратный тиф [Вшивый —] / Vozvratnyï tif [Vchivyï —] } *rus.* 31*a*
Vracející se tyf *tch.* 31*a*
Vrativka *tch.* 31*a*
Vratny tyf *tch.* 31*a*
Вратобоља / Vratobolja } *serb.* 115*c(b)*
Vřed [Měkky —] *tch.* 44*a(a)*
Врожденный сифилис / Vrojdennyï sifilis } *rus.* 30*d(a)*
Všivina *tch.* 153*(a)*
Vulvite s.a.i. *fr.* 25*(a)*
Vuorokuume *fin.* 28*d*
Výdut aorty *tch.* 30*c*
Výdut tepen *tch.* 30*c*

W

Wąglik *pol.* 7*c*
Warm abscess *néer.* 152
Warzenfortsatz-Entzündung *all.* 89*b(a)*
Wasserpocken *all.* 38*e*
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Waterpokken *néer.* 38*e*
Wechselfieber (v) *all.* 28*d*
Wechselfieber [Tägliches —] *all.* 28*c*
Weichselbaum [Molestia de —] *port.* 6
Weigl [Maladie de —] *fr.* 39*c(b)*
Weil [Boala lui —] *roum.* 32*a*
Weil [Enfermedad de —] *esp.* 32*a*
Weil [Maladie de —] *fr.* 32*a*
Weil [Morbo di —] *it.* 32*a*
Weil [Ziekte van —] *néer.* 32*a*
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Weil'sche Krankheit *all.* 32*a*
Weil's disease *angl.* 32*a*
Weils sjukdom *suéd.* 32*a*
Weilsyge *dan.* 32*a*
Weilsyke *nor.* 32*a*
Whooping cough *angl.* 9
Wiewiór *pol.* 25*(a)*
Windpocken (v) *all.* 38*e*
Wolhynie [Fièvre de —] *fr.* 39*c(a)*
Wolhynisches Fieber *all.* 39*c(a)*
Волынская лихорадка / Wolynskaïa likhoradka } *rus.* 39*c(a)*
Wormgaten *néer.* 153*(b)*
Worms *angl.* 42
Wormziekte *néer.* 42*(j)*
Wrzód mieki *pol.* 44*a(a)*
Wszawica *pol.* 153*(a)*
Wundstarrkrampf *all.* 12
Wurmkrankheit (v) *all.* 42*(j)*
Wurmkrankheit der Bergleute (v) *all.* 40
Wut *all.* 38*b*

X

X... en caractères cyrilliques, *cf.* Kh...
X... in Cyrillic characters, *cf.* Kh...

Y

Y... en caractères cyrilliques, *cf.* U...
Y... in Cyrillic characters, *cf.* U...
Yaws *angl. néer.* 32*b(c)*
Yellow fever *angl.* 38*a*
Yilancik *turc* 11
Yochubio *japon.* 39*b(b)*

Z

z.n.a. *(néer.)* = zonder nadere aanduiding
= sans indication plus précise
= without more precise indication
= *non specificatus*
Zádušný [Kašel —] *tch.* 9
Zajikavy kašel *tch.* 9
Zakażenie ropne i posocznicze (nieporodowe) *pol.* 24
Zakazne ronienie krow *pol.* 5*b*
Zánět červikovitého přívěsku *tch.* 121
Zánět dřeně kostní *tch.* 154
Zánět dutiny... čelní *tch.* = *Sinusitis... frontalis* 104*b(a)*
Zánět hrtanu *tch.* 105*(a)*
Zánět mozku [Epidemický —] *tch.* 37*c*
Zánět mozku [Lethargický —] *tch.* 37*c*
Zánět nitroblány srdeční [Prudký bacilarní —] *tch.* 91*a*
Zánět plen mozkových a míšnich [Epidemicky —] *tch.* 6
Zánět plen mozkových [Prostý —] *tch.* 81*a*
Zánět plic *tch.* 109
Zánět plic [Chřipkový —] *tch.* 33*a*
Zánět plic [Katarralni —] *tch.* 107
Zánět plic [Kruposni —] *tch.* 108
Zánět plic [Lalokový —] *tch.* 107
Zánět plic [Lalůčkovy —[*tch.* 107
Zánět podkožního vazina *tch.* 152
Zánět pohrudnice *tch.* 110*b*
Zánět pohrudnice hnisavý *tch.* 110*a*
Zánět pohrudnice suchý *tch.* = *Pleuritis sicca* 110*b*
Zánět přiušnice *tch.* 44*c*
Zánět průdušek *tch.* 106*c*
Zánět průdušek prudký *tch.* 106*a*
Zánět průdušek vleklý *tch.* 106*b*
Zánět průdušinek *tch.* = *Bronchitis capillaris* 107
Zánět očí novorozenců *tch.* 25*(b)*
Zanět šedé hmoty mišni [Prudký —] *tch.* 36
Zánět šedé hmoty mozkové [Prudký —] *tch.* = *Polioencephalitis acuta* 36
Zánět střev *tch.* 119, 120*a*
Zánět ušní *tch.* 89*a(a)*
Zánět úst *tch.* 115*a(a)*
Záněty stredoušni *tch.* 89*a(a)*
Zapalení střev [Choleroidní —] *tch.* 119, 120*a*
Zapalenie dziąseł *pol.* = *Gingivitis* 115*a(a)*
Zapalenie gardła *pol.* 115*c(b)*
Zapalenie jamy ustnej *pol.* = *Stomatitis* 115*a(a)*
Zapalenie jelita grubego *pol.* = *Inflammatio coli* 119, 120*a*
Zapalenie kiszek *pol.* = *Enteritis* 119, 120*a*
Zapalenie krtani *pol.* 105*(a)*
Zapalenie miesni [Epidemiczne —] *pol.* 44*d(c)*
Zapalenie migdałków [Bakteryjne —] *pol.* 115*b*
Zapalenie mózgu *pol.* 37*c*
Zapalenie mózgu ze śpiączką *pol.* 37*c*
Zapalenie opłucnej *pol.* 110
Zapalenie opłucnej [Ropne —] *pol.* 110*a*
Zapalenie opon mózgowych nagminne [Epidemiczne —] *pol.* 6
Zapalenie opon mózgowych zwykłe *pol.* 81*a*
Zapalenie oskrzeli *pol.* 106*c*
Zapalenie płuc *pol.* 107, 109
Zapalenie płuc [Płatowe —] *pol.* 108
Zapalenie przyusznicy *pol.* 44*c*
Zapalenie reumatyczne stawów *pol.* 58
Zapalenie rogów przednich rdzenia *pol.* 36

Zapalenie szipiku kostnego *pol.* 154
Zapálení střev *tch.* 119, 120*a*
Zapalenie ucha środkowego *pol.* 89*a(a)*
Zapalenie wsierdzia [Ostre, Bakt. —] *pol.* 91*a*
Zapalenie wsierdzia [Podostre —] wywolane gronkowcem zieleniejacym *pol.* 91*b*
Zapalenie wyrostka robaczkowego *pol.* 121
Zapalenie wyrostka sutkowego *pol.* 89*b(a)*
Zapalenie zatok czołowych *pol.* = *Sinusitis frontalis* 104*b(a)*
Zapalenie zatok szczekowych *pol.* = *Sinusitis maxillaris* 104*b(a)*

Запаљење костне сржи [Акутно —] / Zapaljenje kostne srži [Akutno —] *serb.* 154
Запаљење мозга / Zapaljenje mozga *serb.* 6
Запаљење мозга [Туберкулозно —] / Zapaljenje mozga [Tuberkulozno —] *serb.* 14*a*
Запаљење очију новородјених [Гнојно—] / Zapaljenje ociju novorodjenikh [Gnoino —] *serb.* 25*(b)*
Запаљење плуђа / Zapaljenje pluća *serb.* 109
Запаљење плуђа [Бронхично —] / Zapaljenje pluća [Bronhično —] *serb.* 107
Запаљење плуђне марамице / Zapaljenje plućne maramice *serb.* 110*b*
Запаљење плуђне марамице [Гнојно —] / Zapaljenje plućne maramice [Gnojno —] *serb.* 110*a*
Запаљење слепог црева / Zapaljenje slepog creva *serb.* 121
Запаљење средњега ува / Zapaljenje srednjega uva *serb.* 89*a(a)*
Заражение [Гнойное —] / Zarajenie [Gnoïnoe —] *russ.* 24
Заражење [Гнојно —] / Zaraženje [Gnojno —] *serb.* 24*b*
Заражење [Септично —] / Zaraženje [Septično —] *serb.* 24*a*
Заразна жутица / Zarazna žutica *serb.* 44*d(g)*
Заразне / Zarazne *serb.* = *contagiosus, a, um*
Заразне болести / Zarazne bolesti *serb.* = *Morbi contagiosi*
Заразни / Zarazni *bulg.* = *contagiosus, a, um*
Žarnu uždegimas *lith.* = *Enteritis* 119, 120*a*
Záškrt *slovaque, tch.* 10
Zatuccem *turc* 110*b*
Zatürree *turc* 109
Заушка / Zauchka *bulg.* 44*c*
Заушница / Zauchnitsa *rus.* 44*c*
Заушњаци / Zaušnjaci *serb.* 44*c*
Zellgewebeentzündung *all.* 152
Зглобни реуматизам / Zglobni reumatizam *serb.* 58
Zgorzel gazowa *pol.* 24*c*
Ziarnica złośliwa *pol.* 44*b*
Ziegenpeter *all.* 44*c*
Zimnica *pol.* 28*d*
Zimnica tropikalna *pol.* 28*c*
Zimnica czwartaczka *pol.* 28*b*
Zimnica trzeciaczka *pol.* 28*a*
Zimnice malarická *tch.* 28*d*
Злокачественная язва / Zlokatchestvennaïa iazva *rus.* 7*a*
Злокачественная малярия / Zlokatchestvennaïa malaria *rus.* 28*c*
Žloutenka [Epidemická —] *tch.* 44*d(g)*
Žloutenka [Infekční —] *tch.* 32*a*
Žloutenka kataralní *tch.* 127
Žlutý mor *tch.* 38*a*

Quatrième Partie

Quelques pays employant, pour certaines statistiques tout au moins, non pas la Nomenclature internationale détaillée, mais l'intermédiaire ou l'abrégée, nous donnons à la Table 1 la correspondance entre le contenu des rubriques de ces trois nomenclatures.

Nous donnons en outre (Table 2) la correspondance entre les rubriques de la Nomenclature détaillée de 1938 et celle de 1929 (4e revision) qui la précéda et qui peut être utile pour l'interprétation des statistiques de la période 1931-1940, ou de celles de quelques pays ayant tardé, du fait de la guerre, à adopter la nomenclature nouvelle.

Pour la même raison, nous reproduisons (Table 3) la série des concordances entre les trois Nomenclatures détaillée, intermédiaire et abrégée de 1929.

L'emploi simple d'une de ces tables ou leur emploi combiné doit donc permettre de savoir à quelles maladies on a affaire dans les rubriques de toutes les statistiques basées sur les Nomenclatures internationales pour la période 1931 à 1950.

Part IV

As some countries use, at least for certain statistics, the Intermediate or the Abridged International Lists, instead of the detailed list, we show in Table 1 the correspondence between the content of the headings in these three lists.

We show also, in Table 2, the correspondence between the headings in the International Detailed List of 1938 and in that of 1929 (4th revision) which preceded it. This may be useful for interpreting statistics of the period 1931-1940, or statistics of some countries which, owing to the war, have delayed their adoption of the new list.

For the same reason, we reproduce in Table 3 the Detailed, Intermediate and Abridged Lists of 1929, with their correspondence.

The use of one, or the combined use of several, of these tables will enable the reader to know what diseases are covered by the headings of all statistics based on the International Lists for the period 1931-1950.

Table 1.

Correspondance des numéros des Nomenclatures Détaillée, Intermédiaire et Abrégée de 1938.

N. B. — Un numéro précédé de la lettre *p* désigne une *partie* seulement de la rubrique de ce numéro.

Correspondence between the Numbers of the Detailed, Intermediate, and Abridged Lists of 1938.

N. B. — The letter "*p*" in italics preceding a number means *part* of that number.

d	i	a	d	i	a	d	i	a	d	i	a
1	*p.* 1	*p.* 1	**51**	*p.* 23	*p.* 15	**101**	*p.* 48	*p.* 25	**151**	*p.* 73	*p.* 37
2	*p.* 1	*p.* 1	**52**	*p.* 23	*p.* 15	**102**	*p.* 48	*p.* 25	**152**	*p.* 73	*p.* 37
3	2	2	**53**	*p.* 23	*p.* 15	**103**	*p.* 48	*p.* 25	**153**	*p.* 73	*p.* 37
4	*p.* 17	*p.* 14	**54**	*p.* 23	*p.* 15	**104**	*p.* 52	*p.* 28	**154**	*p.* 74	*p.* 37
5	*p.* 17	*p.* 14	**55**	*p.* 23	*p.* 15	**105**	*p.* 52	*p.* 28	**155**	*p.* 74	*p.* 37
6	*p.* 17	*p.* 14	**56**	*p.* 24	*p.* 16	**106**	49	26	**156**	*p.* 74	*p.* 37
7	*p.* 17	*p* 14	**57**	*p.* 24	*p.* 16	**107**	*p.* 50	*p.* 27	**157**	75	*p.* 38
8	3	3	**58**	25	*p.* 20	**108**	*p.* 50	*p.* 27	**158**	76	*p.* 38
9	4	4	**59**	*p.* 26	*p.* 17	**109**	*p.* 50	*p.* 27	**159**	77	*p.* 38
10	5	5	**60**	*p.* 26	*p.* 17	**110**	51	*p.* 28	**160**	78	*p.* 38
11	*p.* 17	*p.* 14	**61**	27	18	**111**	*p.* 52	*p.* 28	**161**	79	*p.* 38
12	*p.* 17	*p.* 14	**62**	*p.* 29	*p.* 20	**112**	*p.* 52	*p.* 28	**162**	80	39
13	6	6	**63**	28	*p.* 20	**113**	*p.* 52	*p.* 28	**163**	*p.* 81	*p.* 40
14	*p.* 7	*p.* 7	**64**	*p.* 29	*p.* 20	**114**	*p.* 52	*p.* 28	**164**	*p.* 81	*p.* 40
15	*p.* 7	*p.* 7	**65**	*p.* 29	*p.* 20	**115**	*p.* 60	*p.* 32	**165**	*p.* 82	*p.* 41
16	*p.* 7	*p.* 7	**66**	*p.* 29	*p.* 20	**116**	*p.* 60	*p.* 32	**166**	*p.* 82	*p.* 41
17	*p.* 7	*p.* 7	**67**	*p.* 30	*p.* 20	**117**	53	*p.* 32	**167**	*p.* 82	*p.* 41
18	*p.* 7	*p.* 7	**68**	*p.* 30	*p.* 20	**118**	*p.* 60	*p.* 32	**168**	*p.* 82	*p.* 41
19	*p.* 7	*p.* 7	**69**	*p.* 30	*p.* 20	**119**	54	*p.* 29	**169**	*p.* 84	*p.* 43
20	*p.* 7	*p.* 7	**70**	*p.* 30	*p.* 20	**120**	55	*p.* 29	**170**	83	42
21	*p.* 7	*p.* 7	**71**	*p.* 30	*p.* 20	**121**	56	30	**171**	*p.* 84	*p.* 43
22	*p.* 7	*p.* 7	**72**	*p.* 32	*p.* 20	**122**	57	*p.* 32	**172**	*p.* 84	*p.* 43
23	*p.* 17	*p.* 14	**73**	31	*p.* 20	**123**	*p.* 60	*p.* 32	**173**	*p.* 84	*p.* 43
24	8	*p.* 14	**74**	*p.* 32	*p.* 20	**124**	58	*p.* 31	**174**	*p.* 84	*p.* 43
25	*p.* 17	*p.* 14	**75**	*p.* 32	*p.* 20	**125**	*p.* 59	*p.* 31	**175**	*p.* 84	*p.* 43
26	*p.* 17	*p.* 14	**76**	*p.* 32	*p.* 20	**126**	*p.* 59	*p.* 31	**176**	*p.* 84	*p.* 43
27	9	*p.* 14	**77**	33	19	**127**	*p.* 59	*p.* 31	**177**	*p.* 84	*p.* 43
28	10	8	**78**	*p.* 34	*p.* 20	**128**	*p.* 60	*p.* 32	**178**	*p.* 84	*p.* 43
29	*p.* 17	*p.* 14	**79**	*p.* 34	*p.* 20	**129**	*p.* 60	*p.* 32	**179**	*p.* 84	*p.* 43
30	11	9	**80**	*p.* 40	*p.* 23	**130**	*p.* 61	*p.* 33	**180**	*p.* 84	*p.* 43
31	*p.* 17	*p.* 14	**81**	35	*p.* 21	**131**	*p.* 61	*p.* 33	**181**	*p.* 84	*p.* 43
32	*p.* 17	*p.* 14	**82**	36	*p.* 21	**132**	*p.* 61	*p.* 33	**182**	*p.* 84	*p.* 43
33	12	10	**83**	37	22	**133**	62	*p.* 34	**183**	*p.* 84	*p.* 43
34	13	11	**84**	38	*p.* 23	**134**	63	*p.* 34	**184**	*p.* 84	*p.* 43
35	14	12	**85**	39	*p.* 23	**135**	64	*p.* 34	**185**	*p.* 84	*p.* 43
36	*p.* 17	*p.* 14	**86**	*p.* 40	*p.* 23	**136**	65	*p.* 34	**186**	*p.* 84	*p.* 43
37	*p.* 17	*p.* 14	**87**	*p.* 40	*p.* 23	**137**	66	*p.* 34	**187**	*p.* 84	*p.* 43
38	*p.* 17	*p.* 14	**88**	*p.* 41	*p.* 23	**138**	*p.* 67	*p.* 34	**188**	*p.* 84	*p.* 43
39	15	13	**89**	*p.* 41	*p.* 23	**139**	*p.* 67	*p.* 34	**189**	*p.* 84	*p.* 43
40	*p.* 16	*p.* 14	**90**	42	*p.* 24	**140**	70	*p.* 35	**190**	*p.* 84	*p.* 43
41	*p.* 16	*p.* 14	**91**	*p.* 46	*p.* 24	**141**	69	*p.* 36	**191**	*p.* 84	*p.* 43
42	*p.* 16	*p.* 14	**92**	43	*p.* 24	**142**	*p.* 68	*p.* 36	**192**	*p.* 84	*p.* 43
43	*p.* 17	*p.* 14	**93**	44	*p.* 24	**143**	*p.* 68	*p.* 36	**193**	*p.* 84	*p.* 43
44	*p.* 17	*p.* 14	**94**	45	*p.* 24	**144**	*p.* 68	*p.* 36	**194**	*p.* 84	*p.* 43
45	18	*p.* 15	**95**	*p.* 46	*p.* 24	**145**	*p.* 68	*p.* 36	**195**	*p.* 84	*p.* 43
46	19	*p.* 15	**96**	*p.* 48	*p.* 25	**146**	*p.* 72	*p.* 36	**196**	*p.* 85	*p.* 43
47	20	*p.* 15	**97**	*p.* 47	*p.* 25	**147**	71	*p.* 35	**197**	*p.* 85	*p.* 43
48	21	*p.* 15	**98**	*p.* 47	*p.* 25	**148**	*p.* 72	*p.* 36	**198**	86	*p.* 43
49	*p.* 23	*p.* 15	**99**	*p.* 48	*p.* 25	**149**	*p.* 72	*p.* 36	**199**	*p.* 87	*p.* 44
50	22	*p.* 15	**100**	*p.* 48	*p.* 25	**150**	*p.* 72	*p.* 36	**200**	*p.* 87	*p.* 44

Correspondance approximative entre les numéros des Nomenclatures détaillées de 1929 et 1938.

N. B. — Le signe plus (+) indique que le contenu de la rubrique de 1938 est plus grand que celui de la rubrique correspondante de 1929 ; le signe ± indique que le contenu de la rubrique a été modifié par addition et soustraction ; un numéro précédé de la lettre p. désigne une partie seulement de la rubrique de ce numéro ; un numéro suivi de la lettre N désigne une rubrique nouvelle.

Approximate Correspondence between the Numbers of the Detailed Lists for 1929 and 1938.

N. B. — The plus sign (+) indicates that the 1938 heading is more comprehensive than the corresponding one of 1929 ; the sign ± indicates that the contents of the heading have been altered by adding to it and also by substracting from it. A number preceded by the letter "p." denotes part only of the heading under that number ; a number followed by the letter "N" denotes a new heading.

1929	1938
1	**1**
2	**2**
3	**39**
4	**31**
5	**5**
6	**34**
7	**35**
8	**8**
9	**9**
10	**10**
11	**33**
12	**4**
13	**27**
a	*b*
b	*a*
c	*c*, *d*
14	**3**
15	**11**
16	**36**
17	**37**
18	**6**
19	**26** *a*
20	**7**
21	**38** *b*N
22	**12**
23	**13**
24	**14**
25	**15**
26	**16**
27	**17**
28	**18**
29	**19**
30	**20**
31	**21**
32	**22**
33	**23**
34	**30** +
a	*da*
b, *c*	*db*, *dc*, *dd*
35	**25**, **44** *a*N
36	**24** +
a	*a*
36 *b*	**24** *b*
c	*c*
-	*d*N
37	**38** *a*N
38	**28** +
39	p.**29**, **32**N
40	**40**
41	**41**
42	**42**
43	**43**
44	**44** ±, **26** *b*, *c*, **28** p.*c*, p **29**, **38** *d*, *e*, *f*, **39** *b*, *c*, **44** *c*, *d*, **195** *a*
45	**45**
46	**46**
a	*a*
b	*b*
c	*d*
d	*e*
e	*f*
f	*g*
g	*c*N, *h*
47	**47**
48	**48**
49	**49**
50	**50**
51	**51**, p.**52**
52	**53**
53	p.**52**, **54**, p.**55**
54 *a*	**56** *a*, *b*, *c*
b	*d*N, *e*
55 *a*	**57** *a*, *b*, *c*
b	*d*N, *e*
56	**58**
57	**59**
58	**60**
59	**61**
60	**67**
61	**68**
62	**69**
63	**70**
64	**66** *a*
65	**62**
66	**63**
67	**64**
68	**65**
69	**66** *b*, **71**N
70	**72**
a	*a*
b	*b*
-	*c* N
71	**73** +
a	*a*
b	*b*, *c*, *d*, **75** *a*
72	**74** +
a	*a*
b	*b*, **44** *b*N
73	**75** *b*, *c*
74	**76**
75	**77**
76	p.**79**
77	**78**N, p.**79**
78	**80**
79	**81**
80	**30** *a*
81	**82**
82	**83**
a	*a*
b	*b*, *c*
c	*d*
-	*e* N
83	**30** *b*
84	**84**
a	*b*
b	*a*N, *c*N, *d*
85	**85**
86	**86**
87	**87**
88	**88**
89	**89**
90	**90** +
91	**91**
92	**92**

1929	1938
93	**93**
a	*a*
b	*b*, *c*
c	*d*
94	**94**
95	**95**
96	**30** *c*N, **96**
97	**97**
98	**98**
99	**99**
100	**100**
101	**101**
102	**102**, p.**103**
103	**103** +
104	**104**
105	**105**
106	**106**
107	**107**
108	**108** +
109	**109**, p.**108**
110	**110**
111	**111**
112	**112**
113	**113**
114	**114**
a	*a*, *b*
b	*c*, *d*, *e*
115	**115**
116	**116**
117	**117**
118	**118**
119	**119**
120	**120**
121	**121**
122	**122**
123	**123**, p.**24** *d*N
124	**124**
125	**125**
126	**126**
127	**127**, p.**44** *d*
128	**128**
129	**129**
130	**130**
131	**131**
132	**132**
133	**133**, **157** *h*N
134	**134**
135	**135**
136	**136**
137	**137**
138	**138**
139	**139**
140	**140**
141	**141**
142	**142**
143	**145**
144	**143**, **146**
145	**147**
146	**144** *a*, *b*, p.*d*, **148**
147	**144** *c*, p.*d*
148	**147**
a	*c*
b	*d*
149	**149**
150	**150**
151	**151**
152	**152**
153	**153**, **38** *c*N
154	**154** *a*
155	**154** *b*, *c*, **155**
156	**156**
157	**157** +
a/*d*	*a*/*d*
p.*e*	*h*N
p.*e*	*e*/*g*, *i*, *j*
158	**158**
159	**159**
160	**160**
161	**161**
162	**162**
163	**163** *a*
164	**163** *b*
165	**164** *a*
166	**164** *b*
167	p.**164** *c* +
168	**164** *d*
169	**164** *e*
170	**164** *f*
171	**164** p.*c*, *g*
172	**165**
173	**166**
174	**167**
175	**168**
176	**194**, **175** *b*a*n*
177	**177**
178	**178**, p.**169**N, p.**170**N, p.**171**N, p.**172**N, p.**173**, p.**174**N, p.**175**N, p.**176**N
179	**179**
180	**180**, p.**169**N, p.**170**N, p.**171**N, p.**172**N, p.**173**N, **174**N, p.**175**N, p.**176**N
181	**181**, p.**169**N, p.**170**N, p.**171**N, p.**172**N, p.**173**N, p.**174**N, p.**175**N, p.**176**N
182	**182**, p.**169**N, p.**170**N, p.**171**N, p.**172**N, p.**173**N, p.**174**N, p.**175**N, p.**176**N
183	**183**, p.**169**N, p.**170**N, p.**171**N, p.**172**N, p.**173**N, p.**174**N, p.**175**N, p.**176**N
184	**184**
185	**185**, p.**175**N
186	**186**, p.**169**N, p.**170**N, p.**171**N, p.**172**N, p.**173**N, p.**174**N, p.**175**N, p.**176**N
187	**187**
188	**188**, p.**169**N, p.**170**N, p.**171**N, p.**172**N, p.**174**N, p.**175**N
189	**189**, p.**170**N, p.**171**N, p.**172**N, p.**173**N, p.**174**N, p.**175**N
190	**190**, p.**170**N, p.**171**N, p.**172**N, p **173**N, p.**175**N
191	**191**, p.**170**N, p.**171**N, p.**172**N, p.**173**N, p.**174**N, p.**175**N
192	**192**, p.**173**N, p.**175**N
193	**193**, p.**169**N, p.**170**N, p.**171**N, p.**172**N. p.**173**N, p.**174**N, p.**175**N, p.**176**N
194	**195**, p.**169**N, p.**170**N, p.**171**N, p.**172**N, p.**173**N, p.**174**N, p.**175**N, p.**176**N, **195** *b*N, *c*N, *d*
195	p.**200** *b*, p.**200** *c*
196	**196** *b*, p.**197** *b*
197	p.**197** *a*, p.**197** *b*, **197** *c*
198	**198**
199	**199**
200	**200** *a*, p. *b*N, *c*

Table 3.

CORRESPONDANCE DES NUMÉROS DES NOMENCLATURES DÉTAILLÉE, INTERMÉDIAIRE ET ABRÉGÉE DE 1929.

CORRESPONDENCE BETWEEN THE NUMBERS OF THE DETAILED, INTERMEDIATE, AND ABRIDGED LISTS OF 1929.

d	i	a	d	i	a	d	i	a	d	i	a	d	i	a
1	1	1	41	16	14	81	36	23	121	54	30	161	77	38
2	1	1	42	16	14	82	32	22	122	55	32	162	78	39
3	2	2	43	17	14	83	33	21	123	58	32	163	79	40
4	17	14	44	17	14	84	34	23	124	56	31	164	79	40
5	17	14	45	18	15	85	35	23	125	57	31	165	79	40
6	3	3	46	18	15	86	36	23	126	57	31	166	79	40
7	4	4	47	18	15	87	36	23	127	57	31	167	79	40
8	5	5	48	18	15	88	37	23	128	58	32	168	79	40
9	6	6	49	18	15	89	37	23	129	58	32	169	79	40
10	7	7	50	18	15	90	38	24	130	59	33	170	79	40
11	8	8	51	18	15	91	39	24	131	59	33	171	79	40
12	17	14	52	18	15	92	40	24	132	59	33	172	80	41
13	9	14	53	18	15	93	41	24	133	60	34	173	80	41
14	10	9	54	19	16	94	42	24	134	61	34	174	80	41
15	17	14	55	19	16	95	43	24	135	62	34	175	80	41
16	17	14	56	20	20	96	44	25	136	63	34	176	81	42
17	17	14	57	21	17	97	45	25	137	64	34	177	81	42
18	17	14	58	21	17	98	45	25	138	65	34	178	81	42
19	17	14	59	22	18	99	46	25	139	65	34	179	81	42
20	17	14	60	23	20	100	46	25	140	68	35	180	81	42
21	17	14	61	23	20	101	46	25	141	66	36	181	81	42
22	17	14	62	23	20	102	46	25	142	66	36	182	81	42
23	11	10	63	23	20	103	46	25	143	66	36	183	81	42
24	12	11	64	23	20	104	50	28	144	67	36	184	81	42
25	12	11	65	25	20	105	50	28	145	68	35	185	81	42
26	12	11	66	24	20	106	47	26	146	69	36	186	81	42
27	12	11	67	25	20	107	48	27	147	69	36	187	81	42
28	12	11	68	25	20	108	48	27	148	70	36	188	81	42
29	12	11	69	25	20	109	48	27	149	70	36	189	81	42
30	12	11	70	27	20	110	49	28	150	70	36	190	81	42
31	12	11	71	26	20	111	50	28	151	71	37	191	81	42
32	12	11	72	27	20	112	50	28	152	71	37	192	81	42
33	17	14	73	27	20	113	50	28	153	71	37	193	81	42
34	13	12	74	27	20	114	50	28	154	72	37	194	81	42
35	17	14	75	28	19	115	58	32	155	72	37	195	82	42
36	14	14	76	29	20	116	58	32	156	72	37	196	83	42
37	17	14	77	29	20	117	51	32	157	73	38	197	83	42
38	15	13	78	36	23	118	58	32	158	74	38	198	84	42
39	16	14	79	30	23	119	52	29	159	75	38	199	85	43
40	16	14	80	31	21	120	53	29	160	76	38	200	85	43

Table extraite de : — Table reproduced from : Commission internationale pour la revision décennale des Nomenclatures internationales des Maladies... 4e Session. 16-19 octobre 1929, p. 223. Paris, Impr. Nationale, 1930.

CORRIGENDA

Page	*Rubrique* *Item No.*	*Langue* *Language*	*Ligne* *Line*	*Lire ou ajouter :* *Read or add :*
228	1	*it.*	2	Tifo addominale
240	11	*it.*	1	Risipola (v)
252	14*b*	*it.*	1	Tubercolosi cerebrale
264	15*(b)*	*it.*	4	Tabe meseraica
			5	Peritonite tubercolare
»	17*b*	*it.*	1	Ascesso delle articolazioni
»	17*c*	*it.*	1	Ascesso freddo
276	22*a*	*it.*	2	Tubercolosi miliare
288	24*a*	*it.*	1	Sepsi generalizzata
»	25*(a)*	*it.*	3	Uretrite blenorragica
»	25*(b)*	*it.*	3	Oftalmia blenorragica
300	27*c*	*it.*	3	Giardiasi
»	28*c*	*it.*	4	Malaria estivo-autunnale
312	29*b(a)*	*it.*	3	Kala-azar
324	30*a*	*it.*	3	*delete :* Peste (vv)
»	30*c*	*it.*	1	Anevrisma dell' aorta
»	31*a*	*it.*	2	F. ricorrente da pidocchi
336	32*b(b)*	*it.*	1	*delete :* Sodoka
348	34*a*	*it.*	1	Vaiuolo
360	38*f(b)*	*it.*	2	Febbre dei tre giorni
»	39*a*	*it.*	3	Dermotifo
»	39*b(a)*	*it.*	3	Tifo da pulci
372	39*b(b)*	*it.*	3	Tifo da acari
»	39*b(ca)*	*it.*	3	Tifo da zecche minore
»	39*b(cc)*	*it.*	1	Tifo da zecche maggiore
»	39*b(cd)*	*it.*	1	Tifo da zecche maggiore
»	40	*it.*	1	Elmintiasi
396	43*(a)*	*it.*	1	*delete :* Mighetto
408	44*d(h)*	*it.*	1	Singhiozzo epidemico
420	91*a*	*it.*	1	*delete :* Endocardite lenta
»	91*b*	*it.*	1	Endocardite lenta
432	104*a(a)*	*it.*	2	Raffreddore
456	127	*it.*	2	Ittero catarrale
487	*(col. 1)*	*it.*	24	Cisti idatidea *it.* 41
488	*(col. 2)*	*it.*	30	Dermotifo *it.* 39*a*

LEAGUE OF NATIONS

BULLETIN OF THE HEALTH ORGANISATION

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Authors are alone responsible for views expressed in signed articles.

HEALTH IN EUROPE

A Survey of the Epidemic and Nutritional Situation[1]

by

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[1] The text of this survey was written between May and August 1944.

4565 — S.d.N. 1.030 (F.) 1.525 (A.) 12/44. Imprimerie de « La Tribune de Genève ».

HEALTH IN EUROPE

INTRODUCTION

A complete and accurate study of the influence of the war and of war conditions on public health in Europe will not be possible until the end of hostilities, because it is then, and then only, that all the facts will become available and that a statement of these facts will be possible. But *then* such a study will have only academic interest, and it is *now* that health authorities and relief organisations need information, even if only preliminary in character, that will be helpful to them in drawing up their plans for remedying the present situation, as regards both nutrition and epidemics.

A number of articles and even monographs have already appeared in the medical literature on different aspects of the problem : the shortcomings of rationing, losses in weight of children and adults ; increase in the incidence of certain communicable diseases ; changes in the prevalence and character of tuberculosis ; the increase in both infant and general death rates. Notwithstanding the limitations of area and time to which they are subject, and also their specialised character, these studies are of very great value as an aid in establishing the facts.

The marshalling of these facts has been attempted by a number of authors, chiefly in England and in North America. There has been an understandable, but nevertheless unfortunate, tendency on the part of authors to generalise and to consider as prevailing in whole countries, and even throughout Europe, conditions observed in selected groups of population of large cities. In so doing, some authors have been influenced by newspaper accounts or by reports

of refugees which, while perhaps not lacking in objectivity, are nevertheless not marked by that critical and scientific outlook that is essential for collecting and gauging such facts.

Alarming information is more readily accepted abroad than are more commonplace statements. Moreover, persons who have quitted their own country, and knowing its sufferings—in which they can no longer share—subconsciously tend to exaggerate these ills and, if in a position to do so, try to enlist the sympathy and help of other nations.

Information on the sanitary situation in Europe has so far been available only piecemeal, and no comprehensive study has been based on a large body of statistical material. We propose to furnish such a body of material, collected from the many reports which, in spite of the war, have continued to reach the Service of Epidemiological Intelligence and Public Health Statistics of the League of Nations in Geneva.

We are aware of the limitations of this statistical material, both in its extent and in its significance. Unfortunately, it does not cover adequately those countries which have suffered most from the war and from famine—*viz.*, Poland and Greece. We know that deaths directly due to the war or occurring in military forces are withheld from public records. We also know that enrolment in the forces and auxiliary organisations, systematic transfers of whole populations, migrations of refugees, deportation of labour, attraction of workers to war industries, and the evacuation of specific elements of the population of large cities or of all inhabitants after the destruction of certain towns, have profoundly altered the make-up and the size of populations in many countries, thus making it difficult for death rates to be satisfactorily computed or interpreted.

In this respect, changes are even greater in the case of cities. Omissions in the number of deaths recorded and changes in the population on which death rates should be computed are further sources of inaccuracy.

An endeavour has been made here to introduce corrections on the basis of the information available, and it is believed that—such as they are—the rates computed and reproduced in the present memorandum are sufficiently accurate to give an adequate idea of the trend of mortality in most countries of Europe.

The statistical material collected covers—in addition to notifications of communicable diseases—births, infant deaths, and deaths

from all causes. From these and from available data on population, birth rates, infant mortality and general mortality rates have been computed. Rates have been calculated also for tuberculosis, which experience has shown to be a reliable index of severe malnutrition.

Mortality statistics have been compiled not only for individual countries, but also for large cities or groups of large towns within those countries, in order to bring out more clearly the effects of malnutrition, which common experience has shown to be more prevalent in the cities than in rural areas.

It is believed that divergencies [1] in the trends of general mortality of cities, on the one hand, and of the countries in which they are situated, on the other, give a measure of the influence of insufficient feeding.

With regard to communicable diseases, we do not propose here to give detailed tables of their prevalence in the various countries of Europe during recent years. Such tables have repeatedly appeared in a series of special notes in the *Weekly Epidemiological Record*, and we shall merely extract from them the more significant figures, and add brief comments.

While our aim is to show rather the health situation than its causes, we shall be bound, in the course of this study, to refer to the food situation in relation to health.

To estimate the food situation in Europe, we have made use of: *(a)* computations concerning normal consumption of staple foods in European countries based on documents issued by the Economic Intelligence Service of the League, and documents on war-time rationing and consumption published by the same Service; [2]

[1] We do not mean here the "normal" differences between urban and national rates which result from differences in age distribution of the populations and from more or less permanent social and economic factors, but the wartime *excess* of these differences: resulting largely from the greater influence of the food shortage factor in cities.

[2] *The Problem of Nutrition:* Vol. IV — *Statistics of Food Production, Consumption and Prices.* Prepared by the International Institute of Agriculture. League of Nations document A.12(*c*).1936.II.B. (Ser. L.o.N. P. 1936.II.B.6).

Raw Materials and Foodstuffs. Production by Countries, 1935 and 1938. (Ser. L.o.N. P. 1939.II.A.24.)

War-time Rationing and Consumption. (Ser. L.o.N. P. 1942.II.A.2.)

"Rationing of Foodstuffs in Certain Countries of Europe, 1939-1944". *Monthly Bulletin of Statistics*, League of Nations, June 1944, Vol. 25, No. 6, pages 155-177.

(b) a series of monographs published in 1939 for the European Conference on Rural Life, which cover the agricultural situation in European countries ; *(c)* data on food restrictions, taken from the daily Press, medical periodicals, and private studies ; [1] and *(d)* reports from national health administrations and from relief organisations, both official and private.

In our present attempt to make a preliminary estimate of the health situation of the population of Europe, we have the benefit of the experience gained in the study of the consequences upon public health of the first world war and of the economic depression of 1929-1932. This latter study, which covered the more industrialised countries of the world, was carried out in 1932 by the Health Section of the League.[2] It included a number of special investigations by groups of experts on nutrition surveys and standards at various ages, which found their application during the second world war. It was also the starting-point of a search [3] for a health index more sensitive than ordinary death rates. Although such an index has not yet been established, the search provided a valuable knowledge of the shortcomings and true significance of death rates in assessing the state of public health. This knowledge has been turned to advantage in the present study, particularly in Chapter I, which is devoted to a critical analysis of the material available for judging the present health situation in Europe.

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[1] Among the latter, particularly BOURNE, Geoffrey H. : *Starvation in Europe*. Allen & Unwin, London, 1943.

[2] " The Economic Depression and Public Health ". *Bulletin of the Health Organisation*, 1932, Vol. I, pages 425-476.

[3] STOUMAN, K., & FALK, I. S. : " Health Indices : A Study of Objective Indices of Health in relation to Environment and Sanitation ". *Ibid.*, 1936, Vol. 5, pages 901-1081.

STOUMAN, K. : " Health Indices in an Experimental Study of a Rural District of Hungary ". *Ibid.*, 1937, Vol. 6, pages 766-821.

STOUMAN, K. : " Health Indices established in an Experimental Study of the City of Brussels ". *Ibid.*, 1938, Vol. 7, pages 122-167.

Chapter I.

A CRITICAL STUDY OF THE STATISTICAL MATERIAL AVAILABLE FOR FORMING AN OPINION ON THE HEALTH OF EUROPE

In the absence of any positive and synthetic index of the state of health of populations, recourse must be had to morbidity and mortality rates which, in representing the amount of sickness and of death, express, as it were, the reverse of health.

They do that in a very crude way only, since not all diseases end fatally and not all impairment to health, physical or mental, reaches a stage when it constitutes an actual disease, or at least an illness, interrupting work and therefore appearing in morbidity records.

Morbidity statistics.—For a number of reasons, military and other, morbidity records for the war period are not available and, even if they were, they could scarcely be comparable with those of pre-war years, and they would therefore be valueless for our purpose. Army medical statistics will not, for obvious reasons, be made public before the end of hostilities.

Morbidity statistics of social insurance organisations will apply, in belligerent countries at any rate, to a non-military population, the sex and age composition of which is quite different from the pre-war one.

Moreover, for patriotic reasons or from compulsion, workers in war-time are less likely to absent themselves from work on account of minor ailments or fatigue than they would be under normal circumstances.

Finally, the change of occupation from peace-time callings to war industries would utterly falsify any attempted comparison between peace-time and war-time morbidity rates relating to whole working populations.

Medical statistics concerning school-children are scarcely more satisfactory for our purpose. Normally, they are available only for large cities, and the evacuation to the country of children from these cities makes a large-scale comparison of absenteeism and morbidity rates rather difficult as between war-time and peace-time.

1. General Mortality Rates

In spite of their limitations, mortality rates constitute a trustworthy index of prevailing health conditions, and the general mortality rate, which summarises mortality from all causes and at all ages is probably the best index we have at present of the health situation considered as a whole.

It must be acknowledged, however, that, as mortality varies considerably with age, and as the age distribution of population varies from country to country, general mortality rates are not strictly comparable as between one country and another, unless they are standardised for age and sex.[1]

In normal times, the age composition of population in a given country varies very slowly and therefore the crude death rate of one year is quite comparable with that of other years, not too far distant.

In war-time, however, this is not the case in belligerent countries. There, a large proportion of the young and middle-aged population enters the army and other military services, the mortality statistics of which are established quite apart from the ordinary civilian statistics and, as a rule, are withheld. Apart from children and adolescents, who normally display a low mortality, civilian statistics cover in war-time an abnormally large proportion of " bad risks "—*i.e.*, infants and old people. This age factor alone should make the crude—*i.e.* civilian—mortality rate rise in war-time.

Unfortunately, details for age and sex distribution of mortality are, for most countries, not yet available for war years and therefore the standardisation that would have done away with this cause of error in comparisons is not possible.

In some belligerent countries, such as Germany and Italy, civilian deaths due to enemy action are omitted from the published statistics, just as are military deaths. This omission, which would be a cause of error in a computation of the population and its changes, is an advantage in a study concerned with the health of the population, in which a heightened general mortality might suggest unfavourable health conditions rather than war casualties.

[1] An international table of standardised rates will be found on pages 65-67 of the *Annual Epidemiological Report* for 1938 (E.I.23.1941).

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Adjustment of population figures.—General mortality rates take into account the recorded number of deaths, which is comparatively easy to determine, and the corresponding population. The latter is far more difficult to ascertain in the case of belligerent or occupied countries.

Territorial changes resulting from recent annexations naturally necessitate adjustments in the figures for population of the countries concerned. The latter provide rough estimates of the populations inhabiting the transferred areas.

Transfers of populations themselves from one country or from one region to another are more difficult to take into account. Certain organised migrations, such as those of people of German ancestry from the Baltic States, the Soviet Union and Yugoslavia into the Reich during the winters of 1939/40 and 1940/41, have been the subject of fairly precise information published by the German authorities. For other migrations, of greater magnitude, official figures are lacking or, at any rate, unreliable. Such is the case for transfers of foreign labour from the various occupied countries into Germany, for the transfer of Poles from Western Poland (Wartheland) into Central Poland (Generalgouvernement), etc. In the computation of populations, we have not only taken into account, whenever possible, available estimates of prisoners-of-war and of transferred labour for the years up to 1942, but have also made use of the comprehensive study on migrations by E. M. KULISCHER: *The Displacement of Population in Europe* (International Labour Office, Montreal, 1943).

Changes in urban populations.—In view of the greater sensitiveness of urban populations to food restrictions, we have given mortality figures for single cities or groups of cities, in addition to figures covering whole countries.

Reliable population figures for cities are even more difficult to obtain than are figures for whole countries. They are liable to the same causes of error as those for countries (mobilisation, deportations) with, in addition, internal migrations, such as take place as the result, on the one hand, of the development of war industries in the cities and, on the other, of evacuation.

These evacuations have affected a large proportion of the population of certain cities, for instance, during the refugee movement of May-June 1940 in Belgium and France, or following the partial

destruction of a city by air bombardments, such as took place in 1942-1943 in Germany, or through preventive evacuation of special groups—*e.g.*, children, pregnant women and elderly people—from exposed cities, such as took place on a large scale in Paris and London in the autumn of 1939 and again in London in the summer of 1940.

In some cases, rationing censuses serve as guides to the numbers of persons in cities. In other cases, official statistics, published week after week, concerning populations are obviously wrong, as they take no account of population changes brought about by heavy air-raids, as a result not only of loss of life during the raid, but also of the dispersal enforced by the destruction wrought. Such is the case, for instance, in regard to the population of a number of German cities (Cologne, Essen, etc.). One can well imagine how difficult it is for local authorities to record population changes during the chaotic period immediately following partial destruction of a city and the dispersal, temporary or otherwise, of many of its inhabitants.

Difficulties in computing urban rates based on the true population apply to both mortality rates and birth rates. They do not apply to infant mortality rates which, being based on recorded births and infant deaths, do not take the population figures into account. Birth rates and infant death rates for cities are, on the other hand, particularly affected by the selective evacuation of pregnant women and of infants.

We have outlined above the principal obstacles to accuracy that are encountered in the computation of general mortality rates; in Chapter II, which deals with *each* country individually, particulars are given concerning the actual difficulties encountered in the elaboration of their statistics, and concerning the adjustments made in order to overcome them.

We fully realise that, even with the corrections made and those suggested, the rates given can lay no claim to absolute accuracy.

We believe, however, that, such as they are and pending the time when more reliable data will be available after the war, these rates can be used to follow the trend of mortality during war years in the various countries.

In order to facilitate the interpretation of recent figures, we give, for each country, for purposes of comparison, the rates established for a period covering the eleven years preceding the war. Minimum, median and maximum rates recorded during that period for both

yearly and monthly series have been selected for the purpose, in order to show the range of normal variation in annual and monthly rates.

The unusually high rates, those which exceed the upper quartile in each of the series, are printed in heavy type. [1]

Medical significance of general mortality rates.—The above review of the purely *statistical* factors influencing general mortality *rates* has no other aim than to facilitate an estimate of the medical factors involved, by freeing the figures from the influence of non-medical causes.

It is proper at this stage to envisage *how* general mortality may be, and in some countries is, affected by the war. Apart from war casualties (including the bombing and shooting of civilians) war may increase mortality :

(1) by favouring the spread of epidemics ;

(2) by producing general or localised food shortage.

If intense, the shortage may lead to actual starvation affecting all ages—particularly children, whose bodily reserves are comparatively less and whose requirements are comparatively greater than those of adults. (This was the case in Greece, for instance, in 1941-1942, and in Russian prisoners' camps in Germany in 1941 and 1942.) If more moderate, food shortage will induce hunger and emaciation, and all the variety of malnutrition symptoms.

Chronic food insufficiency or inadequacy will result in emaciation first and then, gradually, in the syndrome of famine œdema, with consequent heart failure and death. This was the fate of many inmates of asylums for the insane or the aged in occupied countries such as Belgium and France, and of a very much greater number of civilian prisoners of all ages and of both sexes in gaols and concentration camps. [2]

It may be pointed out that people under detention are, as a rule, not only unable to obtain supplements to the official basic rations

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[1] For fuller explanations, see our note " Interpretation of International Statistics of Communicable Diseases ". *Weekly Epidemiological Record*, **19** : No. 7, 32-34, 17.II.1944.

[2] We know of one particular camp in France in which as many as 80 people died each month from famine disease.

but, only too often, are unable even to procure the latter, as their keepers are in a position to retain part of the rations for their own consumption or for profit.

If food shortage is less stringent or less continuous, it may cause a fall in muscular tone, blood pressure, and general fatigue and weakness, and may lower the bodily resistance to infection. Decompensation of the heart, after a comparatively small physical effort or as the result of even a slight degree of pulmonary congestion due to an infection of the respiratory tract, such as influenza, will cause death. This is frequently the case among elderly people whose official rations are particularly scanty.

In such cases, the death certificate will attribute death to heart disease, or to a pulmonary condition, probably, without any mention, even as a contributory cause, of the underlying nutritional insufficiency.

Lowered resistance to infection is particularly obvious in the case of tuberculosis. Wherever food has been deficient in quantity and quality, tuberculosis has assumed a higher incidence and a much greater severity (*i.e.*, a more rapid and fatal course) and has consequently resulted in a greatly heightened mortality. As, in European countries, tuberculosis is responsible for some 4 to 10% of all deaths, a significant rise in tuberculosis mortality is likely to make itself felt in the general mortality rate.

The lowering of the resistance to other communicable diseases is constantly asserted by writers and, indeed, is quite probable *a priori*. There are, indeed, many instances on record of increase in the incidence of communicable diseases such as typhus, when associated with starvation. At present, in Western and Central Europe, there does not seem to be any appreciable increase in the severity of the endemo-epidemic diseases, even of those like diphtheria and scarlet fever, the incidence curve of which has, during the war, shown a well-marked upward trend. Indeed, *there is so far no indication that common communicable diseases have contributed in any significant degree to the rise in general mortality wherever it has been observed*, except perhaps in the case of influenza.

It is quite possible, and indeed likely, that influenza, prevailing at the same rate as in pre-war years, may have caused more deaths among elderly people in a state of circulatory imbalance, owing to severe malnutrition, than it would have done under normal

circumstances. It must be aeknowledged that, so far, this is pure conjecture.

Influenza and influenza-like winter infections are the main causes of the yearly fluctuations of general mortality in Europe, and more generally in countries of temperate climate, where hygiene has reduced the influence of the summer waves of infant mortality.

One must therefore carefully scrutinise the seasonal distribution of deaths before coming to conclusions as to the causes of a rise in the yearly rate of general mortality. An instance showing the need for care in this respect is furnished by the rise in the 1940 rates observed in France, Belgium and the Netherlands.

One might have been tempted to aseribe this rise entirely to the effects of the invasion of these countries.

But elose examination of the figures reveals that an unusually high mortality prevailed during the first quarter of the ycar—*i.e.*, *previous* to the invasion ; it was apparently due to one wintcr wave of influenza.

This instanee points to the necessity for resisting a natural and common tendeney to ascribe *to* the war all ehanges of mortality observed *during* the war.

It also shows that the superfieial study of annual rates should not be regarded as sufficient and that attention should be given to statistics split into smaller time units—quarters, months, or four-weekly periods.

That is the reason for our adoption of these units, whenever possible, in the tabular and graphic representation of our statisties in the present study.

Pending the publication of full statisties of *causes of death*, we are not in a position to gauge the full significanee of the general mortality rate and we must have recourse to induction and speculation. We have, however, in the infant mortality rate one of its important components, as infant deaths make up from 6 to 18% of all deaths in European countries.

2. Infant Mortality Rates

These rates, being the number of infant deaths (exclusive of still-births) per thousand live births, do not take into account the population figures, and thus escape all the causes of error which tend to vitiate other death rates.

Moreover, the exclusion from published statistics of deaths occurring among special groups, such as the members of the Services, internees, etc., is less likely to affect statistics relating to infants than those relating to adults.

Infant death rates for whole countries can therefore be considered as reliable.

Normally, the number of births occurring in any country exhibits marked and regular seasonal variations, but only slow changes from one year to another. Statisticians are therefore quite justified in computing infant mortality rates from the number of births and of infant deaths occurring within a year or even within a smaller period —quarter or month.

This method has, indeed, become more satisfactory—that is to say, it has fitted the facts more and more closely during the last decennia—as deaths occurring during the second half of the first year of life have become fewer, and as mortality has tended to be restricted to the first months if not the first weeks of the life of the newly-born.

When infant mortality is not restricted to the first months, and when, owing to abrupt mobilisation or demobilisation, the number of births presents rapid and considerable falls or rises, then the usual mode of computing the infant death rate on the basis of the deaths and births occurring within a given short period becomes unsatisfactory.

The number of births may be very small and the number of neonatal deaths accordingly small, but late deaths of infants born previously in normal numbers will not be reduced and they will heighten the rate computed on a small number of births.

In the case of France, for instance, where rapid mobilisation on a large scale, in September 1939, caused a severe fall of the birth rate during the third quarter of 1940, the infant death rate, computed on deaths and births of that quarter, was 93 $^0/_{00}$. It would have been 88 $^0/_{00}$ only if computed on the deaths of that quarter, in relation to the mean quarterly births of that quarter and of the three preceding quarters.

In order to avoid such effects of rapid changes in the birth rates, Germany, "Bohemia and Moravia" and the Netherlands compute their mortality rates on the births occurring during the year preceding the period in question. This has the effect of smoothing the infant mortality curve.

The rates of the other countries which do not adopt this method are at present more sensitive to actual changes in infant mortality conditions, but they will show an abnormal and purely " statistical " fall, 9 to 12 months after the return of men to their homes after the war, when births will suddenly increase.

Statistical fallacies of infant death rates in the cities.—Apart from this minor point, we have expressed the opinion that, as a rule, infant death rates for countries were quite reliable, since population changes did not enter into their reckoning.

But, when one deals with urban rates of infant mortality, this remains true only in so far as the number of births has not been artificially and abruptly modified by evacuation of pregnant women and the number of infant deaths by evacuation of infants.

If evacuation of infants is more complete than that of prospective mothers, then the urban infant mortality rate presents a fictitious fall. Inversely, it will present a fictitious rise if evacuation of pregnant women has been more complete than that of infants.

As evacuations have fluctuated considerably with varying official arrangements, the feeling of security or of danger, and the extent of actual destruction due to air attacks, and as we have as yet no reliable records of the actual evacuation movements of women and infants, we have been unable to introduce corrections for this factor in the rates computed.

The rates given are therefore unreliable for Paris in 1939 and 1940, for English cities for the same years and also for 1941, and for the large German cities from 1942 onwards, the amount of error increasing for the latter with the extent of aerial bombardment.

It must be acknowledged that, while rates applying to individual cities, such as London, Coventry, Cologne, Hamburg, Berlin, etc., are quite distorted and therefore worthless for certain periods owing to evacuation, rates applying to the bulk of the English or German large towns, with a population of some 21 and 22 millions respectively in pre-war days, still retain something of their significance, as only a proportion of the cities concerned have been seriously affected by evacuations.

While reserving for another study a discussion of the medical significance of infant mortality rates, we feel it necessary to state here that they are not an absolute measure of the health of infants. Malnutrition in several of its forms, including rickets, insufficient

body-weight, retarded growth and various forms of ill-health are compatible with life and may not directly or immediately influence infant death rates.

3. Tuberculosis Mortality Rates

The experience of the first world war and of the post-war inflation period in the cities of some countries has shown that tuberculosis is a comparatively sensitive index of an insufficient or unsatisfactory food supply.

It has even revealed that the food factor ranked first among the "social" factors influencing the course of the disease and its mortality.[1] [2]

In spite of the influence of that factor, it is not the only one, and it would be quite misleading to ascribe to it all changes in the rate. Whilst intending to go more deeply into that subject later in a special study, we may mention here the influence of increased strenuousness and longer hours of work in war industries. This has been noticeable not only during the war, in belligerent countries, but even in pre-war years, in countries then at peace, in areas where war industries were feverishly increasing their output.

Influenza prevalence has a marked effect on the tuberculosis death rate. Whether the precarious respiratory and circulatory balance of patients suffering from pulmonary tuberculosis is upset through the congestion induced by influenza, or through another mechanism, the fact remains that every winter influenza epidemic is accompanied by a marked rise in the number of tuberculosis deaths, which is followed the following quarter by a compensatory fall.

This phenomenon was particularly obvious at the time of the severe 1918 influenza pandemic, but it is still noticeable, on a smaller scale, at every recurrence of epidemic influenza ; it was observed, during the war, in the first quarter of 1940 in Western Europe and, to a lesser extent, in the first quarter of 1941.

A similar reaction is to be expected from the bigger influenza wave of the winter of 1943/44.

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[1] Biraud, Y. (1930) : « La mortalité tuberculeuse et son évolution ». *Revue de Phtisiologie*, **11** : N° 1, pages 58 *et seq.*

[2] Faber, K. (1938) : "Tuberculosis and Nutrition". *Acta Tubercul. Scandin.*, **12** : fasc. 4, pages 287-335.

4. Statistics of Communicable Diseases

Both the medical profession and the general public, on the basis of the tradition of ancient wars and of the first world war and its aftermath, *expect* epidemics to develop as a result of the war.

The statistics of communicable diseases from belligerent and occupied countries in Europe are therefore eagerly awaited and will be the subject of careful scrutiny. It is considered desirable to indicate here to what extent such statistics are reliable and also to mention certain factors which may have some bearing on their significance.[1]

It must be emphasised that, owing to national differences in notification, usages and procedure, figures of communicable diseases are by no means comparable as between one country and another. This fact, and the incompleteness of registration in most countries at all times make the use of morbidity rates in international studies quite misleading. Figures available for notified cases of communicable diseases can, however, be used in establishing a comparison of their prevalence within a given country as between one period and another. This comparison is quite valid, as the proportion of missed or unreported cases varies little from year to year and therefore does not affect the significance of the trend.

In order to estimate the situation concerning a particular disease, recent figures, whether monthly or four-weekly, are compared with those of the preceding month, those of the same month in the preceding year, and the median figure observed for the same month in the course of a series of years.

In the tables, given in Chapter II, the figures for communicable diseases are printed in heavy type whenever they exceed the upper quartile of the series covering the 11 years preceding the war — *i.e.* 1928-1938.

Such figures are clearly above " normal ".

To what extent are the present data really comparable with these pre-war standards ? In most countries they are definitely comparable, because the civilian population, to which the figures apply,

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[1] The reader will find a more complete treatment of the subject in our note on " The Interpretation of International Statistics of Communicable Diseases ", *Weekly Epidemiological Record*, 19, No. 7, pages 32-34, 17.II.1944.

and the number of physicians diagnosing and notifying cases, have not declined in a proportion approaching the considerable fluctuations that communicable diseases exhibit in their prevalence even in peace-time.

In Germany, for instance, a large proportion of the adult population, both male and female, is mobilised, and therefore escapes the usual form of notification, but whatever estimate is made of the proportion of the mobilised in Germany, it is not of the same order of magnitude as the fluctuations of communicable diseases in that country.

The recorded rate for diphtheria is often doubled or trebled in the course of two or three years. The number of scarlet fever cases may increase five-, six-, or sevenfold in about the same time. Fluctuations are no less with regard to cerebrospinal meningitis and epidemic influenza, so that variations recorded in war-time are still quite significant.

Nevertheless, in judging the figures, one must bear in mind that the number of physicians available for diagnosis among the civilian population has a marked influence not only on the number of notifications, but on the apparent fatality rates of communicable diseases.

The structure of armies is such that medical officers must be attached to every military unit of importance, and large numbers of them must be held in readiness and remain comparatively inactive in anticipation of possible emergencies in field hospitals. Mobilisation in a country therefore results in a most abnormal distribution of available medical aid as between the military and civilian poputions. Medical men left to care for civilians are, in consequence, overworked. Restriction in motor and other forms of transport adds to the difficulties of the population in obtaining their services. The physicians tend naturally to care only for the more urgent or severe cases, and thus many light cases escape notice, treatment and notification. As deaths from communicable disease are still properly recorded, there results an apparent increase in the fatality rate.

A similar situation prevails with regard to hospitals. Many of them are converted into military hospitals; thus, the space remaining for the civilian population is apt to be crowded. Crowding is increased not only as a result of bombing and destruction of hospital buildings themselves, but owing to the destruction of ordinary residential houses as well. What remains of hospital accommodation must be

allotted not only to air-raid casualties, but also to patients who would normally have been treated at home but whose homes are destroyed. The dearth of physicians and nurses available for civilians contributes to increase the number of patients seeking admission to hospitals.

It is clear that, in these circumstances, only the more severe cases of communicable disease will be accepted by hospitals. Hospital physicians have sometimes voiced their alarm on observing the high proportion of severe and complicated cases of diseases such as diphtheria and scarlet fever, and have failed to realise that this was essentially due to lack of accommodation for lighter cases. Indeed, their statistics showed a reduction in the actual number of patients hospitalised, in spite of the rise in the prevalence of these diseases in the population at large.

The prevalence of endemo-epidemic disease depends in part on the proportion of receptive individuals in the general population, and therefore on the number of births. Except, perhaps, in Poland and other parts of Eastern Europe particularly affected by the war, the effect of the latter on the birth rate has not been such that it could have any significant bearing on the trend of communicable diseases. Births in some countries at war have suffered an unexpectedly small reduction (Germany, France) and in other countries have even risen (Great Britain, United States of America) (*cf.* graph, page 674).

5. Data in the Medical Literature on Malnutrition and Deficiency Diseases

Just as the daily Press gives a prominent place to the unusual and exceptional—crime, accidents, etc.—so the medical Press normally contains more records of rare diseases and exceptional cases than of commonplace daily routine observations. Normally, therefore, the medical Press can in no way be considered as a faithful mirror of the relative importance of the prevailing diseases so far as the community as a whole is concerned.

This tendency has persisted during the war. Observations of the more acute forms of deficiency diseases have received a good deal of attention, but references to such cases have not increased *pari passu* with the increase in prevalence of these diseases. On the other hand, the results of special nutritional or related surveys and other

collective observations make their appearance where single observations and individual cases tend to disappear from the pages of medical journals.

Both in normal circumstances and during war-time, the diseases prevailing among the poorer sections of the urban population—*i.e.*, those persons attending public clinics and hospitals—are the subject of far greater attention and of more numerous publications than are those affecting the wealthier classes in towns, and the rural population generally, because these two groups of population are treated by medical practitioners who have little opportunity for observing large series of similar cases, have no assistants to help them in analysing such series properly, and have less incentive and fewer facilities for writing about them than have the teaching staffs of the large hospitals.

The medical literature, in depicting the health situation of the poorer strata of city dwellers, does not provide an adequate description of the situation as it affects the general population.

The first world war brought to light the fact that food restrictions applied with more stringency and with more dire results to the urban than to the rural population. The second world war has made the fact evident in a much greater number of countries. One would therefore expect that the health situation of urban populations would have deteriorated more than that of rural areas, and therefore more than that of whole countries.

In Chapter II, rates of general, infant and tuberculosis mortality are given for each country, separately for the country as a whole and for large cities. In those countries where food restrictions have not been sufficiently severe to influence public health and mortality, one would expect urban and global rates to have remained stable or, rather, to have moved in a parallel direction. On the other hand, an increase in mortality rates which is larger in the cities than in the country as a whole is *prima facie* evidence of the effects of malnutrition.

6. Significance of Data on Rationing

The present study aims at giving particulars of the health situation in Europe during the war years rather than data on food consumption. Readers wishing for data on consumption and rationing are referred to the relevant publications of the Economic Intelligence

Service of the League of Nations.[1] The figures reproduced in these documents refer, as a rule, to the daily ration, at successive dates, of an adult doing ordinary light work.

We should like to emphasise the fact that these figures can in no way be taken as representing actual consumption, since :

(1) Rationing, as a rule, does not cover all foodstuffs, and very rarely includes, for instance, fresh vegetables and fruit ;

(2) Official rations are often unobtainable in full, certain coupons, as, for instance, those for meat, milk, etc., not being " honoured " for months at a stretch ;

(3) Rations vary according to the size of a community ; they are, for instance, greater in the cities than in the country, rural dwellers being considered to be food producers ;

(4) Rations vary with the age, trade, condition and sometimes with the race and nationality of the individual ;

(5) Where rationing is effectively planned, rations vary considerably also with the season. The availability of certain unrationed foodstuffs in a particular month is taken into account in order to reduce the issue of rationed foods of the same nature ; and

(6) Official rationing does not, naturally, indicate the amounts available on the black and grey markets.

Such as they are, rationing figures may give a useful indication of the *trend* of availability or scarcity of certain types of food (fats, proteins of animal origin) and even of food in general ; but they should not be taken as actual consumption figures.

To reach a satisfactory estimate of actual consumption, a considerable amount of additional information, chiefly non-official and non-statistical, is necessary in order to supplement and qualify the statistical data on rationing. The limited scope of the present memorandum prevents us from reproducing the statistical and other bases of our estimates.

[1] *Cf.* footnote 2, page 561.

Chapter II.

THE HEALTH SITUATION IN THE VARIOUS COUNTRIES OF EUROPE

While general impressions may be obtained of the sanitary [1] situation in Europe—*i.e.*, on the extent of epidemies and the risks of their spreading—it is impossible to make a satisfactory synthesis on the health of the European population, in view of the considerable differences normally existing between the populations of the various parts of Europe in their age composition, their social and economic status, their hygienic development and sanitary institutions. The war has added further and equally important differences. The break-up of communications, both international and intra-national, has increased the unevenness in the food distribution between agricultural and urban areas. The political and military situation has altered, requisitions have favoured certain countries at the expense of others ; so that although, on the whole, there has been a general deterioration in the standard of living and in the nutrition of European peoples, with a consequent lowering of the state of health, the differences existing between different classes and communities have been made greater. Generalisations concerning them are thus rendered more difficult.

In this chapter, we have therefore considered each country separately, grouping the most significant statistics available bearing on the health of the population, figures relating to large cities or groups of cities and towns supplementing in each case those concerning the country taken as a whole.

The figures, as far as obtainable, cover population, annual rates of births, deaths and natural increase, detailed monthly or quarterly rates of infant and general mortality, and the actual number of cases notified of the more common and important communicable diseases.

[1] In the present memorandum, the term " *sanitary* " situation generally refers to the " *epidemic* " situation ; while " *health* " situation refers more particularly to the physical well-being and nutrition of the population. In some cases, however, the term health situation is taken in its general sense, covering both meanings. Such is the case for the title of this chapter.

Footnotes to the tables show some of the statistical difficulties which had to be overcome in computing the rates.

The tables for each country are preceded by a short statement on the incidence of the war on that country, such as date of participation in the war, invasion, occupation, etc., up to May 1944.

Data concerning the countries are presented in geographical order, so as to bring out more clearly differences that may have resulted, between neighbouring and otherwise comparable countries, from the occupation of one and the neutrality of the other, etc. The order adopted is the following :

Western Europe.

British Isles : England and Wales, Scotland, Eire.
Iberian peninsula : Spain and Portugal.
West Coast : France, Belgium, the Netherlands, Denmark.

Northern Europe.

Scandinavian peninsula : Norway, Sweden, Finland.

Central Europe.

Switzerland.
Germany, Austria, Czecho-Slovakia and Hungary.

Eastern Europe.

Poland, the Baltic States, the Soviet Union.

South-Eastern and Southern Europe.

Roumania, Bulgaria, Greece, Turkey.
Yugoslavia, Italy.

Western Europe.

England and Wales

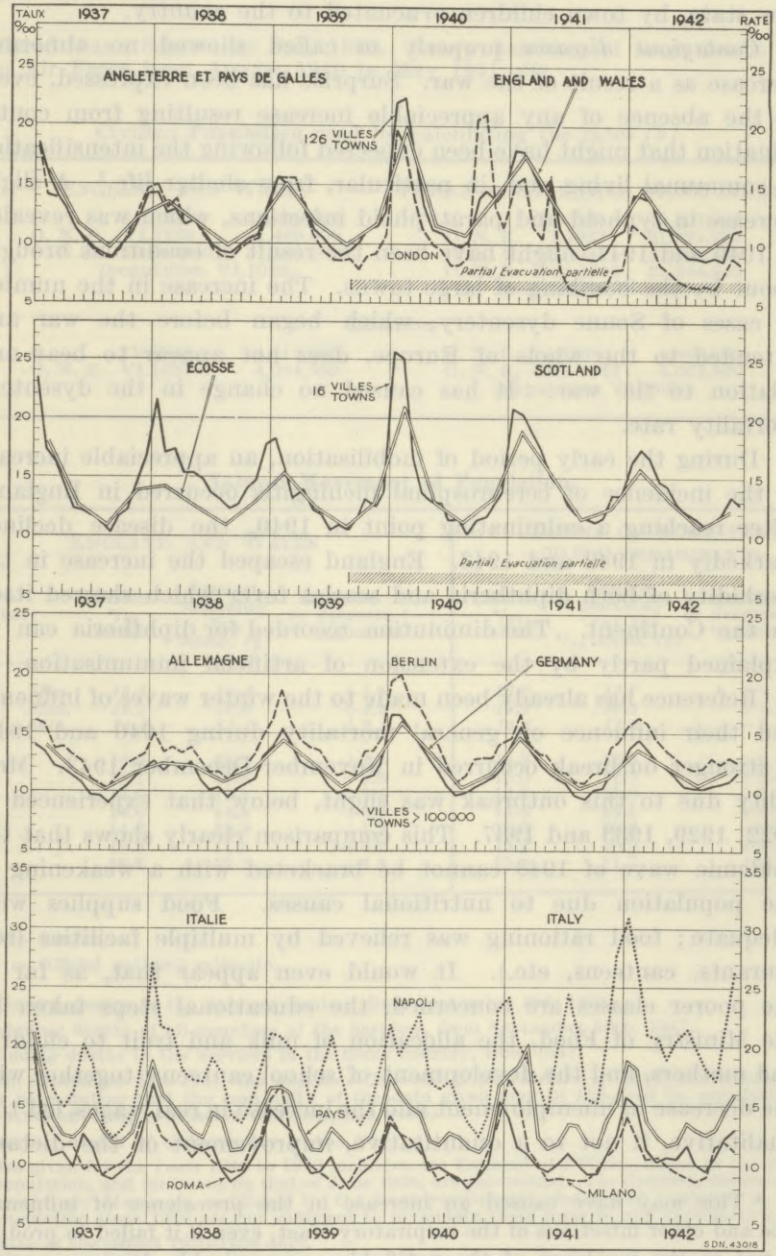
Notwithstanding the war effort, mobilisation extending to a considerable proportion of the population, important displacements, on the one hand, of labour and, on the other, from May 1939 to May 1941, of women and children evacuating bombed towns, the demographic situation and the health situation of England and Wales have remained quite favourable throughout the first five years of war.

After a very slight fall in 1941, the *birth rate* gradually rose to a level which, in 1943, was much higher than during the years immediately preceding the war; and which, in fact, had not been attained since 1928.

Infant mortality, which, before the war, showed a slow but steady decline, continued to follow the same movement, figures for 1942 and 1943 being well below the lowest ever recorded in the country. This is true both for the infant population of the whole of England and for that of the large towns. It should be noted, however, that, during 1941, there was an increase in infant mortality, of a marked character in the towns, particularly during the warm season of the year. *General mortality* rose above its pre-war level, both in 1940 and in 1941, later falling back to normal (*cf.* graph, page 581). The increase was due very largely to the excess mortality occurring during the first quarter of the year, when there was a small epidemic of influenza (particularly in the large towns). But other causes also contributed to the rise in the number of deaths—the bombing of the large towns during the autumn of 1940 and the winter of 1940/41, all the consequences of fatigue caused by repeated alerts, and cross-infections facilitated by life in air-raid shelters. Adaptation to a life of intense industrial work, new to many men and women, placed a considerable strain on them, and this has probably contributed to the slight recrudescence in *tuberculosis mortality* experienced in 1940 and 1941.

Among other factors invoked to explain this recrudescence may be mentioned the fact that many tuberculous patients who, in 1939, were undergoing treatment in sanatoria had to leave them when these were turned into casualty hospitals. An increase in the

General Mortality, per 1,000 Inhabitants, in Some Belligerent Countries: England and Wales, Germany, Italy, Scotland, 1937-1942.



N.B. — For war years, rates are only approximate.

number of cases of tuberculosis meningitis in children has been attributed in part to the increased drinking of infected milk, in the raw state, by town children evacuated to the country.

Contagious diseases properly so called showed no abnormal increase as a result of the war. Surprise has been expressed, even, at the absence of any appreciable increase resulting from contamination that might have been expected following the intensification of communal living and, in particular, from shelter life.[1] A slight increase in typhoid and paratyphoid infections, which was revealed in 1940 and 1941, might have been the result of conditions brought about by the bombing of large towns. The increase in the number of cases of Sonne dysentery, which began before the war and extended to the whole of Europe, does not appear to bear any relation to the war. It has caused no change in the dysentery mortality rate.

During the early period of mobilisation, an appreciable increase in the incidence of cerebrospinal meningitis occurred in England. After reaching a culminating point in 1940, the disease declined markedly in 1942 and 1943. England escaped the increase in the morbidity of both diphtheria and scarlet fever which showed itself on the Continent. The diminution recorded for diphtheria can be explained partly by the extension of artificial immunisation.

Reference has already been made to the winter waves of influenza and their influence on general mortality during 1940 and 1941. A stronger outbreak occurred in November-December 1943. Mortality due to this outbreak was slight, below that experienced in 1922, 1929, 1933 and 1937. This comparison clearly shows that the epidemic wave of 1943 cannot be bracketed with a weakening of the population due to nutritional causes. Food supplies were adequate ; food rationing was relieved by multiple facilities (restaurants, canteens, etc.). It would even appear that, as far as the poorer classes are concerned, the educational steps taken by the Ministry of Food, the allocation of milk and fruit to children and mothers, and the development of school canteens, together with the decrease in unemployment and the increase in real wages, led to a qualitative, if not to a quantitative, improvement of the dietary.

[1] This may have caused an increase in the prevalence of influenza-like and other infections of the respiratory tract, even if it failed to produce a rise in the incidence of the notifiable communicable diseases, against which an adult urban population enjoys a widespread acquired immunity.

England and Wales

Declared war : September 3rd, 1939. Heavy bombing of large towns by the German Air Force from August 1940 to May 1941. *(f)*

Civilian **Population** used for calculating the rates *(h)*.

England and Wales		Towns of more than 50,000 inhabitants	
C. IV.1931 :	39,948,000	C. IV.1931 :	19,837,000
O. N. E. VI.1939 :	41,460,000	O. N. E. VI.1937 :	21,221,285
O. N. E. 1940-1943 :	41,460,000	O. N. E. VI.1938 :	21,244,840
(population VI.1939).		O. N. E. 1939-1943 :	21,244,840
		(population VI.1938).	

London

C. IV.1931 :	4,419,900	O. N. E. VI.1938 :	4,062,800
O. N. E. VI.1937 :	4,094,500	O. N. E. 1939-1943 :	4,062,800
		(population VI.1938).	

Natural Movement of Population.

Year	England and Wales: Births ‰ inhab.	England and Wales: General mortality ‰ inhab. *(g)*	England and Wales: Increase (+) or decrease (−) ‰ inhab.	Towns of more than 50,000 inhabitants: Births ‰ inhab.	Towns of more than 50,000 inhabitants: General mortality *(a) (b)* ‰ inhab. *(g)*	Towns of more than 50,000 inhabitants: Increase (+) or decrease (−) ‰ inhab.
1928-1938 Max.	*16.7*	*13.4*	*+5.0*	*16.9*	*13.7*	*+5.3*
1928-1938 Med.	*15.1*	*12.0*	*+3.0*	*15.0*	*12.2*	*+3.0*
1928-1938 Min.	*14.4*	*11.4*	*+2.1*	*14.4*	*11.6*	*+1.8*
1937	14.9	**12.4**	+2.5	14.9	12.5	+2.4
1938	15.1	11.6	+3.5	15.0	11.7	+3.3
1939	14.9	12.1	+2.8	14.9	11.8	+3.1
1940	14.6	**14.0**	+0.6	14.4	**14.1**	+0.3
1941	14.2	**12.9**	+1.3	12.3	**12.6**	−0.3
1942	15.8	11.6	+4.2	14.2	10.9	+3.3
1943	**16.5**	12.1	+4.4	15.4	11.7	+3.7

C. = Census.

O. N. E. = Official national estimate.

(a) Excluding deaths in the Services (males), from September 3rd, 1939.

(b) Excluding deaths of all members of the Services, from September 20th, 1941.

(e) Including deaths in the Services in the home country, from 1941.

(f) From September 21st 1941, the *Registrar-General's Weekly Return of Births and Deaths* includes a note stating that the continuity of the data given may be impaired by population movements affecting evacuation and reception areas.

(g) Including deaths of civilians due to the war.

(h) Rates given for the years 1940 to 1943 inclusive, for England and Wales, based on the June 1939 population, and for towns on that of June 1938, are provisional. *The Quarterly Return of Births, Deaths and Marriages*, published by the Registrar-General, states that " changes in the total population since 1939 are not sufficient to invalidate these rates to any considerable extent".

(i) Median of the period 1930-1938 only.

(j) From March 31st to June 1st (9 weeks).

(k) From June 2nd to December 28th (30 weeks).

Infant Mortality (deaths of infants under 1 year of age, per 1,000 live births). *(g)*

ENGLAND AND WALES

	Yearly rates	I J	II F	III M	IV A	V M	VI J	VII —	VIII J	IX A	X S	XI O	XII N	XIII D
1928-1938 Max.	*74*		*111*			*62*				*54*			*69*	
1928-1938 Med.	*60*		*80*			*56*				*45*			*61*	
1928-1938 Min.	*53*		*68*			*48*				*41*			*52*	
1937	58		73			54				43			61	
1938	53		68			48				41			52	
1939	50		65			48				40			49	
1940	55		77			46				43			57	
1941	58		75			**59**				43			56	
1942	49		61			47				40			48	
1943	48		60			46				40			52	

TOWNS OF MORE THAN 50,000 INHABITANTS

1928-1938 Max.	*79*	*97*	*136*	*130*	*80*	*68*	*58*	*50*	*51*	*55*	*68*	*71*	*74*	*95*
1928-1938 Med.	*65*	*75*	*80*	*80*	*68*	*59*	*51*	*44*	*45*	*47*	*51*	*54*	*64*	*79*
1928-1938 Min.	*57*	*65*	*69*	*59*	*56*	*48*	*43*	*42*	*39*	*39*	*45*	*41*	*49*	*62*
1937	62	73	70	73	64	57	43	42	41	39	46	49	61	79
1938	57	71	71	59	56	48	45	42	39	40	45	41	49	67
1939	52	70	68	66	62	48	43	40	39	39	46	46	51	67
1940	61	73	90	82	64	53	45	46	47	50	52	56	66	82
1941	**71**	**90**	85	89	**83**	**75**	**64**	**59**	**58**	**52**	55	60	**70**	81
1942	58	81	69	66	59	57	53	46	47	50	54	56	59	64
1943	58	68	68	68	61	53	49	43	47	53	49	54	64	73

General Mortality (rates per 1,000 inhabitants on a yearly basis). *(g)*

ENGLAND AND WALES

	Yearly rates	I J	II F	III M	IV A	V M	VI J	VII —	VIII J	IX A	X S	XI O	XII N	XIII D
1928-1938 Max.	*13.4*		*17.1*			*12.0*				*13.9*			*12.3*	
1928-1938 Med.	*12.0*		*14.6*			*11.7*				*9.7*			*11.7*	
1928-1938 Min.	*11.4*		*9.4*			*10.8*				*9.4*			*11.2*	
1937	**12.4**		**16.2**			11.6				9.7			**12.3**	
1938	11.6		13.6			11.6				**9.9**			11.5	
1939 *(a)*	12.1		15.1			11.7				**9.9**			11.6	
1940 *(a)*	**14.0**		**19.9**			11.6				**10.7**			**13.9**	
1941 *(e)*	**12.9**		**17.5**			**13.6**				9.7			11.0	
1942 *(e)*	11.6		14.8			11.3				9.3			11.0	
1943 *(e)*	12.1		13.5			11.0				9.4			14.6	

TOWNS OF MORE THAN 50,000 INHABITANTS

1928-1938 Max.	*13.7*	*20.6*	*25.0*	*27.5*	*14.7*	*12.4*	*11.1*	*10.0*	*9.3*	*9.8*	*9.9*	*11.0*	*12.3*	*14.7*
1928-1938 Med.	*12.2*	*15.4*	*15.4*	*14.4*	*12.9*	*11.8*	*10.6*	*9.8*	*9.3*	*9.1*	*9.5*	*10.6*	*11.4*	*13.3*
1928-1938 Min.	*11.6*	*12.5*	*12.8*	*13.1*	*11.6*	*10.9*	*10.1*	*9.3*	*8.6*	*8.8*	*9.3*	*9.7*	*10.7*	*11.2*
1937	12.5	**18.5**	15.8	14.0	12.9	11.2	10.3	9.5	9.1	8.8	9.6	10.6	**12.3**	**14.3**
1938	11.7	14.3	12.7	13.0	11.6	**12.0**	**10.9**	9.7	**9.3**	**9.2**	**9.9**	10.4	10.7	13.0
1939 *(a)*	11.8	15.7	15.1	14.1	**13.4**	11.2	10.4	9.6	9.0	**9.4**	**10.1**	**11.3**	11.4	12.6
1940 *(a)*	**14.1**	**22.0**	**22.4**	**17.7**	**13.3**	11.2	**11.0**	**10.0**	**9.7**	**10.2**	**13.9**	**14.5**	**13.7**	**14.6**
1941 *(b)*	**12.6**	**18.1**	**17.8**	**16.2**	**14.6**	**15.6**	**11.4**	**10.0**	8.9	8.8	8.7	9.3	10.8	11.2
1942 *(b)*	10.9	14.2	14.1	13.8	11.5	11.3	9.7	9.0	8.9	8.7	8.9	9.6	11.0	11.3
1943 *(b)*	11.7	13.8	12.8	12.7	11.6	10.8	9.8	9.3	9.1	8.8	9.3	10.2	**14.9**	**19.2**

Tuberculosis Mortality (rates per 100,000 inhabitants on a yearly basis).

	England and Wales					London				
	Yearly rates	Quarterly I	II	III	IV	Yearly rates	Quarterly I	II	III	IV
1928-1938 Max.	*96*	*122*	*99*	*79*	*87*	*109*	*146*	*103*	*84*	*101*
1928-1938 Med.	*82*	*99*	*82*	*66*	*76*	*93*	*105*	*87*	*67*	*81*
1928-1938 Min.	*62*	*68*	*66*	*55*	*59*	*72*	*86*	*73*	*57*	*62*
1937	70	82	72	88	67	79	94	77	58	75
1938	62	69	67	54	59	72	86	73	57	62
1939	62	73	65	52	58	69	101	58	55	63
1940	68	84	69	55	63	65	88	66 (*j*)	41 (*k*)	55
1941	69	82	78	58	59	52	77	50	41	43
1942	62	76	64	51	56	48	61	46	35	49
1943	62	69	63	52	63	51	58	44	45	56
1944							61	48		

Communicable Diseases : Cases notified in England and Wales.

	Median 1928-1938	1937	1938	1939	1940	1941	1942	1943
Typhoid and paratyphoid fevers	*1,781*	1,186	1,322	1,529	2,857	2,325	897	715
Dysentery	*(i) 903*	**4,167**	**4,170**	**1,963**	**2,904**	**6,587**	**7,168**	**7,557**
Diphtheria	*62,774*	65,470	**65,731**	47,698	46,281	50,797	42,583	36,077
Scarlet fever	*105,138*	95,859	99,998	78,277	66,371	59,036	84,782	115,156
Cer.-sp. meningitis	*1,094*	1,157	1,293	1,522	**12,741**	**11,077**	**6,146**	**3,319**
Poliomyelitis	*673*	**791**	**1,517**	**889**	**1,085**	**936**	577	455
Typhus fever	—	1	—	1	—	—	1	—
Smallpox	*631*	4	18	1	2	—	5	—
Notified cases	*172,994*	168,635	174,049	131,880	132,241	130,758	142,159	163,279
Composite epidemic index	100	97.5	100.6	76.2	76.4	75.6	82.2	94.4

Scotland

The health situation in Scotland was little different from that in England. The increase in industrial labour, mobilisation, and the bombing of large towns with its consequences, had the same effects.

As in England, the *birth rate*, which for decades had been steadily declining, has gone up since 1940. *Infant mortality*, higher than in England but on the decline, remained close to the pre-war minimum, apart from a spurt in 1940 and 1941, with which influenza was not unconnected. The same cause was, in part, responsible also for the temporary rise in *general mortality* which took place in 1940 and 1941 (*cf.* graph, page 581).

Statistics for large towns in Scotland, as in England, are to a certain extent obscured by changes in population due, on the one hand, to mobilisation and the evacuation of women and children and,

on the other, to the concentration of labour in war industries. The arrest of the downward trend of the *tuberculosis death rate* was marked in Scotland, as in England. It was particularly noticeable in large towns. The increase recorded did not, however, exceed 20%. A very small wave of diphtheria occurred in Scotland in 1940-1941. The disease then sank below the pre-war levels. Meningitis, after the 1940 wave, is also on the decrease.

The sanitary situation of the country may be considered to be entirely satisfactory.

Scotland

Went to war : September 3rd, 1939. Heavy bombardments of the large towns by the German air force from September 1940 to May 1941.

Civilian **Population** used for calculating the rates.

Scotland	16 Scottish Towns
C. IV.1931 : 4,843,000	C. IV.1931 : 2,453,476
O. N. E. VI.1939 : 5,006,700	O. N. E. VI.1939 : 2,576,200
O. N. E. III.1940 to 1943 : 5,006,700 (population VI.1939).	O. N. E. VI.1940 : 2,408,700
	O. N. E. I.1941 : 2,394,700
	O. N. E. 1942 to 1943 : 2,394,700 (population I.1941).

Natural Movement of Population.

Year	Scotland: Births °/₀₀ inhab.	Scotland: General mortality °/₀₀ inhab.	Scotland: Increase (+) or decrease (−) °/₀₀ inhab.	16 Scottish Towns: Births °/₀₀ inhab.	16 Scottish Towns: General mortality °/₀₀ inhab.	16 Scottish Towns: Increase (+) or decrease (−) °/₀₀ inhab.
1928-1938 Max.	*20.0*	*14.7*	*+6.5*	*20.9*	*15.5*	*+6.8*
1928-1938 Med.	*18.0*	*13.3*	*+5.1*	*18.7*	*13.8*	*+5.3*
1928-1938 Min.	*17.6*	*12.6*	*+3.7*	*18.1*	*12.8*	*+4.2*
1937	17.6	**13.9**	+3.7	18.4	**14.2**	+4.2
1938	17.7	12.6	+5.1	18.5	12.8	+5.7
1939	17.4	12.9	+4.5	17.9	13.0	+4.9
1940	17.3	**14.5**	+2.8	17.3	**14.6**	+2.7
1941	17.9	**14.5**	+3.4	18.6	**15.1**	+3.5
1942	18.1	13.0	+5.1	18.7	13.6	+5.1
1942 (I.I-30.IX)	18.3	13.2	+5.1			
1943 (I.I-30.IX)	**19.2**	12.8	+6.4	19.9	13.8	+6.1

C. = Census. O. N. E. = Official national estimate.
E. S. = Estimate of the Epidemiological Intelligence Service of the L. o. N.

(a) From September 1939, deaths of members of the Services and those of civilians due to the war are included in the rates for Scotland, but are excluded from statistics for towns.

(b) Excluding deaths due to the war.

(f) Approximate rates for the first and third periods of four weeks.

Infant Mortality (deaths of infants under 1 year of age, per 1,000 live births).

SCOTLAND

	Yearly rates	I J	II F	III M	IV A	V M	VI J	VII —	VIII J	IX A	X S	XI O	XII N	XIII D
1928-1938 Max.	*87*		*128*			*82*				*69*			*93*	
1928-1938 Med.	*82*		*108*			*73*				*60*			*86*	
1928-1938 Min.	*70*		*80*			*65*				*55*			*75*	
1937	80		97			75				60			**93**	
1938	70		80			65				58			75	
1939	69		88			61				57			69	
1940	78		**112**			67				57			76	
1941	83		109			**84**				63			75	
1942	69		90			64				59			65	
1943			75			58				55				

16 SCOTTISH TOWNS *(b)*

1928-1938 Max.	*98*	*155*	*170*	*151*	*119*	*102*	*83*	*80*	*77*	*86*	*87*	*96*	*127*	*141*
1928-1938 Med.	*91*	*114*	*120*	*113*	*98*	*85*	*73*	*67*	*65*	*68*	*71*	*82*	*102*	*113*
1928-1938 Min.	*77*	*89*	*83*	*83*	*81*	*72*	*71*	*58*	*57*	*51*	*64*	*70*	*91*	*97*
1937	91	109	103	**120**	85	77	72	**78**	65	68	73	**93**	**127**	124
1938	77	95	83	83	81	72	**78**	60	68	52	66	70	91	98
1939	75	102	99	82	74	61	65	62	60	62	77	83	77	82
1940	84	119	131	107	82	80	65	58	64	62	72	75	91	89
1941	96	97	101	**165**	**129**	91	**81**	**73**	69	69	76	77	91	121
1942	79	101	101	85	79	78	68	61	**71**	71	72	74	85	79
1943 *(f)*	72	83	80	80	74	61	62	57	58	61	66	76	89	99
1944		92	74											

General Mortality (rates per 1,000 inhabitants on a yearly basis). *(a)*

SCOTLAND

	Yearly rates	I J	II F	III M	IV A	V M	VI J	VII —	VIII J	IX A	X S	XI O	XII N	XIII D
1928-1938 Max.	*14.7*		*22.1*			*13.7*				*11.1*			*13.9*	
1928-1938 Med.	*13.3*		*16.2*			*13.1*				*10.9*			*13.1*	
1928-1938 Min.	*12.6*		*14.2*			*12.4*				*10.6*			*12.2*	
1937	**13.9**		**18.0**			12.7				10.9			**13.9**	
1938	12.6		14.2			12.5				**11.0**			12.8	
1939	12.9		15.4			12.6				10.8			12.8	
1940	**14.5**		**20.8**			12.9				**11.3**			13.2	
1941	**14.5**		**18.9**			**15.2**				**11.5**			12.5	
1942	13.0		15.7			13.1				10.9			12.2	
1943			14.4			12.7				**11.3**				

16 SCOTTISH TOWNS

1928-1938 Max.	*15.5*	*27.9*	*28.4*	*18.7*	*16.3*	*15.2*	*13.0*	*12.3*	*11.3*	*11.6*	*11.8*	*12.9*	*14.9*	*18.5*
1928-1938 Med.	*13.8*	*17.3*	*16.7*	*16.7*	*14.9*	*14.1*	*12.6*	*11.6*	*10.8*	*10.5*	*11.2*	*12.1*	*13.3*	*15.1*
1928-1938 Min.	*12.8*	*13.8*	*13.4*	*13.8*	*12.8*	*12.3*	*11.9*	*11.0*	*8.9*	*10.1*	*10.7*	*11.4*	*12.7*	*14.1*
1937	**14.2**	**22.8**	17.4	15.8	14.1	12.3	12.1	11.6	**11.2**	10.3	**11.7**	12.3	**14.6**	**16.7**
1938	12.8	17.3	13.4	13.8	13.2	13.2	12.7	11.6	**11.2**	10.6	10.9	12.1	12.7	15.1
1939	13.0	18.3	15.8	13.3	13.9	12.4	11.8	10.5	11.0	10.7	**11.6**	**13.3**	12.5	13.8
1940	**14.6**	**25.7**	**25.1**	16.8	13.7	13.1	12.9	11.4	**11.5**	10.3	**11.4**	11.7	12.9	13.9
1941	**15.1**	**20.8**	**20.4**	**19.1**	**17.5**	**15.2**	**14.3**	**12.8**	**11.9**	**11.6**	11.2	11.6	13.6	13.5
1942	13.6	16.4	17.5	16.8	14.8	14.4	12.7	**12.1**	**11.6**	**11.0**	**11.4**	11.9	13.5	12.8
1943 *(f)*	13.8	15.7	15.8	14.3	13.8	13.5	12.6	**12.5**	**11.3**	**11.0**	**11.9**	**13.4**	**16.4**	**17.8**
1944		16.3	14.2											

Tuberculosis Mortality (rates per 100,000 inhabitants on a yearly basis).

	Scotland					16 Scottish Towns				
	Yearly rates	Quarterly I	II	III	IV	Yearly rates	Quarterly I	II	III	IV
1928-1938 Max.	*97*	*117*	*109*	*87*	*90*	*111*	*143*	*123*	*100*	*100*
1928-1938 Med.	*80*	*97*	*85*	*66*	*70*	*94*	*110*	*100*	*80*	*85*
1928-1938 Min.	*69*	*77*	*76*	*61*	*61*	*87*	*96*	*95*	*74*	*80*
1937	74	86	80	64	65	91	107	97	76	84
1938	69	77	76	61	61	87	98	95	75	80
1939	70	81	76	62	64	89	100	94	77	85
1940	80	95	86	66	73	**109**	...	...	...	**95**
1941	83	94	95	72	72	**110**	**127**	**125**	**92**	**97**
1942	80	87	92	70	71	**105**	117	**122**	87	**97**
1943	79	82	89	70	75	**102**	109	**114**	87	**97**
1944		88					113			

Communicable Diseases : Cases notified in Scotland.

	Median 1932-1938	1937	1938	1939	1940	1941	1942	1943
Typhoid and paratyphoid fevers	*359*	260	359	368	**872**	**1,093**	235	221
Dysentery	*482*	**951**	**2,468**	**1,430**	**2,165**	**2,575**	**2,637**	**3,425**
Diphtheria	*10,389*	10,389	10,787	9,942	**15,707**	**13,697**	10,591	9,280
Scarlet fever	*22,755*	20,584	19,773	12,282	6,848	9,269	14,267	15,128
Cer.-sp. meningitis	*426*	444	**464**	**491**	**2,936**	**2,553**	**1,605**	**1,295**
Poliomyelitis	*37*	30	**201**	46	128	**163**	55	49
Typhus fever	—	—	—	—	**7**	—	—	—
Smallpox	—	—	—	—	—	—	**116**	1
Notified cases	*34,448*	32,658	34,052	24,559	28,663	29,350	29,506	29,399
Composite epidemic index	100	94.8	98.9	71.3	83.2	85.2	85.7	85.3

Eire

Although a neutral country, Eire has felt the economic and nutritional repercussions of the war, as a result of her economic ties with the United Kingdom and also of the crisis in maritime shipping that affected her also.

A large increase in the number of *births* occurred in 1942 and 1943. *Infant mortality* was relatively high in 1941, and was still higher in 1943, when it broke the record for the last 20 years. *General mortality* for the same years 1941 and 1943 was also slightly above the normal (*cf.* graph, page 581). The influenza epidemic at the end of 1943 was a contributing factor, as also was the high infant mortality during the whole of the year. After being stationary up to

1941, *mortality* due to *tuberculosis*, for the whole of the country, was high in 1942 and 1943. This phenomenon is above all attributable to the increase of the disease in the large towns. It is well known that mortality due to tuberculosis is a sensitive index of economic disturbances and inadequate nutrition. The same is true of the recurrence of typhus fever in old endemic rural areas.

Apart from this and a very slight tendency for diphtheria to increase in 1940, Eire's situation from the point of view of *contagious disease* has been quite normal.

EIRE

Neutral country.

Population used for calculating the rates.

EIRE		TOWNS IN EIRE	
C. IV.1936 :	2,968,000	C. IV.1936 :	750,488
O. N. E. VI.1939 :	2,934,000	O. N. E. VI.1939 :	775,611
O. N. E. VI.1940 :	2,958,000	O. N. E. VI.1940 :	781,011
O. N. E. VI.1941 :	2,993,000	O. N. E. VI.1941 :	785,511
O. N. E. VI.1942 :	2,963,000	O. N. E. VI.1942 :	781,017
O. N. E. VI.1943 :	2,951,000	O. N. E. VI.1943 :	793,017

Natural Movement of Population.

Year	EIRE Births ‰ inhab.	EIRE General mortality ‰ inhab.	EIRE Increase (+) or decrease (−) ‰ inhab.	TOWNS IN EIRE Births ‰ inhab.	TOWNS IN EIRE General mortality ‰ inhab.	TOWNS IN EIRE Increase (+) or decrease (−) ‰ inhab.
1928-1938 *Max.*	*20.1*	*15.3*	*+6.3*	*(b) 25.8*	*15.4*	*+11.3*
1928-1938 *Med.*	*19.5*	*14.2*	*+5.6*	*(b) 24.3*	*14.7*	*+ 9.9*
1928-1938 *Min.*	*19.1*	*13.2*	*+3.9*	*(b) 23.6*	*13.0*	*+ 8.8*
1937	19.2	**15.3**	+3.9	23.7	14.7	+ 9.0
1938	19.4	13.6	+5.8	23.6	13.0	+10.6
1939	19.1	14.2	+4.9	22.9	13.0	+ 9.9
1940	19.1	14.2	+4.9	22.3	14.1	+ 8.2
1941	19.0	**14.6**	+4.4	22.6	14.3	+ 8.3
1942	**22.3**	14.1	+8.2	**25.0**	13.8	+11.2
1943	**22.3**	**14.7**	+7.6	**25.0**	14.3	+10.7

C. = Census.

O. N. E. = Official national estimate.

(a) For want of older data, cerebrospinal meningitis and poliomyelitis were not taken into consideration when establishing the epidemic index.

(b) Rates based on the years 1930-1938.

Infant Mortality (deaths of infants under 1 year of age, per 1,000 live births).

EIRE

	Yearly rates	I J	II F	III M	IV A	V M	VI J	VII —	VIII J	IX A	X S	XI O	XII N	XIII D
1928-1938 Max.	*74*		*91*			*73*				*62*			*84*	
1928-1938 Med.	*68*		*81*			*67*				*56*			*68*	
1928-1938 Min.	*63*		*78*			*58*				*50*			*57*	
1937	**73**		**91**			**71**				57			69	
1938	67		81			64				55			66	
1939	66		82			64				53			64	
1940	66		78			63				51			**75**	
1941	**74**		84			**72**				**65**			**74**	
1942	69		81			63				58			**79**	
1943	**80**		**94**			**75**				**71**			**81**	

TOWNS OF EIRE

	Yearly rates	I J	II F	III M	IV A	V M	VI J	VII —	VIII J	IX A	X S	XI O	XII N	XIII D
1930-1938 Max.	*103*	*155*	*145*	*144*	*142*	*98*	*89*	*75*	*82*	*101*	*119*	*129*	*117*	*164*
1930-1938 Med.	*90*	*103*	*118*	*127*	*107*	*83*	*70*	*66*	*72*	*85*	*90*	*92*	*83*	*112*
1930-1938 Min.	*79*	*70*	*97*	*96*	*77*	*66*	*51*	*59*	*55*	*69*	*67*	*63*	*75*	*78*
1937	**99**	**155**	**127**	**144**	**112**	83	70	66	**80**	76	99	81	**104**	112
1938	89	101	**145**	119	107	66	**76**	63	62	72	84	94	83	85
1939	83	**122**	99	81	85	73	65	**72**	63	73	93	62	98	109
1940	88	100	123	109	81	77	73	61	73	79	82	72	100	134
1941	**106**	**117**	107	89	**112**	**83**	**85**	**81**	**93**	**121**	**124**	**146**	**141**	93
1942	97	89	110	93	**113**	**87**	63	52	**83**	**91**	102	**107**	**113**	**132**
1943	**111**	**135**	**147**	118	110	**99**	**82**	**82**	**82**	**126**	**110**	**113**	**102**	139

General Mortality (rates per 1,000 inhabitants on a yearly basis).

EIRE

	Yearly rates	I J	II F	III M	IV A	V M	VI J	VII —	VIII J	IX A	X S	XI O	XII N	XIII D
1928-1938 Max.	*15.3*		*20.8*			*15.3*				*11.9*			*13.9*	
1928-1938 Med.	*14.2*		*17.2*			*14.6*				*11.6*			*13.1*	
1928-1938 Min.	*13.2*		*15.2*			*13.0*				*10.9*			*12.1*	
1937	*15.3*		**20.8**			**15.2**				**11.7**			**13.4**	
1938	13.6		15.6			13.4				**11.7**			**13.9**	
1939	14.2		18.1			14.1				**11.7**			12.9	
1940	14.2		18.1			14.2				11.4			13.0	
1941	**14.6**		**19.6**			**15.1**				11.6			12.2	
1942	14.1		16.4			14.6				11.5			**13.8**	
1943	**14.7**		17.1			14.8				**12.2**			**14.8**	

TOWNS OF EIRE

	Yearly rates	I J	II F	III M	IV A	V M	VI J	VII —	VIII J	IX A	X S	XI O	XII N	XIII D
1930-1938 Max.	*15.4*	*22.4*	*24.6*	*20.6*	*18.8*	*16.8*	*14.6*	*13.6*	*11.6*	*12.9*	*13.4*	*13.6*	*15.3*	*18.6*
1930-1938 Med.	*14.7*	*17.4*	*18.8*	*18.6*	*16.8*	*14.4*	*13.0*	*11.8*	*11.4*	*11.8*	*11.7*	*12.6*	*14.4*	*15.4*
1930-1938 Min.	*13.0*	*15.0*	*15.4*	*15.5*	*13.3*	*12.9*	*12.3*	*10.3*	*10.3*	*10.6*	*11.1*	*11.2*	*12.7*	*13.7*
1937	14.7	**22.4**	**24.5**	18.5	**17.3**	13.3	12.3	11.1	10.6	10.8	11.7	11.2	12.7	15.2
1938	13.0	16.0	15.4	15.5	13.3	12.9	12.3	11.1	10.3	11.0	11.3	12.0	13.0	15.3
1939	13.0	**19.0**	15.2	14.5	14.1	11.9	12.9	11.4	10.5	11.6	11.0	11.3	13.1	13.3
1940	14.1	**21.3**	21.4	18.4	14.8	13.8	13.0	11.9	10.5	9.9	11.7	11.0	12.9	12.9
1941	14.3	**19.9**	18.1	17.5	14.9	14.4	**13.2**	12.1	**11.7**	11.3	12.0	11.4	13.1	**16.1**
1942	13.8	15.5	16.1	17.9	15.2	14.4	**13.4**	11.1	10.7	11.6	**12.4**	12.5	14.4	15.0
1943	14.3	17.6	17.1	16.1	15.1	14.5	12.5	12.1	11.2	11.7	**12.1**	13.1	14.7	**17.9**

Tuberculosis Mortality (rates per 100,000 inhabitants on a yearly basis).

	Eire					Towns of Eire				
	Yearly rates	Quarterly I	II	III	IV	Yearly rates	Quarterly I	II	III	IV
1928-1938 Max.	*137*	*157*	*163*	*123*	*115*	*1932-1938* Max.	*173*	*197*	*203*	*146*	*158*
1928-1938 Med.	*125*	*143*	*141*	*114*	*108*	*1932-1938* Med.	*155*	*175*	*182*	*139*	*131*
1928-1938 Min.	*109*	*121*	*122*	*94*	*98*	*1932-1938* Min.	*144*	*154*	*150*	*124*	*119*
1937	123	143	140	111	100	1937	155	**197**	182	124	119
1938	109	121	122	94	101	1938	144	154	150	127	**147**
1939	113	124	129	104	94	1939	140	149	163	124	123
1940	125	139	**154**	110	95	1940	**165**	**204**	**199**	132	127
1941	124	141	133	112	**110**	1941	155	180	168	127	144
1942	**147**	**157**	**175**	**130**	**125**	1942	**188**	**207**	**221**	**162**	**163**
1943	**143**	**163**	**171**	**122**	**117**	1943	**177**	**216**	195	153	**147**
						1944		**189**			

Communicable Diseases : Cases notified in Eire.

	Median 1928-1938	1937	1938	1939	1940	1941	1942	1943
Typhoid fever	*404*	415	254	386	253	284	415	411
Paratyphoid fevers.	*14*	**22**	14	14	12	3	9	8
Diphtheria	*2,960*	2,508	2,979	2,087	1,891	1,447	2,949	**4,650**
Cer.-sp. meningit. *(a)*	...	...	...	...	88	126	127	141
Scarlet fever	*3,282*	**4,564**	**3,942**	2,778	2,465	2,318	2,747	2,264
Poliomyelitis *(a)*	...	...	...	...	10	86	375	114
Typhus fever	*14*	—	—	5	11	26	28	20
Smallpox	—	—	—	—	—	—	—	—
Notified cases *(a)*	*6,674*	7,509	7,189	5,270	4,632	4,078	6,148	7,353
Composite epidemic index *(a)*	100	112.5	107.7	79.0	69.4	61.1	92.1	110.2

Spain

The present nutritional and sanitary situation in Spain cannot be described without reference to the effects of the civil war, which lasted from July 1936 to April 1939.

It is true that Spain, in spite of her non-belligerence, has suffered from restrictions in imports of foodstuffs owing to the lack of adequate shipping, financial and other difficulties in international trade, etc., but there is no doubt that the hardships entailed by these import restrictions would have been of far less importance to the country if the second world war had found Spain in a sound social, economic, agricultural—and, consequently, nutritional—situation.

It is well known that food deficieney has a cumulative effect and that the lack or insufficieney of certain nutrients can be borne without the appearance of symptoms—or at any rate serious symptoms—during a certain time, but not indefinitely.

We do not propose to give here a full account of the food shortage which was felt by a large part of the Spanish population during the civil war, chiefly on the Republican side, and particularly in Madrid during the winters of 1937/38, 1938/39. It gave rise to a considerable literature, among the authors of whieh GRANDE, COVIAN, JIMENEZ, GARCIA and RUIZ-GIJON deserve particular mention.[1]

In Madrid from August 1937 to February 1939, the food ration amounted to some 800 to 1,000 calories, including scarcely any meat. Two-thirds of the population lost 30% of their initial weight.[2]

Then appeared a series of deficiency diseases which ean be listed as follows in their order of frequency (among patients treated): paresthetic-eausalgic syndrome (22.8%), pellagra in its various forms (21.7%), retrobulbaroptic neuritis (16.3%), famine œdema (14.7%), glossitis (10.7%), hæmorrhages (2.2%), auditory neuritis (1.5%), other deficiency diseases (10.1%).[2]

It is interesting to note that famine œdema reached a culminating point from eight to ten months after neuritic and pellagric symptoms had manifested themselves. The general opinion as to its causation is that it is due to a protein and not a vitamin insufficiency. Indeed, its maximum occurred in January 1939, at a time when the amount of protein in the ration was exceptionally low—15 g. per day, as against 25.5 g. during the preceding winter.[2] Very few cases of insufficient intake of vitamin A (hemeralopia), C (scurvy) and D (riekets) were observed. Thus, over four-fifths of the deficiency diseases which occurred in Madrid during the civil war resulted from an insufficient supply of the B-vitamin complex, and about 17% were due to an insuffieient protein ration.

Since the re-establishment of internal peace in Spain, of course, food is more evenly distributed than during the civil war and, although official rationing has favoured the poorer elements of the

[1] A guide to this literature will be found in " L'alimentation et les troubles alimentaires durant la guerre civile espagnole ". *Les Vitamines*, Hoffmann-La Roche, Bâle, 1944, N° 1, 1-9.

[2] *Loc. cit.*

population,[1] widespread economic distress in the towns results in a not inconsiderable proportion of the population being in a state of actual want, in spite of many efforts at relief.

General mortality (*cf.* graph, page 600) has followed fairly closely the variations in the situation of the country. Whereas, during the quinquennium 1930-1935 it averaged 16.4‰ (it was even down to 15.5‰ in 1935), the rate rose gradually during the civil war, reaching a peak in 1938 with 19.2‰. There was a fall in 1939, as the war went on during only one quarter of that year, and the rate dropped to 16.6‰ in 1940. Another rise in 1941 was due partly to renewed stringency in food supplies and also to the very large number of infant deaths that naturally followed the tremendous temporary rise in the birth rate which, in 1940, followed the return to peace conditions. The 1940 excess of births over 1939 was 50%, the excess of infant mortality in 1941, as against 1940, was over 30%. This is a clear example of a spurious apparent decrease in infant mortality during a year of high natality, followed the next year by a spurious increase in the infant death rate due merely to the increased number of children at risk. During the civil war the *infant death rate* had been consistently high, with a maximum in 1939.

Tuberculosis mortality also went up until 1939, and rose again in 1941 and 1942. In Madrid, pulmonary tuberculosis showed an even greater rise in the course of the civil war (56%), an abrupt fall at the end of it, and a fresh rise during the economic crisis in 1942.

The *birth rate*, though dwindling, was high in the pre-war period and fell gradually during the civil war to a minimum of 16.5 in 1939. After an abrupt rise following the end of hostilities, consummation of deferred marriages, etc., it has resumed its pre-war downward trend. The natural increase of 10‰ in the early 'thirties, after being reduced to nil at the end of the civil war, has gone up to half its pre-war value. It will be recalled that, in Madrid, the *excess of deaths* over births was as high as nearly 12‰ in 1939.

[1] The poor are entitled to larger rations of cheap staple foodstuffs such as bread to compensate the fact that the wealthy may procure more expensive foods not subject to rationing restrictions.

Accurate figures are unfortunately lacking for the civil war years, but those available show, contrary to expectation, the absence of any major *epidemic* apart from typhus. It was only at the close of hostilities that typhus fever broke out in epidemic form, naturally starting from the prisons. The worst of the epidemic occurred in 1941 in Madrid, and in 1942 the disease invaded Catalonia, Barcelona suffering heavily. In 1943, the disease dwindled

Spain

Civil war from July 27th, 1936, to April 1939.
At first non-belligerent, then neutral during the second world war.

Population used for calculating the rates.

Spain			Madrid		
C.	XII.1930 :	23,564,000	C.	XII.1930 :	952,830
O.N.E.	VI.1939 :	25,397,700	O.N.E.	VI.1939 :	1,167,000
O.N.E.	VI.1940 :	25,636,700	O.N.E.	VI.1940 :	1,167,000
O.N.E.	VI.1941 :	25,878,000	O.N.E.	VI.1941 :	1,089,000
E.S.	VI.1942 :	25,995,000	O.N.E.	VI.1942 :	1,089,000
E.S.	VI.1943 :	26,121,000	E.S.	VI.1943 :	1,096,000

Natural Movement of Population.

Year	Spain: Births ‰ inhab.	Spain: General mortality ‰ inhab.	Spain: Increase (+) or decrease (−) ‰ inhab.	Madrid: Births ‰ inhab.	Madrid: General mortality ‰ inhab.	Madrid: Increase (+) or decrease (−) ‰ inhab.
1928-1938 *Max.*	*29.1*	*19.2*	*+11.8*	*24.3*	*21.5*	*+ 8.6*
1928-1938 *Med.*	*27.6*	*16.9*	*+10.3*	*22.9*	*17.8*	*+ 5.7*
1928-1938 *Min.*	*20.1*	*15.7*	*+ 0.9*	*14.5*	*15.0*	*− 6.6*
1935	25.9	15.7	+10.2	20.7	15.0	+ 5.7
1936	24.9	16.7	+ 8.2	20.2	**21.4**	− 1.2
1937	22.7	**18.9**	+ 3.8	15.1	**21.5**	− 6.4
1938	20.1	**19.2**	+ 0.9	14.5	**21.1**	− 6.6
1939	16.5	**18.5**	− 2.0	10.9	**22.3**	−11.4
1940	24.5	16.6	+ 7.9	22.9	15.9	+ 7.0
1941	19.6	**18.7**	+ 0.9	18.5	16.1	+ 2.4
1942	20.3	14.8	+ 5.5	17.9	14.8	+ 3.1
1942 (I-IV)..	19.8	17.5	+ 2.3	18.8	18.9	− 0.1
1943 (I-IV)..	24.2	13.9	+10.3	21.6	14.0	+ 7.6

C. = Census. O. N. E. = Official national estimate.
E. S. = Estimate of the Epidemiological Intelligence Service of the L.o.N.

(a) Monthly rates based on the years 1929-1935.
(b) Quarterly rates based on the years 1929-1935.
(e) Corrected yearly rates ; quarterly rates based on the years 1930-1938.
(f) April-December only.
(g) Very incomplete data during the civil war (July 1936-April 1939).
(h) Provisional rates.

Infant Mortality (deaths of infants under 1 year of age, per 1,000 live births).

SPAIN

	Yearly rates	I J	II F	III M	IV A	V M	VI J	VII —	VIII J	IX A	X S	XI O	XII N	XIII D
1928-1938 Max(b)	*130*		*106*			*120*				*168*			*164*	
1928-1938 Med(b)	*113*		*100*			*108*				*144*			*96*	
1928-1938 Min(b)	*108*		*89*			*98*				*102*			*87*	
1937	**130**		...			...				...			...	
1938	**121**		90			**131**				**162**			**106**	
1939	**136**		**105**			**127**				**201**			**119**	
1940	109		80			107				150			**106**	
1941	**143**		**126**			116				**206**			**128**	
1942	103		92			94				130			95	
1943	...		92			...				...			...	

MADRID

1928-1938 Max(a)	*118*	*175*	*180*	*150*	*103*	*109*	*161*		*191*	*133*	*100*	*97*	*90*	*105*
1928-1938 Med(a)	*101*	*132*	*119*	*92*	*86*	*67*	*109*		*154*	*117*	*79*	*68*	*60*	*91*
1928-1938 Min(a)	*91*	*123*	*104*	*70*	*67*	*62*	*83*		*133*	*93*	*77*	*62*	*57*	*64*
1937	96	...	...	...	...	...	...		...	...	...	...	...	...
1938	91	88	66	80	**120**	**101**	109		120	93	77	**77**	**89**	72
1939	**139**	88	125	**177**	**169**	**105**	**168**		**264**	**160**	**119**	**84**	**115**	**127**
1940	89	106	93	73	69	70	92		120	113	65	68	**95**	**117**
1941	108	**197**	147	76	83	**75**	70		118	**150**	**116**	**79**	**91**	81
1942	91	109	146	74	64	69	97		116	115	71	**74**	57	**105**
1943	...	107	60	62	56	...	...		...	...	...	...	...	...

General Mortality (rates per 1,000 inhabitants on a yearly basis).

SPAIN

	Yearly rates	I J	II F	III M	IV A	V M	VI J	VII —	VIII J	IX A	X S	XI O	XII N	XIII D
1928-1938 Max(a)	*19.2*	*21.7*	*24.8*	*20.2*	*17.8*	*16.0*	*17.1*		*18.8*	*17.5*	*16.2*	*16.0*	*16.0*	*17.9*
1928-1938 Med(a)	*16.9*	*18.6*	*20.5*	*19.2*	*16.1*	*15.0*	*15.3*		*17.2*	*15.9*	*14.6*	*14.3*	*15.4*	*15.9*
1928-1938 Min(a)	*15.7*	*14.7*	*19.0*	*17.8*	*14.8*	*13.7*	*14.0*		*15.6*	*15.1*	*13.5*	*13.6*	*14.2*	*14.8*
1935	15.7	14.7	20.5	19.2	15.4	13.7	14.0		16.1	15.3	13.5	13.6	14.2	15.1
1936	16.7	14.5	...	...	...	...	...		...	...	...	...	...	...
1937	**18.9**	...	...	...	...	...	...		...	...	...	...	...	...
1938	**19.2**	**21.0**	19.4	17.6	**16.6**	**17.5**	**16.9**		16.1	16.2	14.9	15.2	**16.6**	**19.2**
1939	**18.5**	**21.5**	21.3	**19.7**	**18.7**	**18.0**	16.2		18.3	16.8	**16.4**	**16.5**	**18.4**	**19.0**
1940	16.6	20.0	18.6	16.4	16.2	**15.9**	15.5		16.1	15.3	14.4	14.8	15.3	**19.1**
1941	**18.7**	**22.6**	20.8	**21.0**	**20.9**	**18.1**	**16.9**		18.2	**17.7**	**16.7**	**16.8**	**17.7**	17.2
1942	14.8	18.7	19.2	17.7	14.3	13.0	13.0		14.1	13.7	13.2	13.2	13.5	15.0
1943	...	15.0	14.7	14.0	11.8	...	...		...	...	...	...	...	...

MADRID

1928-1938 Max(a)	*21.5*	*31.2*	*25.7*	*20.8*	*17.8*	*15.9*	*18.0*		*19.0*	*15.8*	*13.9*	*17.4*	*19.1*	*19.8*
1928-1938 Med(a)	*17.8*	*22.8*	*22.8*	*17.6*	*14.9*	*14.3*	*14.1*		*15.3*	*13.2*	*12.1*	*13.4*	*15.6*	*18.1*
1928-1938 Min(a)	*15.0*	*19.3*	*21.0*	*15.8*	*13.5*	*11.7*	*12.8*		*14.4*	*11.1*	*11.6*	*12.5*	*13.4*	*15.4*
1937	**21.5**	...	...	...	...	...	...		...	...	...	...	...	...
1938	**21.1**	25.5	23.3	**23.0**	**23.0**	**20.1**	**18.1**		17.6	**16.7**	**16.7**	**17.0**	**21.1**	**24.8**
1939	**22.3**	28.2	**30.9**	**31.2**	**25.7**	**18.6**	14.4		16.8	**16.3**	**18.3**	**23.2**	**22.1**	**22.6**
1940	15.9	24.7	**25.2**	18.5	15.2	15.0	15.0		14.2	12.3	9.6	12.0	13.1	16.8
1941	16.1	21.9	18.7	16.7	16.3	14.5	13.4		14.3	14.0	12.9	14.9	17.8	17.7
1942	14.8	20.3	22.4	19.4	13.5	12.5	12.7		13.2	12.5	11.7	12.1	12.8	15.5
1943	...	17.6	13.4	13.5	11.6	...	...		...	...	...	...	...	...

to a few hundred cases scattered over 10 provinces. A further reduction even took place in 1944.

Smallpox, of which hundreds of cases, mostly of the mild type, occur every year, reached a peak of 1,878 cases in 1940, gradually falling to some 200 cases in 1943. During the years following the civil war, typhoid endemicity remained higher than usual.

The figures and information available show that the sanitary situation of Spain has been unfavourable during the years following the end of the civil war, although it is gradually improving. The return of general and infant mortality to normal suggests that food conditions have improved of late, at any rate so far as the bulk of the population is concerned.

Tuberculosis Mortality (rates per 100,000 inhabitants on a yearly basis).

	Spain (Tuberculosis, all forms.)					Madrid (Pulmonary tuberculosis.)				
	Yearly rates	Quarterly I	II	III	IV	Yearly rates	Quarterly I	II	III	IV
1928-1938 *Max(b)*	*135*	*146*	*142*	*128*	*121*	*164 (e)*	*190*	*166*	*150*	*154*
1928-1938 *Med(b)*	*120*	*129*	*121*	*114*	*111*	*129 (e)*	*146*	*127*	*106*	*118*
1928-1938 *Min(b)*	*102*	*117*	*113*	*102*	*98*	*98 (e)*	*96*	*96*	*87*	*105*
1937	120	...	...	...	...	100	96	96	97	112
1938	129	...	...	...	...	156	**165**	**157**	**148**	**154**
1939	122	**140**	130	108	112	136	**198**	140	96	110
1940	113	120	115	109	109	84	95	75	55	88
1941	125	116	**131**	**126**	**127**	105	97	106	103	116
1942	124	135	127	115	119	134	133	**146**	121	136
1943	...	122	...	...	...	120	145(*h*)	120(*h*)	110	108

Communicable Diseases : Cases notified in Spain.

	Median 1931-1935	1937	1938	1939	1940	1941	1942	1943
		(g)	*(g)*	*(g)*				
Typhoid and paratyphoid fevers	*16,740*	...	...	...	**27,557**	**33,928**	**31,221**	...
Dysentery	*1,040*	...	...	...	1,085	2,013	764	...
Diphtheria	*8,548*	...	...	...	**24,769**	**12,397**	8,020	...
Scarlet fever	*9,281*	...	...	...	4,208	2,399	2,756	...
Cer.-sp. meningitis	*249*	...	...	...	361	344	424	...
Poliomyelitis	*321*	...	...	...	373	233	**738**	...
Typhus fever	*7*	...	...	66 *(f)*	3	**7,090**	**4,061**	**648**
Smallpox	*688*	...	...	831 *(f)*	**1,878**	678	371	210
Notified cases	*36,874*	...	...	...	60,234	59,082	48,355	...
Composite epidemic index	100	...	...	...	163.4	160.2	131.1	...

PORTUGAL

In spite of her neutrality, Portugal has been affected economically by the war, shortage of shipping reducing her imports from overseas and some of the foodstuffs she produces being drained from the country by offers of high prices (oil, canned fish).

This situation may have had an effect on the food consumption and the morbidity and mortality of Portugal, as was the case during the first world war in the Netherlands and in Denmark, which were

PORTUGAL

Neutral country.

Population used for calculating the rates.

PORTUGAL *(a)*			LISBON		
C.	XII.1930 :	6,826,000	C.	XII.1930 :	594,390
E. S.	VI.1939 :	7,590,000	E. S.	VI.1939 :	687,100
E. S.	VI.1940 :	7,702,000	E. S.	VI.1940 :	702,600
O. N. E.	VI.1941 :	7,761,000	E. S.	VI.1941 :	708,700
E. S.	VI.1942 :	7,862,000	E. S.	VI.1942 :	714,900
E. S.	VI.1943 :	7,925,000	E. S.	VI.1943 :	721,000

Natural Movement of Population.

Year		PORTUGAL Births ‰ inhab.	General mortality ‰ inhab.	Increase (+) or decrease (−) ‰ inhab.	LISBON Births ‰ inhab.	General mortality ‰ inhab.	Increase (+) or decrease (−) ‰ inhab.
1930-1938	*Max.*	*29.9*	*17.2*	*+12.9*	*21.3*	*20.5*	*+1.8*
	Med.	*28.4*	*16.8*	*+11.8*	*19.5*	*19.3*	*−0.5*
	Min.	*26.6*	*15.4*	*+11.0*	*16.1*	*18.0*	*−2.4*
1937		26.8	15.8	+11.0	16.7	18.3	−1.6
1938		26.6	15.4	+11.2	16.1	18.5	−2.4
1939		26.2	15.3	+10.9	15.3	16.2	−0.9
1940		24.4	15.6	+ 8.8	13.5	17.3	−3.8
1941		23.7	**17.4**	+ 6.3	14.1	17.2	−3.1
1942		23.8	16.1	+ 7.7	13.4	15.9	−2.5
1942 (I.I-30.IX)		23.5	15.8	+ 7.7	13.5 *(j)*	15.8 *(j)*	−2.3 *(j)*
1943 (1.1-30.IX)		25.1	15.3	+ 9.8	13.5 *(k)*	15.2 *(k)*	−1.7 *(k)*

C. = Census.
E. S. = Estimate of the Epidemiological Intelligence Service of the L.o.N.
O. N. E. = Official national estimate.

(a) Including adjacent islands. *(b)* Corrected yearly rates. *(d)* Deaths.
(e) 11 months. *(f)* 48 weeks.
(g) As comparable retrospective data are lacking, quarterly rates do not indicate whether the upper quartile has been exceeded.
(h) On the basis of 7 cases for each death in 1941, the number of cases of typhus fever has been estimated as 42 in 1940 and 35 in 1942.
(i) Median based on 1936-1938.
(j) 1.I.-31.X.1942. *(k)* 1.I.-31.X.1943.

Infant Mortality (deaths of infants under 1 year of age, per 1,000 live births).

	Portugal					Lisbon					
	Yearly rates	Quarterly *(g)* I	II	III	IV		Yearly rates	Quarterly I	II	III	IV
1930-1938 Max.	*151*	...	...	...	...	*1930-1938* Max.	*220*	...	...	...	...
1930-1938 Med.	*144*	...	...	...	...	*1930-1938* Med.	*199*	...	...	...	...
1930-1938 Min.	*137*	...	...	...	...	*1930-1938* Min.	*182*	...	...	...	...
1937 *(b)* ...	**151**	120	107	221	150	1937	**208**	...	...	...	...
1938 *(b)* ...	137	98	120	192	133	1938	182	...	...	...	...
1939 *(b)* ...	120	108	97	177	120	1939	133	...	...	...	...
1940	126	87	106	188	129	1940	176	...	...	...	...
1941	**151**	104	98	218	189	1941	154	...	...	...	...
1942 *(b)* ...	131	101	101	186	139	1942	...	...	...	...	...
1943	...	115	115	200	...	1943	...	...	...	...	...

General Mortality (rates per 1,000 inhabitants on a yearly basis).

Portugal

	Yearly rates	Quarterly I	II	III	IV
1930-1938 Max.	*17.2*	*18.7*	*15.3*	*18.5*	*18.5*
1930-1938 Med.	*16.8*	*17.5*	*14.2*	*17.2*	*17.4*
1930-1938 Min.	*15.4*	*16.2*	*13.3*	*15.7*	*15.7*
1937	15.8	16.2	13.3	17.2	16.5
1938	15.4	16.3	13.8	15.7	15.7
1939	15.3	15.8	13.3	15.7	16.4
1940	15.6	15.4	13.3	16.3	**17.6**
1941	**17.4**	17.4	**14.7**	17.2	**20.2**
1942	16.1	16.9	14.1	16.3	17.1
1943	...	15.4	13.4	17.2	...

Lisbon

	Yearly rates	I J	II F	III M	IV A	V M	VI J	VII —	VIII J	IX A	X S	XI O	XII N	XIII D
1930-1938 Max.	*20.5*	*24.2*	*27.9*	*23.3*	*24.2*	*20.7*	*19.4*		*24.3*	*21.7*	*20.9*	*21.8*	*21.7*	*22.2*
1930-1938 Med.	*19.3*	*21.9*	*23.3*	*21.0*	*18.4*	*18.0*	*17.9*		*19.1*	*19.3*	*18.8*	*19.9*	*19.3*	*20.2*
1930-1938 Min.	*18.0*	*18.6*	*19.4*	*16.4*	*15.6*	*15.5*	*15.5*		*17.1*	*17.4*	*16.0*	*16.1*	*16.9*	*17.2*
1937	18.3	19.5	21.8	16.4	15.6	15.5	15.5		19.5	19.3	18.9	18.4	19.8	19.3
1938	18.5	20.8	23.3	21.0	18.7	18.0	17.3		17.1	18.0	16.4	16.1	16.9	18.9
1939	16.2	17.5	17.4	17.2	15.8	15.1	15.8		15.9	15.0	14.8	15.3	16.6	17.7
1940	17.3	17.5	20.5	18.2	17.0	16.8	**18.2**		14.9	15.9	17.1	16.8	16.3	18.2
1941	17.2	19.7	20.3	16.6	17.1	18.3	14.9		15.8	16.7	16.8	16.9	16.9	18.1
1942	15.9	17.8	19.5	19.1	15.4	14.9	13.9		15.9	12.9	13.3	15.2	15.1	18.5
1943	...	17.6	15.0	15.6	16.1	15.0	16.0		14.2	14.0	13.5	14.7	16.0	...

also affected by the overseas blockade and by the attraction of food exports to Germany as a result of high prices. There was, in fact a rise in *general mortality* in 1941, and the rate for 1942 was still a little high, but the 1943 figures have returned to normal (*cf.* graph, page 600). During the war, general mortality in Lisbon has remained quite normal, and the 1942 rates even constituted a low record.

Infant mortality was high in 1941 for Portugal; normal for Lisbon.

Tuberculosis death rates for the whole of Portugal were comparatively high in 1941 and 1942. They were normal in Lisbon.

Apart from meningitis, the prevalence of *epidemic diseases*, which was fairly high in 1939, has since been decreasing.

Having regard to the fairly high range of normal variations of mortality in a warm country like Portugal, there is no indication from the available official statistics which would suggest that the war has adversely influenced either the nutritional or the sanitary situation of the country.

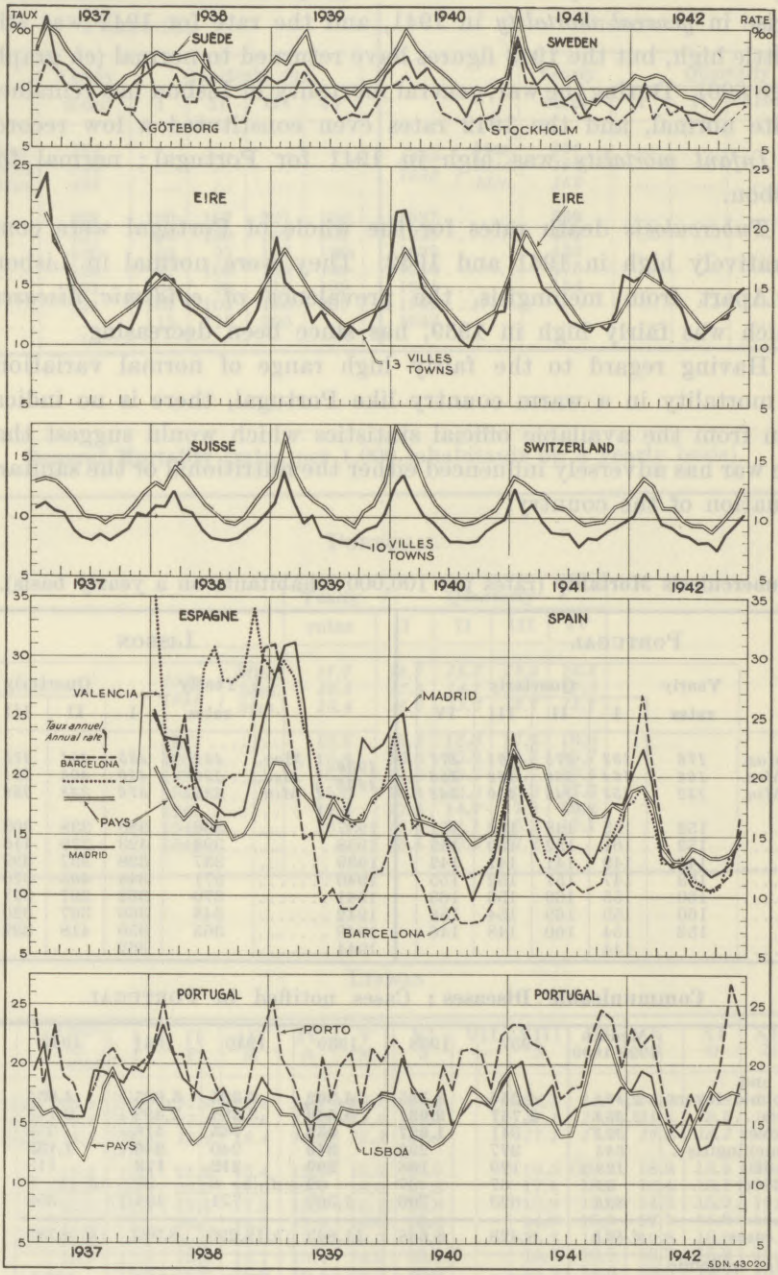
Tuberculosis Mortality (rates per 100,000 inhabitants on a yearly basis).

	Portugal					Lisbon				
	Yearly rates	Quarterly I	II	III	IV	Yearly rates	Quarterly I	II	III	IV
1930-1938 Max.	*176*	*192*	*178*	*191*	*177*	*451*	*485*	*451*	*475*	*440*
1930-1938 Med.	*168*	*164*	*166*	*162*	*156*	*396*	*410*	*405*	*391*	*385*
1930-1938 Min.	*152*	*152*	*148*	*150*	*142*	*380*	*376*	*338*	*360*	*374*
1937	152	155	148	152	151	366	383	338	363	380
1938	153	161	152	150	149	398	423	379	416	374
1939	148	149	141	141	142	337	338	337	339	335
1940	153	147	157	152	155	371	344	405	370	366
1941	160	155	168	156	163	370	362	397	371	350
1942	160	159	169	154	158	348	366	367	320	339
1943	152	154	160	148	146	365	350	418	332	362
1944		144					362			

Communicable Diseases: Cases notified in Portugal.

	Median 1928-1938	1937	1938	1939	1940	1941	1942	1943
Typhoid and paratyphoid fevers	*2,865*	**4,244**	3,385	**4,302**	**6,395**	**5,035**(*e*)	**4,654**	
Diphtheria	*2,358*	**2,747**	**3,031**	**4,548**	**3,894**	2,492(*e*)	2,145	
Scarlet fever	*331*	341	**1,027**	**953**	**745**	418(*e*)	358	
Cer.-sp. meningitis	*285*	277	296	**340**	240	**346**(*e*)	**1,132**	
Poliomyelitis	*129*(*i*)	129	166	**299**	**212**	**178**	113	
Typhus fever	*37*	67	37	32	(*d*) (*h*) 6	50	(*d*) (*h*) 5	
Smallpox	*836*	623	706	1,369	771	464(*f*)	352	
Notified cases	*6,841*	8,428	8,648	11,843	(*h*)12,299	8,983	(*h*) 8,789	
Composite epidemic index	100	123.2	126.4	173.1	179.8	131.3	128.5	

General Mortality, per 1,000 Inhabitants, in Some Neutral Countries: Eire, Portugal, Spain, Sweden and Switzerland, 1937-1942.



N.B. — During the years of civil war, rates for Spain are approximate.

France

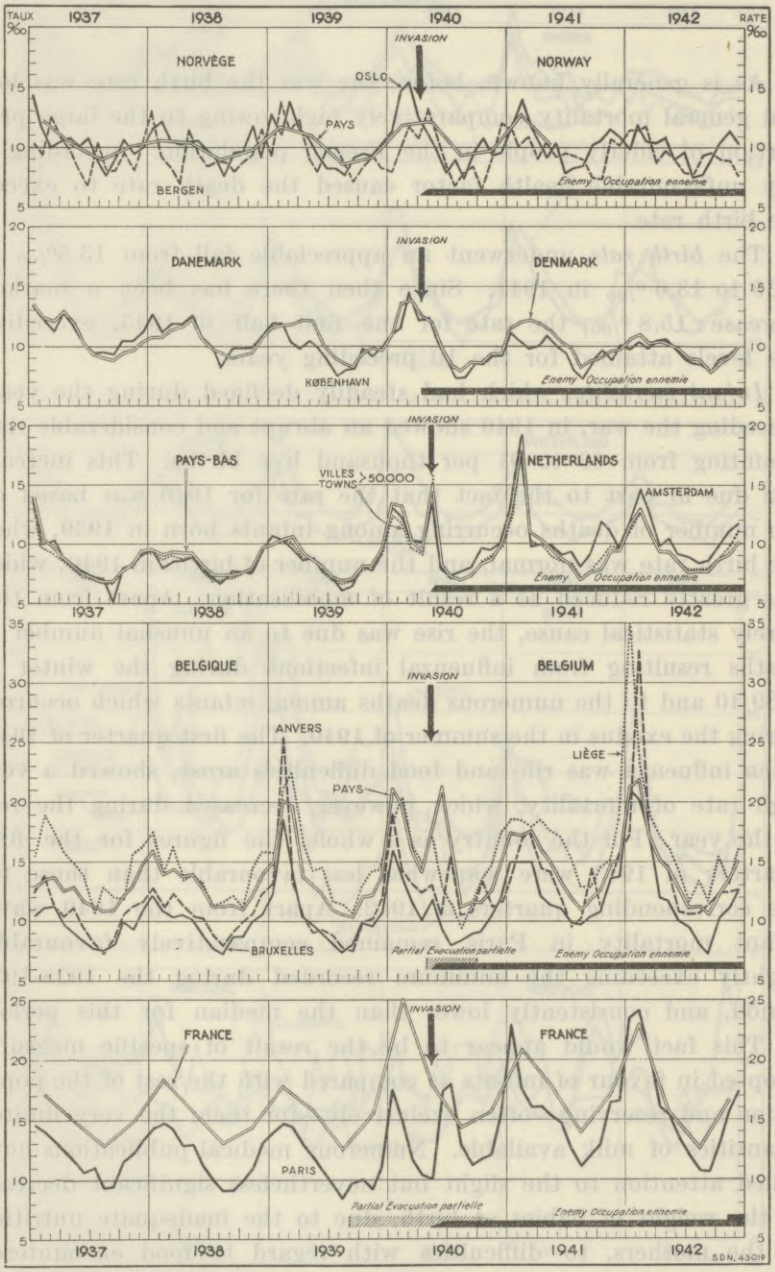
As is generally known, before the war the birth rate was low and general mortality comparatively high, owing to the large proportion of elderly people in the French population. Accordingly, any unfavourable health factor caused the death rate to exceed the birth rate.

The *birth rate* underwent an appreciable fall from 13.6‰ in 1939 to 13.0 ‰ in 1941. Since then there has been a marked increase : 15.8 ‰, the rate for the first half of 1943, exceeding the levels attained for the 10 preceding years.

Infant mortality, which had steadily declined during the years preceding the war, in 1940 showed an abrupt and considerable rise, mounting from 63 to 91 per thousand live births. This increase was due in part to the fact that the rate for 1940 was based on the number of deaths occurring among infants born in 1939, when the birth rate was normal, and the number of births in 1940, which was greatly reduced as a result of mobilisation. Apart from this purely statistical cause, the rise was due to an unusual number of deaths resulting from influenzal infections during the winter of 1939/40 and to the numerous deaths among infants which occurred during the exodus in the summer of 1940. The first quarter of 1941, when influenza was rife and food difficulties arose, showed a very high rate of mortality, which, however, decreased during the rest of the year. For the country as a whole, the figures for the first quarters of 1943 were somewhat less favourable than those for the corresponding quarters of 1942. Apart from the 1940 wave, infant mortality in Paris remained comparatively favourable, slightly exceeding the minimum recorded during the 1928-1938 period, and consistently lower than the median for this period.

This fact would appear to be the result of specific measures adopted in favour of infants as compared with the rest of the population and reserving—often exclusively—for them the very limited quantities of milk available. Numerous medical publications have called attention to the slight but nevertheless significant decrease in the weight of babies at birth, due to the inadequate nutrition of the mothers, to difficulties with regard to food encountered in the towns owing to irregular deliveries of food supplies and

General Mortality, per 1,000 Inhabitants, in Some Occupied Countries: Belgium, Denmark, France, the Netherlands and Norway, 1937-1942.



N.B. — Rates for war years are approximate.

to the indifferent quality of the products available, to difficulties in breast-feeding due to the malnutrition of the mothers, and difficulties in artificial feeding due to the scarcity of milk, the poor quality of the flour used in baby-foods and to the inadequacy of the supply of vitamins.

Success in the maintenance of a low infant mortality rate does not necessarily imply a satisfactory state of health among infants, many of whom show a retarded development in weight, suffer from rickets and display other signs of vitamin deficiency. Retarded development as regards height and weight is also the rule among children of school-age and pre-school-age, in large towns and in families with low incomes (particularly the working-classes). The results of inadequate feeding are especially noticeable among adolescents, notwithstanding the larger rations to which they are entitled.

General mortality was very high during the first quarter of 1940 (cf. graph, page 602). It remained high during the summer months, as the result of the bad conditions obtaining during the exodus, bombing and machine-gunning of civilians, difficulties in caring for wounded and sick persons, local food shortages, at first temporary but subsequently—in the south, which was encumbered with refugees—permanent. It still remained high in 1941. It appears that another winter influenza epidemic contributed to this during the first quarter, but throughout the whole of the year it appears that food shortage played a major part. This shortage was very largely the result of the ruthless requisitioning of food stores and mass purchases by the German authorities. Ill-advised State intervention, however, local economic autarchy, the division of the country into two watertight compartments by the so-called line of demarcation, and the reduction of means of transport all played their part in creating this situation. It appears that, during the ensuing years, there was a progressive adaptation of the system of production and apportionment of foodstuffs, and, despite the decrease in official rations, an increase in actual consumption, private enterprise making up for the impotence of the public authorities. Although still above normal, the mortality rate for 1942 was lower than that for 1941, and the rate for 1943 was again slightly lower. It must be borne in mind that these rates, which cover the whole of the country and are influenced by the comparatively favourable situation of the rural population (not including that of the exclusively wine-producing regions), imperfectly reflect

the rise of mortality which occurred in the large towns where the food shortage was most acute—Paris, still more Lyons, and above all Marseilles, Toulon and other towns of the Mediterranean coast.

This excess mortality was particularly marked among old people, whose food rations were especially low, and among whom loss of weight frequently reached a figure of 30 kg. (66 lbs), as against 5 to 15 kg. (11 to 33 lbs) in younger adults. Famine disease (cachexia and famine œdema) appeared, moreover, in homes for the aged almost at the same time as in prisons and internment camps situated in the south—that is to say, as early as the winter of 1940/41.[1]

The excess mortality recorded in France is due in part to the rise in *tuberculosis mortality* which, at the end of 1943, with a rate of 152 per 100,000 inhabitants,[2] represented more than 7.4% of the total mortality. Before the war, mortality from tuberculosis was consistently decreasing and it is necessary to go back to 1931 to find a rate as high as this.

The increase was particularly high in the " départements " of the Mediterranean region. In Bouches-du-Rhône (Marseilles) the tuberculosis mortality rate reached 221 in 1943, as against 129 in 1938 (+71%), in the Var (Toulon) 209 as against 131 (+59%). In Paris the increase was about 40% between 1938 and 1941. Since then a certain decrease has occurred there, but it is difficult to see how far this improvement is real or only apparent, as it may be the result of an exodus of sick persons to the country, where food was more easily obtainable.

The excess tuberculosis mortality was encountered in all age-groups but more particularly among adults (often older adults), who showed the rapidly evolutive forms of the disease, which as a rule occur only in infants : miliary tuberculosis, meningitis, caseous pneumonia, etc. These forms were particularly common in 1941.

It should be added that a large number of prisoners of war repatriated from Germany as seriously disabled contributed to the rise in tuberculosis mortality in France.

Venereal disease did not increase during the first part of the war (1939/40), as it did in 1914-1918. In 1942, nevertheless, a rise was recorded, the number of cases of primary or secondary syphilis

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[1] In a further article in this *Bulletin*, details on the incidence and gravity of deficiency diseases and malnutrition will be found.

[2] Rate corrected by taking into account deaths from unknown causes, the uncorrected rate being 141 per 100,000.

cases treated in public dispensaries increasing by a third.[1]

Other contagious diseases did not contribute to an appreciable extent to the rise in the mortality rate recorded. The number of deaths corresponding to the number of cases notified is much too low to have been able appreciably to influence the general mortality rate. An increase in typhoid and paratyphoid fevers has been recorded since 1941. The incidence of meningitis which rose in 1940 as a result of mobilisation rapidly returned to normal. No increase in the incidence of scarlet fever has occurred. As to diphtheria, its incidence has been on the increase from 1940 to 1943. Diphtheria morbidity for 1943 was the highest recorded in France for many years, while remaining lower than that for most countries of Continental Europe. In many regions of France, lack of serum aggravated the prognosis of the disease.

To sum up, apart from losses due directly to the war (military operations, bombardments, executions, in the neighbourhood of 300,000 to 400,000 deaths), there has been a notable increase in mortality among the French population during the war—which would appear to be due in particular to the results of malnutrition.

This was observable above all in the towns—the larger the town the more noticeable it became—and more particularly in groups of the population which, through confinement, physical weakness or lack of money, were unable to supplement the rations officially accorded.

Various symptoms of malnutrition, including very marked loss of weight, are much more general than the limited increase in mortality would lead one to think.

Notwithstanding the prevalence of malnutrition, there was no unusual incidence of epidemic disease as a whole (except diphtheria), nor were the cases abnormally serious.

The rise in tuberculosis mortality was marked.

As a whole, it is the food situation rather than the epidemic situation which is disquieting ; it may become disastrous in the towns if a continued interruption of the means of communication hinders the arrival of foodstuffs, particularly of milk for infants, thus aggravating the food scarcity and, at the same time, prevents the population from seeking relief in the country.

[1] In a sample of 22 dispensaries distributed in as many towns, the number of these cases and the number of consultations, during the years 1938 to 1942, was 665 (183,000), 617 (184,000), 706 (169,000), 676 (160,000), 1,080 (171,000). It is interesting to compare these figures with those for 1921 : 2,447 (110,000). Cavaillon (1943) : *Prophylax. antivén.*, **15** : 213.

France

Declared war : September 3rd, 1939. Invaded : May 17th, 1940 ; armistice : June 22nd, 1940. Partially occupied (north-west zone) from June 1940 ; completely occupied, November 1942. Liberation begun June 6th, 1944.

Civilian **Population** used for calculating the rates (except for 1936 : average yearly population).

France			Paris		
C.	III.1936 :	41,906,000	C.	III.1936 :	2,830,000
O. N. E.	1939 :	41,980,000	O. N. E. and E. S.	1939 *(f)* :	2,487,000
O. N. E. and E. S.	1940 *(a) (p)* :	39,340,000	O. N. E. and E. S.	1940 *(f)* :	1,992,000
O. N. E.	1941 *(a) (b)* :	38,000,000	O. N. E. and E. S.	1941 :	2,304,500
E. S.	1942 *(a) (b)* :	37,800,000	O. N. E.	1942 :	2,246,000
E. S.	1943 *(e) (b)* :	37,000,000	O. N. E. and E. S.	1943 :	2,229,000

Natural Movement of Population.

Year	France Births ‰ inhab.	France General mortality ‰ inhab.	France Increase (+) or decrease (−) ‰ inhab.	Paris Births ‰ inhab.	Paris General mortality ‰ inhab.	Paris Increase (+) or decrease (−) ‰ inhab.
1928-1938 *Max.*	*18.3*	*17.9*	*+2.4*	*15.0*	*15.0*	*+1.4*
1928-1938 *Med.*	*16.2*	*15.7*	*+0.4*	*12.7*	*13.0*	*−0.3*
1928-1938 *Min.*	*14.6*	*15.0*	*−0.8*	*10.9*	*11.9*	*−1.0*
1937	14.7	15.0	−0.3	11.1	11.9	−0.8
1938	14.6	15.4	−0.8	10.9	11.9	−1.0
1939	14.6	15.3	−0.7	10.0	12.1	−2.1
1940 *(a) (b) (n)*	13.6	**18.7**	−5.1	11.4 *(k)*	**15.9** *(k)*	−4.5 *(k)*
1941 *(a) (b)* ..	13.0	**17.4**	−4.4	11.0	**15.5**	−4.4
1942 *(a) (b) (n)*	14.4	**16.9**	−2.5	12.2	**16.1**	−3.9
1942 *(a) (b) (n)* (1st half)	14.5	19.2 *(h)*	−4.7			
1943 *(e) (b) (n)* (1st half)	15.8	17.8 *(h)*	−2.0	I-X.1943 13.3	**14.6**	−1.3

C. = Census.

E. S. = Estimate of the Epidemiological Intelligence Service of the L.o.N.

O. N. E. = Official national estimate *(Bulletin de la statistique générale de la France)*.

(a) 87 " départements ", excluding : Bas-Rhin, Haut-Rhin, and Moselle.

(b) Prisoners of war and, from 1942, civilian workers in Germany deducted.

(e) 86 " départements ", excluding : Bas-Rhin, Haut-Rhin, Moselle and Corsica.

(f) Monthly approximate adjustment.

(h) Owing to lack of comparability with rctrospective data, does not indicate whether the upper quartile has been exceeded.

(i) 85 " départements ", excluding : Bas-Rhin, Haut-Rhin, Moselle, Corsica and Indre.

(j) 84 " départements ", excluding : Bas-Rhin, Haut-Rhin, Moselle, Corsica, Loiret, Savoy.

(k) Approximate rates, according to a monthly population adjustment.

(l) Monthly rates taken from the period 1929-1937.

(m) The higher rates for June and July do not correspond to any increase in the number of deaths registered, but to the population calculated to be in Paris during thcse months, sick persons in hospitals not being evacuated like other inhabitants.

(n) Rates calculated by the Epidemiological Intelligence Service of the L.o.N.

(o) Provisional rates based on figures furnished by the *Institut National d'Hygiène*, Paris.

(p) Prisoners and war losses deducted for the last two quarters and for the calculation of the average yearly population.

Infant Mortality (deaths of infants under 1 year of age, per 1,000 live births).

FRANCE

	Yearly rates	I J	II F	III M	IV A	V M	VI J	VII —	VIII J	IX A	X S	XI O	XII N	XIII D
1928-1938 Max.	*96*		*112*			*85*				*101*			*87*	
1928-1938 Med.	*75*		*83*			*78*				*63*			*65*	
1928-1938 Min.	*65*		*73*			*65*				*55*			*57*	
1937	65		80			65				57			57	
1938	66		66			69				61			60	
1939	63		77			66				50			58	
1940 *(a)*....	**91**		**95**			**91**				**93**			**71**	
1941 *(a)*....	73		**105**			73				59			65	
1942 *(a)*....	70		82			65				64			69	
1943 *(e)*....			84			75								

PARIS

1928-1938 Max.	*101*	*110*	*120*	*119*	*112*	*110*	*105*		*91*	*113*	*119*	*100*	*88*	*116*
1928-1938 Med.	*80*	*92*	*94*	*102*	*96*	*81*	*72*		*60*	*66*	*60*	*64*	*64*	*87*
1928-1938 Min.	*59*	*67*	*59*	*70*	*59*	*60*	*54*		*46*	*39*	*45*	*45*	*56*	*62*
1937	59	92	71	70	59	60	54		50	39	45	53	59	62
1938	66	78	59	78	79	81	**105**		48	66	46	45	58	87
1939 *(n)* ...	61	93	74	68	68	50	53		42	41	58	**75**	51	67
1940 *(k)* *(n)*	76	79	79	81	82	73	**133**		**91**	**84**	56	60	68	72
1941 *(n)* ...	65	62	104	**136**	84	57	60		56	47	53	47	55	67
1942 *(n)* ...	60	69	83	80	62	56	53		41	48	42	47	60	86
1943 *(n)* ...	...	78	79	67	70	65	56		55	46	46	44	...	...

General Mortality (rates per 1,000 inhabitants on a yearly basis).

FRANCE

	Yearly rates	I J	II F	III M	IV A	V M	VI J	VII —	VIII J	IX A	X S	XI O	XII N	XIII D
1928-1938 Max.	*17.9*		*25.4*			*17.1*				*14.4*			*15.8*	
1928-1938 Med.	*15.7*		*19.1*			*15.7*				*13.0*			*14.9*	
1928-1938 Min.	*15.0*		*17.2*			*14.4*				*12.7*			*14.1*	
1937	15.0		17.2			15.0				13.0			15.0	
1938	15.4		18.4			15.7				13.1			14.4	
1939	15.3		17.9			15.8				12.5			**15.1**	
1940 *(a)*(*b*)(*n*)	**18.7**		**25.1**			**17.8**				**15.1** *(p)*			**16.5** *(p)*	
1941 *(a)*(*b*)..	**17.4**		**20.8**			**17.8**				**14.0**			**17.0**	
1942 *(a)*(*b*)(*n*)	**16.9**		**22.8**			15.6				12.7			**16.6**	
1943 *(e)*(*b*)(*n*)	16.2 *(o)*		19.3			**16.2**				**13.2** *(o)*			**16.0** *(o)* *(j)*	

PARIS

1928-1938 Max.	*15.0*	*19.1*	*28.7*	*19.1*	*16.1*	*14.4*	*12.7*		*12.0*	*10.6*	*11.2*	*12.5*	*13.7*	*16.4*
1928-1938 Med.	*13.0*	*14.4*	*15.8*	*15.0*	*13.6*	*13.0*	*11.6*		*10.7*	*9.3*	*9.4*	*11.6*	*12.3*	*13.3*
1928-1938 Min.	*11.9*	*13.5*	*14.1*	*13.2*	*12.5*	*11.6*	*10.6*		*9.2*	*8.6*	*8.6*	*9.9*	*11.0*	*12.6*
1937	11.9	14.6	14.1	13.3	12.5	11.6	10.6		11.0	9.3	9.4	11.6	12.3	13.3
1938	11.9	14.3	14.7	14.3	12.9	13.0	11.2		10.0	9.1	9.1	9.9	11.0	13.0
1939 *(n)* ...	12.1	14.0	14.6	14.4	12.9	11.8	10.8		10.0	9.0	**11.8**	11.7	11.2	12.9
1940(*k*)(*m*)(*n*)	**15.9**	**20.7**	**19.4**	13.8	12.6	11.9	**21.1**		**21.2**	**15.2**	**13.7**	**13.8**	**14.6**	**18.3**
1941 *(n)* ...	**15.5**	**23.0**	**17.8**	16.2	**16.2**	**15.5**	**14.8**		**12.6**	**11.2**	**12.1**	**13.1**	**16.6**	**16.3**
1942 *(n)* ...	**16.1**	**23.7**	**24.3**	**20.2**	**16.2**	**15.5**	**13.6**		**12.1**	**10.8**	**10.7**	**13.2**	**15.9**	**17.5**
1943 *(n)* ...		17.1	**17.5**	**19.5**	**16.3**	**14.2**	**12.5**		**12.0**	10.2	**13.2**	**13.6**		

Tuberculosis Mortality (rates per 100,000 inhabitants on a yearly basis).

	France					Paris				
	Yearly rates	Quarterly I	II	III	IV	Yearly rates	Quarterly 1	II	III	IV
1928-1936 Max.	*166*	...	...	...	...	*235*	*287*	*261*	*206*	*210*
1928-1936 Med.	*139*	...	...	...	...	*187*	*223*	*201*	*155*	*169*
1928-1936 Min.	*118*	...	...	...	...	*155*	*183*	*173*	*131*	*131*
1937	...	...	...	...	...	164	183	187	143	145
1938	...	...	...	...	...	155	186	173	131	131
1939	...	...	...	...	...	164	169	180	152	150
1940	...	...	...	...	...	194	210	187	**195**	184
1941	...	...	...	...	...	215	219	244	**198**	**200**
1942	...	...	...	...	...	198	241	229	164	159
1943 *(o)*....	141*(j)*	152*(a)*	166*(i)*	126*(e)*	119*(j)*	185	228	213	142	157
1943 *n)*										

Communicable Diseases : Cases notified in France.

	Median 1928-1938	1937	1938	1939	1940	1941	1942	1943
Typhoid and paratyphoid fevers ...	*5,868*	5,868	4,120	4,373	4,304	**7,934**	**10,612**	**13,761**
Dysentery	*73*	**129**	73	67	**651**	**250**	80	23
Diphtheria	*19,893*	19,187	16,800	14,019	13,568	20,018	**31,466**	**46,539**
Scarlet fever	*18,431*	18,658	18,431	14,640	10,951	11,201	11,980	17,085
Cer.-sp. meningitis .	*461*	462	408	328	**2,321**	**1,143**	**585**	406
Poliomyelitis	*487*	**592**	**788**	460	342	484	322	**1,783**
Typhus fever	*0*	**1**	**1**	—	**3**	**3**	**230**	**4**
Smallpox	*6*	5	2	5	5	8	**63**	5
Notified cases	*45,219*	44,902	40,623	33,892	32,145	41,041	55,338	79,606
Composite epidemic index	100	99.3	89.8	75.0	71.1	90.8	122.4	176.0

Belgium

Many of the observations regarding the food and sanitary situation in France are also applicable to Belgium. At the beginning of 1940 there was the same rise in *general mortality* as a result of winter respiratory infections (*cf.* graph, page 602). In May, during the invasion, mortality rose steeply, as much among the civilian population as in the army, as a direct result of military operations. It was impossible to register in Belgium a proportion of the deaths of Belgian subjects which, during the mass evacuations, occurred in France between May and August.

The peak of mortality recorded during that critical year was not exceeded in 1941 and 1942, although the rates for these years

were unusually high, owing to the effects of insufficient food. This excess of mortality in 1941, as compared with 1938, was about 12% for the whole of Belgium. It was higher in 1942—28%, and rose to 36% in 1943. In Belgium, as in France, food shortage was much more acutely felt in the towns, and particularly in the cities, than in the country.

Increase in *tuberculosis mortality* was also markedly higher in Brussels than in the country as a whole—60% instead of 40% from 1939 to 1941.

The *infant mortality* rate did not follow the upward trend of the general mortality rate. In both Belgium and the city of Brussels, after a rise in 1940 and 1941, the pre-war downward trend again asserted itself. The 1942 rate for Brussels and the 1943 rate for Belgium as a whole are, indeed, the lowest recorded. This again appears to be the result of a deliberate policy of protecting infants at all costs. This result has been achieved in spite of the difficulties experienced in providing suitable infant-foods. Food difficulties often necessitated the premature weaning of their babies by nursing-mothers.

Among the population generally, signs of malnutrition were widespread ; in adults loss of weight varying from 20 to 30 pounds was very common. From chilblains and traces of rickets in breast-fed children to pellagra, " famine osteopathy " and famine œdema, all forms of malnutrition were observed ; famine œdema was of frequent occurrence among the inmates of prisons as early as the winter of 1940/41 : it afterwards spread to the poorer sections of the urban population.

Malnutrition does not appear to have influenced the severity of the common communicable diseases. Their incidence, however, has shown a significant rise in Belgium, as in Germany : enteric fevers in 1942 and 1943, diphtheria and scarlet fever from 1941 onwards. In 1943 the incidence of diphtheria was nearly eight times the median of pre-war years, and that of scarlet fever five times. These two diseases account chiefly for the rise in the epidemic index in recent years. The country remained practically free from typhus fever.

To sum up, malnutrition is common in this country, which, owing to the extent to which it has become urbanised, scarcely produced

Belgium

Invaded by the German Army : May 10th, 1940. Evacuation of a large number of the population to France. Capitulation : May 28th, 1940. Return of the greater number of evacuated persons in July and August. At the beginning of 1943, there were 77,000 war prisoners and 300,000 deported Belgian civilian workers in Germany.

Civilian **Population** used for calculating the rates.

Belgium		(Greater) Brussels	
C. XII.1930 :	8,092,000	C. XII.1930 :	851,000
O. N. E. XII.1938 :	8,386.600	O. N. E. I.1939 :	894,400
E. S. VI.1940 :	8,345.000 *(e)*	O. N. E. I.1940 :	890,900
O. N. E. XII.1940 :	8,294.700 *(e)*	O. N. E. I.1941 :	888,400
O. N. E. XII.1941 :	8,257.400	O. N. E. I.1942 :	879,700
O. N. E. XII.1942 :	8,238,400	O. N. E. I 1943 :	925,600
Brussels (town) *(a)*			
C XII.1930 :	200,400	O. N. E. I.1941 :	188,400
O. N. E. I.1939 :	191,700	O. N. E. I.1942 :	185,500
O. N. E. I.1940 :	189,000	1943 *(a)*	

Natural Movement of Population.

Year	Belgium *(g)* Births °/₀₀ inhab.	General mortality °/₀₀ inhab.	Increase (+) or decrease (−) °/₀₀ inhab.	(Greater) Brussels Births °/₀₀ inhab.	General mortality °/₀₀ inhab.	Increase (+) or decrease (−) °/₀₀ inhab.
1928-1938 Max.	*18.8*	*15.1*	*+5.5*	*12.2*	*11.3*	*+2.4*
1928-1938 Med.	*16.5*	*13.1*	*+3.4*	*11.0*	*9.7*	*+0.8*
1928-1938 Min.	*15.2*	*12.2*	*+2.2*	*9.9*	*9.1*	*+0.4*
1937	15.3	13.1	+2.2	9.9	9.5	+0.4
1938	15.8	13.1	+2.7	10.5	9.7	+0.8
1939	15.3	**13.8**	+1.5	10.5	10.6	−0.1
1940	13.4	**16.1**	−2.7	9.6	10.8	−1.2
1941	12.1	**14.7**	−2.6	8.9	12.2	−3.3
1942	13.1	**14.7**	−1.6	9.9	12.5	−2.6
1943 *(h)* ...	14.7	**13.4**	+1.3	10.8	13.2	−2.4

C. = Census.

E. S. = Estimate of the Epidemiological Intelligence Service of the L. o. N.

O. N. E. = Official national estimate.

(a) From 1928 to 1942, infant mortality rates refer to Brussels (town). From 1943, these rates are given only for Greater Brussels.

(b) Including war losses and deaths of civilians due to the war.

(e) From 1940 included, excluding the 41 communes of Eupen, Malmédy, and Moresnet districts.

(f) For want of retrospective data, monthly rates do not indicate whether the upper quartile has been exceeded.

(g) Yearly rates corrected up to 1942 inclusive.

(h) General mortality rates for 1943 are calculated on a population of 8,238,400 (on December 31st, 1942), which does not take into account deportation of civilian workers. According to E. M. Kulischer, *The Displacement of Population in Europe*, I.L.O., Montreal, 1943, page 160, the number of deported civilian Belgian workers in Germany rose to about 300,000 at the beginning of 1943. The annual general mortality rate corresponding to the number of population from which this figure has been deducted would be 13.9 °/₀₀.

(i) Median for 1932-1938 only.

(j) Median for 1930-1938 only.

(k) Incomplete : only 36 weeks.

Infant Mortality (deaths of infants under 1 year of age, per 1,000 live births). *(b)*

BELGIUM *(f)*

	Yearly rates	I J	II F	III M	IV A	V M	VI J	VII —	VIII J	IX A	X S	XI O	XII N	XIII D
1928-1938 Max.	*104*	...	...	...	...	...	...		...	...	...	...	...	...
1928-1938 Med.	*85*	...	...	...	...	...	...		...	...	...	...	...	...
1928-1938 Min.	*75*	...	...	...	...	...	...		...	...	...	...	...	...
1937	80	122	120	95	87	78	66		59	63	60	63	74	74
1938	75	82	66	82	89	98	88		62	60	66	60	71	80
1939	76	87	100	99	98	70	59		50	56	57	65	79	90
1940	**89**	138	132	105	81	89	91		64	70	66	60	66	72
1941	85	105	164	129	111	83	70		54	47	63	66	80	80
1942	77	104	97	81	76	83	69		54	58	64	75	79	99
1943	68	110	86	77	73	60	50		52	59	58	58	57	77

BRUSSELS (town) *(a)*

	Yearly rates	I J	II F	III M	IV A	V M	VI J	VII —	VIII J	IX A	X S	XI O	XII N	XIII D
1928-1938 Max.	*104*	*168*	*157*	*189*	*171*	*213*	*108*		*101*	*86*	*109*	*96*	*167*	*146*
1928-1938 Med.	*78*	*92*	*89*	*103*	*88*	*68*	*64*		*63*	*49*	*58*	*70*	*62*	*82*
1928-1938 Min.	*66*	*73*	*60*	*60*	*63*	*40*	*26*		*29*	*39*	*27*	*53*	*31*	*55*
1937	**92**	**168**	73	81	89	**117**	**79**		**101**	**74**	51	83	58	**119**
1938	**102**	79	60	113	**128**	**213**	69		**95**	42	**109**	**96**	**167**	82
1939	66	**123**	75	92	67	46	42		38	28	49	**106**	58	75
1940	44	90	56	46	41	38	26		13	49	35	39	42	48
1941	54	70	18	67	50	19	40		59	24	63	34	43	29
1942	43	65	40	**122**	38	23	20		35	70	13	47	48	62
1943 *(a)*	52	69	49	43	55	61	45		39	56	49	53	50	58

General Mortality (rates per 1,000 inhabitants on a yearly basis). *(b)*

BELGIUM *(f)*

	Yearly rates	I J	II F	III M	IV A	V M	VI J	VII —	VIII J	IX A	X S	XI O	XII N	XIII D
1928-1938 Max.	*15.1*	...	...	...	...	...	...		...	...	...	...	...	...
1928-1938 Med.	*13.1*	...	...	...	...	...	...		...	...	...	...	...	...
1928-1938 Min.	*12.2*	...	...	...	...	...	...		...	...	...	...	...	...
1937	13.1	14.7	15.4	15.2	13.9	12.9	11.7		11.3	10.9	11.0	12.2	13.2	14.8
1938	13.1	15.9	14.0	14.4	13.7	13.9	12.8		11.3	11.3	11.4	11.1	12.2	14.0
1939	**13.8**	14.9	20.5	19.7	14.7	13.0	12.4		11.0	10.6	10.4	12.1	12.2	13.7
1940	**16.1**	20.9	19.6	15.9	14.1	33.4	17.2		12.6	11.2	11.5	11.6	12.5	14.9
1941	**14.7**	17.4	17.2	17.5	15.8	14.9	13.2		11.9	11.0	11.8	12.6	14.9	15.0
1942	**14.7**	21.3	21.8	17.2	15.1	14.3	12.7		11.1	11.2	10.6	12.2	13.4	15.1
1943 *(h)*	**13.4**	16.3	14.9	15.8	15.3	12.8	10.8		10.7	10.4	11.9	12.2	13.2	16.8

(Greater) BRUSSELS

	Yearly rates	I J	II F	III M	IV A	V M	VI J	VII —	VIII J	IX A	X S	XI O	XII N	XIII D
1928-1938 Max.	*11.3*	*13.8*	*22.7*	*16.6*	*11.5*	*11.3*	*9.2*		*8.3*	*8.2*	*8.9*	*9.6*	*11.1*	*12.5*
1928-1938 Med.	*9.7*	*11.2*	*11.5*	*11.3*	*10.5*	*9.8*	*8.4*		*7.8*	*7.6*	*8.1*	*8.9*	*9.4*	*10.5*
1928-1938 Min.	*9.1*	*10.2*	*10.3*	*10.3*	*9.6*	*9.1*	*8.0*		*6.9*	*6.6*	*6.9*	*7.3*	*8.3*	*9.7*
1937	9.5	10.5	11.2	11.3	9.8	9.1	8.4		7.8	7.6	8.1	**10.0**	9.4	11.0
1938	9.7	**12.2**	10.9	10.4	10.5	**10.2**	**8.8**		7.8	7.3	**8.8**	8.8	**10.0**	10.6
1939	**10.6**	**12.3**	**18.6**	**13.7**	10.3	9.7	**9.0**		8.1	7.4	8.1	**9.9**	9.6	10.7
1940	**10.8**	**15.8**	13.9	11.5	10.1	**11.8**	**9.3**		8.0	**8.4**	**8.9**	9.1	**10.4**	**11.9**
1941	**12.2**	**15.0**	15.0	**15.0**	**12.1**	**11.4**	**11.0**		**9.8**	**9.7**	**10.3**	**10.7**	**13.5**	**13.7**
1942	**12.5**	**20.8**	**20.2**	**17.5**	**11.9**	**11.5**	**10.0**		**9.5**	**8.0**	7.2	**10.2**	**11.0**	**12.7**
1943	**13.2**	**16.3**	14.7	**15.3**	**13.6**	**12.3**	**10.6**		**10.8**	**10.0**	**14.3**	**11.9**	**12.8**	**16.3**

one-third of its normal food requirements, and with increased effort could scarcely exceed one-half.

The severity of manifestations of malnutrition is greater in the towns than in the country, but, so far, infants have remained comparatively unaffected. The definite rise in diphtheria and scarlet fever morbidity was not accompanied by any appreciable increase in fatality.

It is worthy of note that, after a sharp fall, the birth rate showed a very significant rise in 1942 and 1943, notwithstanding the number of men who were prisoners or deported to Germany.

Tuberculosis Mortality (rates per 100,000 inhabitants on a yearly basis).

	Belgium					(Greater) Brussels				
	Yearly rates	Quarterly I	II	III	IV	Yearly rates	Quarterly I	II	III	IV
1928-1938 Max.	*97*	...	...	...	...	*90*	*107*	*111*	*70*	*82*
1928-1938 Med.	*80*	...	...	...	...	*71*	*89*	*72*	*57*	*61*
1928-1938 Min.	*69*	...	...	...	...	*55*	*69*	*59*	*41*	*51*
1937	71	...	...	...	...	62	75	63	52	60
1938	69	...	...	...	...	55	69	59	41	51
1939	68	...	...	...	...	61	74	60	54	54
1940	69	...	...	...	...	68	79	71	54	67
1941	**98**	...	...	...	...	**98**	**111**	**105**	**88**	**89**
1942	**95**	...	...	...	...	**85**	**106**	87	62	**84**
1943	...	...	...	...	...	**85**	91	83	**71**	**94**

Communicable Diseases : Cases notified in Belgium.

	Median 1928-1938	1937	1938	1939	1940(*k*)	1941	1942	1943
Typhoid fever	*361(i)*	244	222	285	221	349	**671**	**635**
Paratyphoid fevers	*97*	**119**	64	66	69	112	**246**	**267**
Dysentery	*2*	1	1	0	**40**	**35**	**26**	**34**
Diphtheria	*2,090*	2,090	**2,513**	**2,419**	2,265	**4,271**	**5,464**	**16,072**
Scarlet fever	*2,472*	3,068	**4,236**	3,085	**3,855**	**3,270**	**4,536**	**12,470**
Cer.-sp. meningitis	*34*	20	19	32	32	**118**	**122**	**65**
Poliomyelitis	*59(j)*	**160**	44	**157**	37	111	63	68
Typhus fever	—	—	—	—	—	—	—	—
Smallpox	—	—	—	—	—	—	—	1
Notified cases	*5,115*	5,702	7,099	6,044	6,519(*k*)	8,266	11,128	29,612
Composite epidemic index	100	111.5	138.8	118.2	127.4(*k*)	161.6	217.6	578.9

The Netherlands

Owing to the high birth rate, and the resulting favourable make-up of the Netherlands population, the *general mortality* rate before the war was the lowest in Europe. As a result of the war, this rate has risen (*cf.* graph, page 602). During the first months of 1940 and 1941 it was influenced by a wave of influenza-like infections ; in May 1940, the direct effect of the invasion and of military operations was felt. The rate would, indeed, have shown a somewhat sharper rise if the effects of the bombing of Rotterdam had been taken into account [1]. The 1941 rate exceeded that of 1939 by 16%. A slight decrease took place afterwards. In the towns the rise was more marked and more sustained, revealing the effects of malnutrition. In Amsterdam the figures for the first 10 months of 1943 exceeded by 30% those of 1939.

With regard to *tuberculosis mortality*, a similar situation prevailed. In the whole of the Netherlands, the increase for 1943 as compared with 1939 was 70%. In Amsterdam it was 114%. It should be noted that, while these percentages are obviously significant, the number of deaths on which they are based is comparatively small, as efficient health services and high social and economic standards in pre-war years had resulted in an exceedingly low incidence of tuberculosis.

Infant mortality was apparently unaffected by the war and its results. The slight rise which took place in 1941 was followed by a resumption of the pre-war downward trend. Even in 1941 the rate did not exceed the median for the 11 years preceding the war. Before the war, the rates for Amsterdam were lower than those for the country as a whole ; they have remained lower, but the margin in their favour has decreased.

Among *communicable diseases*, diphtheria calls for special mention owing to its increased incidence from 1941 onwards. In 1943 the incidence was 13 times higher than the median for pre-war years. In 1943 there was an unusually high incidence of poliomyelitis, and also 4 cases of typhus fever of German origin.

[1] The official mortality statistics of Rotterdam showed for some weeks after the raid the effects of fatal wounds then sustained, but were not allowed to take account of the inhabitants crushed or burnt to death during the raid—some 30,000 according to estimates.

It would appear that, while malnutrition has made itself felt in the Netherlands, its severity has not been such as to influence general mortality to a very great extent. It was serious enough, however, to influence the tuberculosis death rate—a far more sensitive index. The epidemic situation remained quite normal, apart from the unusually large wave of diphtheria.

The vitality of the country has been shown by a definite rise of the birth rate in 1942 and again in 1943, which, in spite of the somewhat heightened mortality, has brought the natural increase of population in 1943 to a higher level than was reached during the 10 years preceding the war.

The Netherlands

Invaded by the German Army : May 10th, 1940; capitulation : May 14th, 1940.

Civilian **Population** used for calculating the rates.

Netherlands			Amsterdam		
C.	XII.1930 :	7,935,600	C.	XII.1930 :	757,400
O. N. E.	1.I.1939 :	8,727,300	O. N. E.	1.VII.1939 :	793,800
E. S.	VI.1940 :	8,878,600 *(a)*	O. N. E.	1.VII.1940 :	801,100
A. P.	1941 :	8,965,500	O. N. E.	1.VII.1941 :	799,900
O. N. E.	1.VII.1942 :	9,051,400	O. N. E.	1.VII.1942 :	800,300
O. N. E.	1.VII.1943 :	9,095,526 *(j)*	O. N. E.	1.VII.1943 :	796,800

Natural Movement of Population.

Year	Netherlands Births ‰ inhab.	Netherlands General mortality ‰ inhab.	Netherlands Increase (+) or decrease (−) ‰ inhab.	Amsterdam Births ‰ inhab.	Amsterdam General mortality ‰ inhab.	Amsterdam Increase (+) or decrease (−) ‰ inhab.
1928-1938 *Max.*	*23.3*	*10.7*	*+14.0*	*18.2*	*10.0*	*+9.4*
Med.	*20.8*	*8.8*	*+12.1*	*15.4*	*8.6*	*+7.3*
Min.	*19.8*	*8.4*	*+11.0*	*14.5*	*8.1*	*+5.7*
1937	19.8	8.8	+11.0	14.5	**8.8**	+5.7
1938	20.5	8.5	+12.0	15.4	8.1	+7.3
1939	20.6	8.6	+12.0	15.6	8.4	+7.2
1940	20.8 *(b)*	**9.9** *(b)*	+10.9	16.2	**9.9**	+6.3
1941	20.3	**10.0**	+10.3	15.2	**10.8**	+4.4
1942	21.0 *(b)*	9.5	+11.5	15.3	**10.6**	+4.7
1943 *(e)*	**22.9**	**9.7**	+13.2	17.4	**10.9**	+6.5

A. P. = Average population. C. = Census. O. N. E. = Official national estimate.
E. S. = Estimate of the Epidemiological Intelligence Service of the L. o. N.

(a) Average yearly population. *(b)* Corrected yearly rate. *(e)* First 10 months.
(g) Including war losses and deaths of civilians due to the war.
(h) Amœbic and bacillary. *(i)* Median for the period 1932-1938 only.
(j) At the beginning of 1943, the number of civilian workers of the Netherlands deported to Germany was estimated at 300,000.

Infant Mortality (deaths of infants under 1 year of age, per 1,000 live births). *(g)*

NETHERLANDS

	Yearly rates	I J	II F	III M	IV A	V M	VI J	VII —	VIII J	IX A	X S	XI O	XII N	XIII D
1928-1938 Max.	*59*	*71*	*93*	*95*	*65*	*56*	*44*	M	*43*	*42*	*53*	*51*	*47*	*58*
1928-1938 Med.	*44*	*54*	*58*	*55*	*50*	*44*	*37*	M	*34*	*33*	*35*	*37*	*39*	*51*
1928-1938 Min.	*37*	*40*	*37*	*39*	*43*	*34*	*30*	M	*28*	*27*	*29*	*32*	*34*	*37*
1937	38	54	53	51	43	34	30	M	28	27	31	34	35	37
1938	37	41	38	39	44	39	33	M	31	32	30	33	34	45
1939	34	38	39	35	37	31	31	M	29	25	32	32	35	41
1940 *(b)*....	39	62	65	45	39	35	35	M	27	25	29	30	36	48
1941 *(b)*....	43	59	66	51	48	47	38	M	32	32	31	33	**45**	44
1942	40	58	55	42	36	38	32	M	29	28	34	38	39	46
1943 *(e)*....	38	49	41	45	40	39	33	M	31	38	37	39	...	...

AMSTERDAM

1928-1938 Max.	*42*	*58*	*68*	*62*	*47*	*56*	*43*	*39*	*46*	*40*	*42*	*42*	*42*	*49*
1928-1938 Med.	*33*	*43*	*37*	*42*	*35*	*33*	*34*	*31*	*24*	*27*	*30*	*27*	*33*	*41*
1928-1938 Min.	*28*	*26*	*24*	*30*	*23*	*21*	*25*	*22*	*16*	*21*	*22*	*21*	*21*	*26*
1937	30	**50**	38	41	18	25	29	M	23	21	25	**32**	**38**	26
1938	29	33	20	41	26	**39**	23	M	27	27	26	**31**	26	33
1939	28	37	32	25	41	32	31	M	27	19	22	19	20	32
1940	29	43	39	29	26	22	22	M	26	15	31	30	**38**	30
1941	34	**54**	43	27	27	**47**	30	M	31	28	15	28	**52**	30
1942	31	38	38	39	29	33	24	M	20	26	25	**35**	**46**	32
1943 *(e)*....	35	**50**	30	32	37	**38**	39	M	**39**	32	28	22	...	...

General Mortality (rates per 1,000 inhabitants on a yearly basis). *(g)*

NETHERLANDS

	Yearly rates	I J	II F	III M	IV A	V M	VI J	VII —	VIII J	IX A	X S	XI O	XII N	XIII D
1928-1938 Max.	*10.7*	*14.1*	*17.8*	*18.5*	*11.5*	*10.2*	*8.6*	M	*8.4*	*8.0*	*8.0*	*8.7*	*9.1*	*10.4*
1928-1938 Med.	*8.8*	*10.2*	*10.8*	*10.6*	*9.7*	*9.1*	*8.3*	M	*7.4*	*7.4*	*7.3*	*7.8*	*8.3*	*9.6*
1928-1938 Min.	*8.4*	*9.5*	*9.2*	*9.6*	*8.9*	*8.3*	*7.6*	M	*7.0*	*6.9*	*6.8*	*7.3*	*7.9*	*8.6*
1937	8.8	**14.0**	10.4	9.7	8.9	8.3	7.5	M	7.2	6.9	6.9	7.8	8.3	9.4
1938	8.5	9.5	9.1	9.4	9.3	**9.3**	8.2	M	7.4	7.4	7.6	7.6	7.9	7.7
1939	8.6	9.9	10.4	10.6	9.2	8.6	8.2	M	7.2	6.9	7.1	**8.2**	8.2	9.5
1940 *(b)* ...	**9.9**	**13.4**	**13.2**	10.2	9.5	**14.9**	8.2	M	7.2	7.0	7.4	**8.4**	**8.7**	10.0
1941	**10.0**	**12.7**	**19.1**	11.0	10.3	**9.7**	**8.8**	M	**8.3**	7.0	**7.6**	7.9	**9.4**	9.4
1942	9.5	**11.7**	**12.9**	11.0	9.3	**9.3**	**8.5**	M	7.6	**7.7**	**7.7**	**8.7**	**9.1**	**10.2**
1943 *(e)*....	**9.7**	10.9	10.2	11.6	10.6	**9.9**	**8.6**	M	**8.6**	**8.4**	**8.7**	**9.6**	...	...

AMSTERDAM

1928-1938 Max.	*10.0*	*14.2*	*18.5*	*15.6*	*11.1*	*10.1*	*9.2*	*8.8*	*8.0*	*7.9*	*8.1*	*8.4*	*9.3*	*11.8*
1928-1938 Med.	*8.6*	*9.7*	*10.1*	*10.3*	*9.4*	*8.8*	*8.3*	*7.6*	*7.1*	*7.3*	*7.5*	*7.7*	*8.4*	*9.5*
1928-1938 Min.	*8.1*	*8.5*	*8.2*	*8.7*	*8.4*	*7.5*	*7.5*	*7.3*	*6.4*	*6.3*	*6.8*	*7.5*	*7.5*	*8.7*
1937	**8.8**	**13.9**	10.0	10.4	8.7	8.2	7.7	M	7.2	7.1	4.9	**7.9**	8.2	9.2
1938	8.1	9.5	8.0	8.5	8.6	8.9	7.8	M	7.3	6.7	**7.7**	7.6	7.5	9.4
1939	8.4	**10.6**	10.3	9.6	8.6	8.8	7.7	M	7.1	6.1	6.4	**8.2**	7.7	9.4
1940	**9.9**	**13.1**	**12.3**	10.2	10.1	**13.4**	7.8	M	**7.4**	**7.6**	**8.0**	**9.6**	**9.5**	**10.1**
1941	**10.8**	**12.9**	**18.9**	9.9	10.0	**9.9**	**9.1**	M	**9.4**	**8.2**	**9.1**	**11.8**	**11.0**	9.6
1942	**10.6**	**11.9**	**14.6**	**12.5**	9.8	**10.4**	**9.1**	M	**8.8**	**8.4**	**8.6**	**9.9**	**11.0**	**10.8**
1943 *(e)*....	**10.9**	**11.7**	11.2	**12.8**	**12.0**	**11.0**	**10.2**	M	**11.8**	**9.4**	**8.7**	**9.8**	...	...

Tuberculosis Mortality (rates per 100,000 inhabitants on a yearly basis).

	Netherlands					Amsterdam				
	Yearly rates	Quarterly I	II	III	IV	Yearly rates	Quarterly I	II	III	IV
1928-1938 Max.	*86*	*104*	*97*	*74*	*72*	*83*	*111*	*82*	*72*	*68*
1928-1938 Med.	*60*	*70*	*64*	*52*	*54*	*56*	*62*	*59*	*47*	*53*
1928-1938 Min.	*45*	*46*	*52*	*42*	*40*	*41*	*36*	*46*	*35*	*38*
1937	48	54	53	42	43	44	55	47	38	38
1938	45	46	52	43	40	41	36	46	43	39
1939	41	44	46	38	36	35	41	42	26	32
1940	44	47	48	40	39	37	40	44	32	32
1941	59	58	70	55	53	60	49	67	59	62
1942	61	60	69	58	58	**71**	65	**85**	**66**	**70**
1943	70	74	79	63	**65**	**75**	74	**95**	**71**	61

Communicable Diseases : Cases notified in the Netherlands.

	Median 1928-1938	1937	1938	1939	1940	1941	1942	1943
Typhoid fever	*359(i)*	237	204	172	108	167	**526**	**534**
Paratyphoid fevers	*258(i)*	523	206	286	266	310	305	345
Dysentery *(h)*	*390*	**425**	**1,019**	**712**	**1,193**	**3,428**	**4,119**	**5,360**
Diphtheria	*4,251*	1,073	1,273	1,273	1,730	**5,501**	**19,527**	**56,603**
Scarlet fever	*11,004*	10,109	11,555	10,257	8,841	7,198	12,694	28,576
Cer.-sp. meningitis	*129*	99	129	150	**277**	**448**	**198**	**160**
Poliomyelitis	*148*	60	691	403	111	452	198	**1,966**
Typhus fever	*0*	1	—	—	—	—	—	4
Smallpox	*0*	—	—	—	—	—	—	—
Notified cases	*16,539*	12,527	15,077	13.253	12,526	17,504	37,567	93,548
Composite epidemic index	100	75.7	91.2	80.1	75.6	105.8	227.1	565.6

Denmark

The *general mortality* rates recorded in Denmark, apart from that for 1940, are lower than those observed before the war (*cf.* graph, page 602). 1942 and 1943 are, indeed, record years—a rate of 9.6‰ of the population. For Copenhagen, the rates recorded from 1939 to 1943 inclusive are below the minimum of pre-war years.

The pre-war downward trend of *tuberculosis mortality* was checked and followed by stabilisation both in Denmark as a whole and in the city of Copenhagen.

On the other hand, the *infant mortality* rate continued to decline and reached low records in 1943 for both Denmark and the capital.

From the information available, it would appear that malnutrition does not prevail in Denmark as elsewhere in Europe. This unusual situation is largely attributable to the fact that Denmark is a producer

and exporter of foodstuffs, particularly milk and milk products, and probably suffered less interference than most other countries in its economic organisation as a result of occupation. Its exports went to Germany instead of to other countries.

Denmark has suffered from no unusual *epidemic prevalence*, the only fact worthy of notice being the continuance of a slight excess over the normal in the incidence of scarlet fever. There was no typhus fever or smallpox recorded among the civilian population. The situation as regards both the nutritional and the sanitary situation can be considered not merely as normal, but as satisfactory. In 1942 and 1943, the birth rate and the natural increase have shown a definite rise, far in excess of recent pre-war records.

DENMARK

Occupied by the German Army : April 9th, 1940.

Population used for calculating the rates.

DENMARK			COPENHAGEN		
C.	XI.1930 :	3,551,000	C.	XI.1932 :	625,200
E. S.	VI.1939 :	3,779,000	E. S.	VI.1939 :	697,000
E. S.	VI.1940 :	3,821,000	E. S.	VI.1940 :	700,700
E. S.	VI.1941 :	3,825,000	E. S.	VI.1941 :	702,800
O. N. E.	1.VII.1942 :	3,903,000	E. S.	VI.1942 :	707,800
O. N. E.	VI.1943 :	3,949,000			

Natural Movement of Population.

Year	DENMARK Births °/₀₀ inhab.	General mortality °/₀₀ inhab.	Increase (+) or decrease (−) °/₀₀ inhab.	COPENHAGEN Births °/₀₀ inhab.	General mortality °/₀₀ inhab.	Increase (+) or decrease (−) °/₀₀ inhab.
1928-1938 Max.	*19.6*	*11.4*	*+ 8.6*	*18.7*	*11.6*	*+7.9*
1928-1938 Med.	*18.0*	*11.0*	*+ 7.2*	*15.6*	*11.2*	*+4.4*
1928-1938 Min.	*17.3*	*10.3*	*+ 6.6*	*14.7*	*10.6*	*+3.1*
1937	18.0	10.8	+ 7.2	**17.8**	11.0	+6.8
1938	18.1 *(a)*	10.3 *(a)*	+ 7.8	**18.7**	10.8	+7.9
1939	17.8 *(a)*	10.1 *(a)*	+ 7.7	**18.2**	9.6	+8.6
1940	18.3 *(a)*	10.4	+ 7.9	**19.1**	10.0	+9.1
1941	18.5 *(a)*	10.3 *(a)*	+ 8.2	**16.9**	9.8	+7.1
1942.....	**20.4**	9.6	+10.8	**18.3**	9.5	+8.8
1943	**21.4**	9.6	+11.8	**20.0**	10.3	+9.7

C = Census. O. N. E. = Official national estimate.
E. S. = Estimate of the Epidemiological Intelligence Service of the L.o.N.
(a) Corrected yearly rate. *(b)* Median 1932-1938.
(e) Number of civilian workers deported to Germany at the beginning of 1943 : 48,000.
(f) For want of comparability, the monthly rates do not indicate whether the upper quartile has been exceeded.

Infant Mortality (deaths of infants under 1 year of age, per 1,000 live births).

DENMARK *(f)*

	Yearly rates	I J	II F	III M	IV A	V M	VI J	VII —	VIII J	IX A	X S	XI O	XII N	XIII D
1928-1938 Max.	*83*	...	...	...	...	...	...	M	...	...	...	...	...	...
1928-1938 Med.	*71*	...	...	...	...	...	...	M	...	...	...	...	...	...
1928-1938 Min.	*59*	...	...	...	...	...	...	M	...	...	...	...	...	...
1937	66	67	78	83	102	96	58	M	48	45	45	53	48	58
1938	59	59	67	76	70	71	59	M	48	51	42	44	50	60
1939 *(a)*	58	76	80	80	68	62	55	M	37	46	34	39	47	50
1940 *(a)*	50	57	55	64	62	62	51	M	34	32	33	35	48	51
1941	55	81	69	66	22	60	48	M	55	44	40	40	49	46
1942	47	62	51	51	52	54	44	M	46	37	30	40	45	54
1943	45	52			43			M	37			45		

COPENHAGEN

1928-1938 Max.	*71*	*105*	*74*	*79*	*85*	*82*	*66*	*77*	*61*	*48*	*48*	*68*	*68*	*64*
1928-1938 Med.	*56*	*57*	*56*	*52*	*52*	*62*	*45*	*41*	*38*	*38*	*34*	*43*	*46*	*47*
1928-1938 Min.	*40*	*39*	*41*	*33*	*36*	*37*	*33*	*29*	*29*	*29*	*24*	*28*	*29*	*20*
1937	40	48	52	48	47	59	29	M	29	38	28	29	35	43
1938	40	53	51	52	44	45	50	37	29	38	36	32	35	20
1939	34	28	37	31	37	45	33	24	27	21	**45**	31	43	43
1940	30	37	35	32	35	34	33	28	13	26	25	25	31	43
1941	35	38	38	33	27	26	37	30	27	36	33	46	43	37
1942	36	45	30	43	40	56	27	25	34	34	31	31	32	37
1943	34	40	46	33	29	32	23	M	20	28	20	30	42	40

General Mortality (rates per 1,000 inhabitants on a yearly basis).

DENMARK *(f)*

	Yearly rates	I J	II F	III M	IV A	V M	VI J	VII —	VIII J	IX A	X S	XI O	XII N	XIII D
1928-1938 Max.	*11.4*	...	...	...	...	...	...	M	...	...	...	...	...	...
1928-1938 Med.	*11.0*	...	...	...	...	...	...	M	...	...	...	...	...	...
1928-1938 Min.	*10.3*	...	...	...	...	...	...	M	...	...	...	...	...	...
1937	10.8	13.5	12.4	12.1	12.9	11.9	10.3	M	9.3	9.0	8.8	9.4	9.6	10.3
1938 *(a)*	10.3	10.7	10.7	11.4	11.6	12.3	10.6	M	9.7	9.4	9.0	9.2	9.4	10.7
1939 *(a)*	10.1	11.5	11.7	11.8	11.2	11.1	10.2	M	9.1	8.6	8.3	9.4	9.9	9.7
1940	10.4	11.0	13.6	13.8	12.5	12.0	9.7	M	8.4	8.0	8.7	8.6	9.4	9.4
1941 *(a)*	10.3	11.6	12.1	12.2	11.9	11.3	10.3	M	9.9	8.3	8.8	9.1	9.6	9.8
1942	9.6	10.6	11.0	10.3	10.4	10.2	9.7	M	9.0	8.9	7.8	8.5	9.0	10.0
1943	9.6	11.1	10.7	10.4	10.2	9.5	8.8	M	8.6	8.5	8.0	8.9	10.3	10.3

COPENHAGEN

1928-1938 Max.	*11.6*	*14.0*	*16.6*	*15.0*	*13.6*	*12.6*	*12.4*	*10.9*	*9.4*	*10.0*	*9.3*	*10.3*	*10.8*	*13.6*
1928-1938 Med.	*11.2*	*12.0*	*11.6*	*11.9*	*11.6*	*11.8*	*10.5*	*9.8*	*8.7*	*8.7*	*9.1*	*9.8*	*10.0*	*10.0*
1928-1938 Min.	*10.6*	*11.1*	*10.4*	*10.4*	*10.4*	*10.8*	*9.7*	*9.1*	*7.9*	*7.6*	*8.3*	*8.9*	*9.5*	*9.6*
1937	11.0	**13.5**	12.3	11.3	12.8	11.8	10.4	M	**9.4**	**9.5**	**9.3**	**10.5**	10.4	11.2
1938	10.8	12.7	11.2	11.9	11.9	**12.6**	10.7	M	**9.8**	8.2	**9.9**	9.9	9.9	10.7
1939	9.6	11.9	11.9	10.2	9.8	10.5	9.7	8.8	7.5	8.1	8.2	9.8	9.2	9.8
1940	10.0	11.1	11.5	**14.6**	12.2	12.4	10.5	8.9	7.3	7.4	8.6	8.5	9.2	9.2
1941	9.8	11.2	10.4	9.9	10.0	10.9	10.0	9.5	7.9	7.3	8.8	9.7	8.9	10.1
1942	9.5	9.8	10.4	9.9	10.5	10.6	9.8	9.0	8.6	8.4	8.0	9.1	9.4	10.0
1943	10.3	11.7	11.5	11.3	10.3	10.2	9.6	M	**9.9**	8.2	9.0	**10.4**	10.4	**11.6**

Tuberculosis Mortality (rates per 100,000 inhabitants on a yearly basis).

	Denmark					Copenhagen				
	Yearly rates	Quarterly I	II	III	IV	Yearly rates	Quarterly I	II	III	IV
1928-1938 Max.	*74*	*83*	*92*	*70*	*62*	*95*	*107*	*118*	*90*	*84*
1928-1938 Med.	*58*	*64*	*65*	*53*	*51*	*76*	*80*	*87*	*72*	*64*
1928-1938 Min.	*41*	*44*	*47*	*37*	*36*	*51*	*56*	*59*	*42*	*43*
1937	44	51	50	39	38	54	67	59	44	45
1938	41	44	47	37	36	51	58	59	42	43
1939	34	35	41	30	32	40	41	52	29	41
1940	35	37	43	28	33	44	37	51	36	50
1941	35	36	42	32	30	45	45	58	37	40
1942	35	36	37	31	34	44	43	51	38	42
1943	34	38	38	29	31	47	50	54	38	45
1944							45	39		

Communicable Diseases : Cases notified in Denmark.

	Median 1928-1938	1937	1938	1939	1940	1941	1942	1943
Typhoid fever	*74(b)*	47	43	20	21	60	44	37
Paratyphoid fevers	*134(b)*	**198**	98	110	69	78	76	84
Bacillary dysentery	*606*	**1,103**	367	402	399	**1,525**	474	**1,066**
Diphtheria	*3,037*	1,348	871	1,106	860	917	1,661	2,527
Scarlet fever	*3,285*	**9,516**	**8,950**	**9,079**	**7,336**	**9,136**	**14,302**	**16,124**
Cer.-sp. meningitis	*63*	62	60	56	55	**198**	**122**	**167**
Poliomyelitis	*164*	**1,245**	**562**	40	11	257	**615**	30
Typhus fever	—	—	—	—	—	—	—	—
Smallpox	—	—	—	—	—	—	—	—
Notified cases	*7,363*	13,519	10.951	10,813	8,751	12,171	17,294	20,035
Composite epidemic index	100	183.6	148.7	146.9	118.9	165.3	234.9	272.1

Northern Europe.

Norway

It is not possible to make a satisfactory estimate of the health situation prevailing in Norway, as vital statistics for the whole of the country have been withheld by censorship since the middle of 1941. We have to rely on the figures published for Oslo, although under war conditions the population of cities is less stable than that of countries as a whole, and the rates based on city populations are not so reliable as national rates.

Bearing this reservation in mind, we may indicate that, apart from a temporary rise in 1940, due to heightened mortality during the spring, the rate for *general mortality* for Oslo has remained practically stable for the last 7 years (*cf.* graph, page 602). The rate for 1943 was even slightly more favourable than those for previous years. The slight excess over normal observed in January 1944 is related to the wave of mild epidemic influenza which swept over Western Europe at that time.

Infant mortality has remained exceedingly low throughout the war period ; as the rate for infant mortality does not take into consideration population but the actual number of births, it is more trustworthy than either the general or the tubercnlosis death rates.

The downward trend of *mortality* from *tuberculosis* continued uninterrupted until the end of 1942. It rose, however, at the end of 1943 and early in 1944. There is an apparent inconsistency between the fact that satisfactory mortality rates have been maintained in Oslo and the many unofficial reports which indicate that the food situation in Norway in recent years has been very unsatisfactory and, in many localities and on many occasions, really acute. In pre-war years, Norwegian agriculture was far from meeting the requirements of the country. Moreover, the fish provided by the Norwegian fishing fleet, which formed a substantial part of the nation's diet, has been largely diverted to Germany, with a consequent shortage of proteins and vitamin-containing fish oils. School meals provided by the Swedish relief organisation can benefit but one section of the total population.

The geographical structure of Norway, and the consequent wide dispersal of the inhabitants and the seclusion in which they live, resulted in an unusually low prevalence of communicable diseases in normal years. The proportion of immune persons in the population, particularly in the outlying districts, was correspondingly low. This fact was observed more especially as regards tubercnlosis, but also held good as regards other communicable diseases.

It is therefore not surprising that the movements of young men, due to the attempted mobilisation in 1940 and since then to conscription for forced labour for the building of fortifications, resulted in a marked increase in the prevalence of epidemic disease as from the end of 1940 onwards. This was particularly the case as regards scarlet fever, which, in 1942, attained a prevalence three times greater than the pre-war median, and as regards diphtheria, of which,

in 1943, the incidence rose to 23 times that median. In 1941 there was also an abnormal increase in cases of cerebrospinal meningitis (20 times the pre-war figure) and in poliomyelitis (30 times that figure). The incidence of these diseases somewhat receded in 1942 and 1943, though remaining definitely higher than normal. The movements of these various communicable diseases are in keeping with what we know of the effects of the intermingling of germ-carriers and non-immune persons from comparatively isolated communities.

NORWAY

Invaded by the German Army : April 9th, 1940.

Population used for calculating the rates.

NORWAY			OSLO		
C.	XII.1930 :	2,814,000	C.	XII.1930 :	253 124
E. S.	VI.1939 :	2,929,400	O. N. E.	XII.1937 :	275,084
E. S.	VI.1940 :	2,944,700	O. N. E.	XII.1938 :	274,223
E. S.	VI.1941 :	2,958,000	O. N. E.	X.1939 :	275.004
			O. N. E.	XII.1940 :	271,608
			O. N. E.	XII.1941 :	273,914
			O. N. E.	XII.1942 :	268,721
			O. N. E.	XII.1943 :	264 387

Natural Movement of Population.

Year	NORWAY Births ‰ inhab.	NORWAY General mortality ‰ inhab.	NORWAY Increase (+) or decrease (−) ‰ inhab.	OSLO Births ‰ inhab.	OSLO General mortality ‰ inhab.	OSLO Increase (+) or decrease (−) ‰ inhab.
1928-1938 *Max.*	*17.9*	*11.5*	*+7.0*	*9.4*	*11.7*	*−1.3*
1928-1938 *Med.*	*15.6*	*10.4*	*+5.4*	*8.9*	*10.8*	*−2.3*
1928-1938 *Min.*	*14.4*	*9.9*	*+4.1*	*7.7*	*10.1*	*−3.2*
1937	15.1	10.4	+4.7	8.9	10.7	−1.8
1938	15.6	10.0	+5.6	9.1	10.4	−1.3
1939	15.9	10.2	+5.7	**9.8**	10.5	−0.7
1940	16.3	10.7	+5.6	**9.6**	**11.5**	−1.9
1940 (1.I-30.VI)	16.7	**12.0**	+4.7	...	...	...
1941	...	...	...	**9.7**	10.7	−1.0
1941 (1.I-30.VI)	15.8	**11.5**	+4.3	...	...	...
1942	...	...	...	**11.5**	10.6	+0.9
1943	...	...	...	**12.9**	10.0	+2.9

C. = Census. O. N. E. = Official national estimate.
E. S. = Estimate of the Epidemiological Intelligence Service of the L.o.N.

(e) Corrected yearly rates.
(f) For want of comparability, quarterly rates do not indicate whether the upper quartile has been exceeded.

Infant Mortality (deaths of infants under 1 year of age, per 1,000 live births).

NORWAY

1928-1938			1937	1938	1939
Max.	*Med.*	*Min.*			
54	*46*	*37*	42	37	37

OSLO

	Yearly rates	I J	II F	III M	IV A	V M	VI J	VII —	VIII J	IX A	X S	XI O	XII N	XIII D
1928-1938 *Max.*	*48*	*66*	*96*	*62*	*97*	*50*	*74*	*67*	*47*	*48*	*51*	*41*	*72*	*56*
1928-1938 *Med.*	*32*	*35*	*38*	*26*	*36*	*32*	*30*	*33*	*25*	*21*	*29*	*21*	*36*	*32*
1928-1938 *Min.*	*21*	*6*	*22*	*16*	*0*	*15*	*10*	*10*	*5*	*6*	*0*	*9*	*0*	*14*
1937 *(e)*	27	27	38	25	0	30	10	33	5	21	**51**	**33**	17	32
1938 *(e)*	38	**62**	**47**	**50**	37	15	30	16	**36**	19	33	**41**	37	28
1939 *(e)*	30	32	22	19	27	27	37	27	23	**32**	**45**	**44**	26	11
1940 *(e)*	32	30	13	**60**	23	29	21	32	**45**	19	25	**60**	34	26
1941 *(e)*	33	37	**65**	**55**	37	19	19	26	17	25	16	29	42	18
1942 *(e)*	27	28	38	28	30	45	**39**	28	34	**29**	8	25	45	**42**
1943	31	50	22	37	24	34	16	38	13	19	20	**33**	43	**48**
1944	...	40	35	49	29	...	...	...	...	...	...	...	...	...

General Mortality (rates per 1,000 inhabitants on a yearly basis).

NORWAY *(f)*

	Yearly rates	I J	II F	III M	IV A	V M	VI J	VII —	VIII J	IX A	X S	XI O	XII N	XIII D
1928-1938 *Max.*	*11.5*		...			...				...			...	
1928-1938 *Med.*	*10.4*		...			...				...			...	
1928-1938 *Min.*	*9.9*		...			...				...			...	
1937	10.4		12.3			10.2				9.0			10.1	
1938	10.0		10.6			10.4				9.0			9.8	
1939	10.2		11.7			10.9				8.6			9.2	
1940	10.7		11.8			12.1				9.3			9.7	
1941	**11.5**		12.7			10.2				...			...	
1942														
1943														

OSLO

	(e)													
1928-1938 *Max.*	*11.7*	*14.6*	*17.5*	*19.9*	*14.4*	*11.7*	*11.4*	*10.8*	*9.5*	*9.7*	*10.1*	*10.6*	*11.9*	*11.2*
1928-1938 *Med.*	*10.8*	*11.5*	*11.6*	*11.9*	*10.5*	*10.6*	*10.5*	*9.9*	*8.9*	*8.2*	*9.5*	*9.6*	*10.0*	*10.6*
1928-1938 *Min.*	*10.1*	*10.4*	*10.8*	*9.6*	*10.3*	*9.1*	*9.3*	*8.5*	*7.9*	*7.6*	*8.4*	*9.0*	*8.8*	*9.1*
1937 *(e)*	10.7	**14.3**	11.6	9.9	10.5	10.8	10.1	**10.7**	8.9	7.6	9.1	**10.6**	9.4	10.0
1938 *(e)*	10.4	11.5	12.2	10.6	10.3	10.5	10.5	9.7	8.9	7.6	9.5	9.7	**11.9**	9.1
1939 *(e)*	10.5	12.8	11.1	**13.8**	11.6	10.8	9.3	8.2	8.2	7.6	9.1	9.4	9.3	10.2
1940 *(e)*	**11.5**	11.2	**14.7**	**15.3**	**12.5**	11.2	9.6	7.9	9.0	7.3	9.5	**10.7**	9.3	9.5
1941 *(e)*	10.7	11.6	**14.1**	12.2	10.7	9.1	10.3	7.8	8.3	8.4	**10.1**	**10.6**	**11.5**	10.8
1942 *(e)*	10.6	9.8	11.9	10.5	10.3	10.7	8.9	9.5	7.5	7.6	9.0	**10.4**	**11.3**	10.3
1943	10.0	10.5	10.7	9.8	10.3	10.7	9.9	6.3	**9.3**	8.7	**10.6**	9.2	9.6	**11.5**
1944	...	**13.3**	11.4	11.4	**12.3**	...	...	...	...	...	...	...	...	...

Tuberculosis Mortality (rates per 100,000 inhabitants on a yearly basis).

	Norway					Oslo				
	Yearly rates	Quarterly I	II	III	IV	Yearly rates	Quarterly I	II	III	IV
1928-1938 Max.	*158*	...	...	...	...	*165*	*195*	*177*	*144*	*139*
1928-1938 Med.	*120*	...	...	...	...	*108*	*142*	*121*	*108*	*95*
1928-1938 Min.	*88*	...	...	...	...	*91*	*83*	*94*	*79*	*67*
1937	98	...	...	...	...	98	104	108	81	82
1938	88	...	...	...	...	91	99	120	79	72
1939	86	...	...	...	...	75	88	70	63	58
1940 *(e)* ...						76	67	72	60	66
1941 *(e)* ...						66	85	63	60	59
1942 *(e)* ...						60	58	69	41	63
1943						69	66	74	62	75
1944							79	66		

Communicable Diseases: Cases notified in NORWAY.

	Median 1928-1938	1937	1938	1939	1940	1941	1942	1943
Typhoid fever	*58*	49	42	61	72	73	58	87
Paratyphoid fevers.	*99*	69	24	45	59	53	25	**296**
Dysentery	*71*	**715**	25	36	66	**302**	**1,227**	**405**
Diphtheria	*974*	406	190	54	138	**2,611**	**8,349**	**22,787**
Scarlet fever	*4,258*	4,979	3,317	**5,373**	3,825	**6,131**	**13,908**	**11,673**
Cer.-sp. meningitis .	*25*	22	26	28	**88**	**490**	**259**	**191**
Poliomyelitis	*117*	178	102	59	53	**1,726**	**279**	**457**
Smallpox	—	—	—	—	—	—	—	—
Notified cases	*5,602*	6,418	3,726	5,656	4,301	11,386	24,105	35,896
Composite epidemic index	100	114.6	66.5	101.0	76.8	203.2	430.3	640.8

SWEDEN

The downward tendency of *general mortality* has continued unchecked during the war years, both in Sweden as a whole and in Stockholm, low records being observed in 1942 in Sweden and in 1943 in Stockholm (*cf.* graph, page 600). Sweden as well as Norway escaped even the waves of influenza-like diseases which raised the general mortality in Western Europe during the first quarter of 1940 and of 1941.

Throughout the war period, *tuberculosis* death rates remained below the minimum of the preceding decade. In Stockholm their downward movement continued unchecked.

Infant mortality also declined, in 1942 and 1943 reaching low records both in Sweden as a whole and in the capital.

There is thus no indication of any deterioration in the health situation.

The only unusual fact regarding *epidemic diseases* is the high prevalence of scarlet fever, which, however, remains below the peak reached in 1939. The increased incidence of meningitis is not abnormal in a country which has had to undergo partial mobilisation. Mobilisation has also been blamed for the significant rise in venereal diseases recorded in 1942 and 1943, particularly in Stockholm.[1]

SWEDEN

Neutral country.

Population used for calculating the rates.

SWEDEN			STOCKHOLM		
C.	XII.1930 :	6,142,000	C.	XII.1930 :	502,200
O. N. E.	VI.1939 :	6,325 800	O. N. E.	VI.1939 :	577,200
O. N. E.	VI.1940 :	6 356 400	O. N. E.	VI.1940 :	587,000
O. N. E.	VI.1941 :	6 389 000	O. N. E.	VI.1941 :	595,300
O. N. E.	VI.1942 :	6,432 300	O. N. E.	VI.1942 :	606,900
O. N. E.	VI.1943 :	6,490.500	O. N. E.	VI.1943 :	624,000

Natural Movement of Population.

Year	SWEDEN Births °/₀₀ inhab.	SWEDEN General mortality °/₀₀ inhab.	SWEDEN Increase (+) or decrease (−) °/₀₀ inhab.	STOCKHOLM Births °/₀₀ inhab.	STOCKHOLM General mortality °/₀₀ inhab.	STOCKHOLM Increase (+) or decrease (−) °/₀₀ inhab.
1928-1938 *Max.*	*16.1*	*12.5*	*+4.1*	*12.7*	*11.5*	*+ 2.1*
1928-1938 *Med.*	*14.5*	*11.7*	*+2.5*	*10.6*	*11.0*	*− 0.4*
1928-1938 *Min.*	*13.7*	*11.2*	*+2.1*	*9.8*	*10.6*	*− 1.5*
1937	14.4	**12.0**	+2.4	**11.4**	**11.1**	+ 0.3
1938	14.9	11.5	+3.4	**12.7**	10.6	+ 2.1
1939	**15.4**	11.5	+3.9	**13.4**	10.4	+ 3.0
1940	15.1	11.4	+3.7	**13.6**	10.7	+ 2.9
1941	**15.6**	11.3	+4.3	**15.7**	10.6	+ 5.1
1942	**17.7**	9.9	+7.8	**18.0**	8.9	+ 9.1
1943	**19.3**	10.1	+9.2	**20.0**	9.5	+11.5

C. = Census.
O. N. E. = Official national estimate.

(a) Corrected yearly rates.
(b) Median 1930-1938.
(c) Monthly rates based on the period 1930-1938.

[1] There the number of new cases of gonorrhœa recorded doubled between 1940 and 1943 ; that of syphilis increased fourfold.

Infant Mortality (deaths of infants under 1 year of age, per 1,000 live births).

SWEDEN

	Yearly rates	I J	II F	III M	IV A	V M	VI J	VII —	VIII J	IX A	X S	XI O	XII N	XIII D
1928-1938 Max.	*59*	*86*	*80*	*76*	*75*	*65*	*61*		*53*	*48*	*42*	*52*	*58*	*68*
1928-1938 Med.	*50*	*56*	*60*	*55*	*53*	*55*	*53*		*41*	*41*	*34*	*41*	*49*	*52*
1928-1938 Min.	*42*	*47*	*50*	*48*	*47*	*43*	*41*		*33*	*32*	*30*	*35*	*39*	*42*
1937	45	49	56	59	49	45	41		36	36	31	36	49	55
1938	42	47	50	50	48	48	45		38	36	30	35	39	42
1939	39	49	47	45	47	43	45		32	26	29	33	39	40
1940	39	49	60	51	38	38	39		31	26	27	35	36	38
1941	37		45			41				29			33	
1942	29		37			29				24			27	
1943	29		33			28				33			31	

STOCKHOLM

	Yearly rates	I J	II F	III M	IV A	V M	VI J	VII —	VIII J	IX A	X S	XI O	XII N	XIII D
1930-1938 Max.	*48*	*73*	*61*	*53*	*62*	*63*	*60*		*52*	*63*	*42*	*44*	*43*	*60*
1930-1938 Med.	*38*	*45*	*39*	*45*	*43*	*42*	*39*		*29*	*34*	*30*	*32*	*40*	*45*
1930-1938 Min.	*28*	*22*	*27*	*30*	*21*	*24*	*29*		*20*	*27*	*24*	*20*	*17*	*16*
1937	38	45	33	50	36	46	34		20	**41**	24	35	**42**	49
1938	35	22	35	48	30	42	38		29	**41**	**32**	26	40	36
1939	32	23	29	40	43	36	21		26	20	25	36	**43**	43
1940	34	37	**53**	36	37	27	42		24	17	**33**	27	38	32
1941	29	32	28	44	29	30	37		29	27	22	24	20	29
1942	22	27	31	19	22	26	17		17	22	17	29	21	17
1943	23	...	...	...	...	...	...		...	...	...	...	...	...

General Mortality (rates per 1,000 inhabitants on a yearly basis).

SWEDEN

	Yearly rates	I J	II F	III M	IV A	V M	VI J	VII —	VIII J	IX A	X S	XI O	XII N	XIII D
1928-1938 Max.	*12.5*	*18.2*	*17.5*	*14.8*	*13.8*	*13.4*	*12.5*		*11.3*	*10.4*	*10.4*	*11.3*	*11.5*	*12.7*
1928-1938 Med.	*11.7*	*13.0*	*12.9*	*13.0*	*12.8*	*12.3*	*11.8*		*10.7*	*9.8*	*10.0*	*10.5*	*10.9*	*11.8*
1928-1938 Min.	*11.2*	*11.6*	*11.9*	*12.3*	*12.2*	*11.8*	*11.1*		*10.1*	*9.3*	*9.6*	*10.1*	*10.5*	*11.1*
1937	**12.0**	13.3	**15.1**	**14.5**	13.1	12.2	11.4		10.5	9.7	10.0	**10.9**	**11.3**	**12.4**
1938	11.5	13.0	12.5	12.5	12.7	**12.9**	11.8		10.9	**10.1**	9.9	10.2	10.6	11.3
1939	11.5	12.6	12.1	13.4	**14.8**	12.1	11.4		10.1	9.7	9.5	10.4	10.6	11.6
1940	11.4	13.5	**14.9**	13.4	12.4	11.6	10.9		10.1	9.3	10.0	9.9	10.5	10.9
1941	11.3	**17.0**	13.5	11.5	12.1	11.9	10.4		**11.0**	9.0	8.7	9.7	9.8	10.2
1942	9.9	11.1	10.8	11.1	10.9	10.2	9.4		9.3	8.8	8.3	9.6	9.4	10.0
1943	10.1		10.6			10.4				9.1			10.5	

STOCKHOLM

	Yearly rates	I J	II F	III M	IV A	V M	VI J	VII —	VIII J	IX A	X S	XI O	XII N	XIII D
1928-1938 Max.	*11.5*	*16.8*	*15.9*	*13.8*	*13.2*	*12.1*	*11.8*		*10.7*	*10.5*	*10.4*	*10.8*	*11.0*	*12.0*
1928-1938 Med.	*11.0*	*12.0*	*12.0*	*12.2*	*11.8*	*11.2*	*11.3*		*9.6*	*9.5*	*9.9*	*9.9*	*9.9*	*11.0*
1928-1938 Min.	*10.6*	*9.6*	*10.4*	*11.1*	*10.8*	*10.3*	*9.9*		*9.2*	*8.8*	*8.7*	*9.3*	*9.7*	*9.9*
1937	**11.1**	**13.1**	**15.9**	12.2	11.8	11.0	10.7		9.2	**9.8**	9.0	9.6	**10.5**	**11.1**
1938	10.6	12.0	11.9	11.6	11.1	11.2	10.9		9.6	9.6	9.3	9.3	9.7	11.0
1939	10.4	10.6	11.1	12.1	**12.2**	11.0	10.1		9.3	9.5	8.5	9.8	9.4	**11.3**
1940 *(a)* ...	10.7	**12.5**	**12.9**	11.2	11.6	10.6	9.8		9.2	7.8	9.7	9.6	**10.2**	11.0
1941 *(a)* ...	10.6	**14.7**	11.8	10.7	11.4	10.2	9.3		9.9	8.0	9.3	9.1	8.6	9.8
1942 *(a)* ...	8.9	8.6	10.2	9.5	9.2	8.8	8.1		8.5	7.8	7.7	8.6	9.0	9.3
1943 *(a)* ...	9.5	9.1	9.2	10.5	10.5	8.3	8.4		8.2	8.0	8.6	9.4	9.4	9.8

Attention should be directed to the very unusual increase in the *birth rate* which has taken place during the last three years and was particularly marked in Stockholm (50% above the 1939 figure, the proportion being 25% for the whole country). It should also be noted that this rise began very slowly in 1933, but was greatly accelerated during the war.

Tuberculosis Mortality (rates per 100,000 inhabitants on a yearly basis).

	Sweden					Stockholm				
	Yearly rates	Quarterly I	II	III	IV	Yearly rates	Quarterly I	II	III	IV
1928-1938 Max.	*130*	*140*	*148*	*122*	*120*	*156*	*168*	*177*	*144*	*151*
1928-1938 Med.	*104*	*113*	*120*	*93*	*90*	*111*	*136*	*135*	*100*	*90*
1928-1938 Min.	*82*	*91*	*94*	*73*	*72*	*84*	*100*	*82*	*77*	*74*
1937	86	92	99	77	77	84	100	82	78	79
1938	82	91	94	73	72	92	105	100	77	86
1939	75	83	82	70	65	83	77	91	87	76
1940	71	76	80	67	61	74	80	76	66	73
1941	75	77	87	71	64	75	73	89	69	71
1942	68	...	...	...	...	70	75	73	68	63
1943	...	...	...	...	...	71	75	81	68	62
1944							85	74		

Communicable Diseases : Cases notified in Sweden.

	Median 1928-1938	1937	1938	1939	1940	1941	1942	1943
Typhoid and paratyphoid fevers	*447(b)*	**545**	447	497	464	**578**	240	445
Bacillary dysentery.	*522*	328	522	279	171	565	1,304	**1,792**
Diphtheria	*1,708*	299	716	188	290	252	1,285	2,496
Scarlet fever	*8,046*	**14,601**	**26,279**	**40,889**	**24,550**	**13,402**	**20,716**	**29,911**
Cer.-sp. meningitis	*58*	49	53	31	29	**110**	**110**	**139**
Poliomyelitis	*937*	**2,041**	**1,450**	607	441	737	773	**2,169**
Typhus fever	—	—	—	—	—	—	—	—
Smallpox	—	—	—	—	—	—	—	—
Notified cases	*11,718*	17,863	29,467	42,491	25,945	15,644	24,428	39,952
Composite epidemic index	100	152.4	251.5	362.6	221.4	133.5	208.5	340.9

Finland

Vital statistics are not available for Finland since June 1941, and it is therefore not possible to judge of the effect on them of the campaign started against Russia at that time.

At all events, the short winter campaign of 1939/40 resulted in a rise in *general mortality* from 13.1 per 1,000 population in 1938 to 14.8 in 1939, and 20.0 in 1940, reverting to 13.9 during the first half of 1941. But this campaign was not accompanied by any significant increase in *communicable diseases*, only a small rise in water-borne infections being noticeable. The absence of typhus fever in a country so close to Russia is particularly remarkable and can only be ascribed to the traditional habits of personal cleanliness of the Finns, which make epidemics of louse-borne diseases impossible.

In peace-time, Finland had to import cereals and other foodstuffs ; mobilisation hampered production, imports from overseas were practically impossible, except to a limited extent from Germany. It is therefore not surprising that there should have been many accounts of food shortage in Finland and consequent malnutrition, particularly among children. Appeals to Swedish and other relief organisations were made with success on their behalf. We have unfortunately been unable to obtain precise information as to the extent and severity of malnutrition, though various forms of vitamin-deficiency have been mentioned—a quite likely state of affairs in a country where the climate is not favourable to fruit production.

FINLAND

Communicable Diseases : Cases notified in FINLAND.

	Median 1928-1938	1937	1938	1939	1940	1941	1942	1943
Typhoid fever	*384 (a)*	**499**	255	146	380	**666**	...	...
Paratyphoid fevers .	*742 (a)*	838	444	902	**1,948**	**1,566**	...	...
Dysentery	*47*	51	29	9	**137**	22	...	...
Diphtheria	*1,993*	**4,339**	2,936	2,797	3,246	1,932	...	...
Scarlet fever	*4,654*	**10,976**	**8,409**	6,440	7,392	3,355	...	...
Poliomyelitis	*191*	207	619	85	**572**	81	...	...
Typhus fever	—	1	—	—	—	—	...	...
Smallpox	*2*	2	—	—	—	—	...	...
Notified cases	*8,013*	16,913	12,692	10,379	13,675	7,622	...	...
Composite epidemic index	100	211.1	158.4	129.5	170.7	95.1	...	...

(a) Median based on the period 1932-1938 only.

Central Europe.

SWITZERLAND

Apart from a temporary rise in the first quarter of 1940, the *general mortality* rate in Switzerland during the war was practically stable, with a slight downward tendency. This is true not only for the country as a whole, but for the towns with a population exceeding 30,000 inhabitants. (*Cf.* graph, page 600.)

Tuberculosis mortality has also remained fairly stable, at a low level, a new low record being reached in 1943. This was the case in the towns as well as in the rest of the country.

Infant mortality in 1942 reached a low record, in both town and country.

These findings are in keeping with the satisfactory food situation that has prevailed in spite of the considerable difficulties experienced in importing foodstuffs into a country which produced but a part of its normal consumption. Storage on a large scale begun before the war, the preparation in advance of a scheme of rationing and the systematic extension of cultivation (WAHLEN plan) are to a large extent responsible for the success achieved. The only element of the population which has suffered consists of town dwellers with low incomes, who have had difficulty in paying for the food to which they are entitled, the index number of the cost of foodstuffs having risen by 66% between September 1939 and April 1944. For other groups, rationing has been a source of difficulties rather than an actual hardship. Mention should also be made of the preventive action undertaken in certain cantons and large towns against vitamin deficiencies, by the distribution of vitamin tablets to school-children.

There has been a noticeable absence of abnormal prevalence of *communicable diseases*. The European diphtheria pandemic has not affected Switzerland, except for a slight rise in the prevalence of the disease in 1943. The meningitis rate showed a marked rise in 1940, as in all countries where mobilisation took place, but has greatly declined since. Mobilisation is also blamed for a definite increase in the prevalence of venereal disease. In 1942 Sonne

dysentery in a mild form made its appearance, as did epidemic hepatitis. Neither affected the mortality rate. Neither smallpox nor typhus fever occurred (except laboratory cases).

The *birth rate* rose steeply from 1941 onwards. The increase as compared with 1939 was 26% in the country as a whole and nearly 40% in towns with more than 30,000 inhabitants. The rise coincided with legislation granting subsidies to families of mobilised men.

SWITZERLAND

Neutral country.

Population used for calculating the rates.

SWITZERLAND			TOWNS WITH MORE THAN 30,000 INHABITANTS		
C.	XII.1930 :	4,066,400	C.	XII.1930 :	947,618
O. N. E.	VI.1939 :	4,205,600	O. N. E.	VI.1939 :	1,076,200
O. N. E.	VI.1940 :	4,226,400	O. N. E.	VI.1940 :	1,083,900
O. N. E.	VI.1941 :	4,253,700	O. N. E.	VI.1941 :	1,089,900
O. N. E.	VI.1942 :	4,283,300	O. N. E.	VI.1942 :	1,098,500
E. S.	VI.1943 :	4,313,000	E. S.	VI.1943 :	1,108,000

Natural Movement of Population.

Year	SWITZERLAND Births °/₀₀ inhab.	General mortality °/₀₀ inhab.	Increase (+) or decrease (−) °/₀₀ inhab.	TOWNS WITH MORE THAN 30,000 INHABITANTS *(g)* Births °/₀₀ inhab.	General mortality °/₀₀ inhab.	Increase (+) or decrease (−) °/₀₀ inhab.
1928-1938 *Max.*	*17.4*	*12.5*	*+5.6*	*12.1*	*10.9*	*+1.3*
1928-1938 *M d.*	*16.4*	*11.6*	*+4.6*	*(g) 11.6*	*10.6*	*+1.1*
1928-1938 *Min.*	*15.0*	*11.3*	*+3.6*	*10.9*	*10.4*	*+0.3*
1937	15.0	11.3	+3.7	10.9	10.4	+0.5
1938	15.2	11.6	+3.6	11.0	10.7	+0.3
1939	15.2	11.8	+3.4	11.2	**10.9**	+0.3
1940	15.2	12.0	+3.2	**11.9**	**11.0**	+0.9
1941	16.9	11.1	+5.8	**13.8**	10.6	+3.2
1942	**18.4**	11.0	+7.4	**15.3**	10.4	+4.9
1943 *(a)*.....	**19.2**	11.0	+8.2	**15.7**	10.5	+5.2

C. = Census.
E. S. = Estimate of the Epidemiological Intelligence Service of the L.o.N.
O. N. E. = Official national estimate.

(a) Provisional rates.
(b) Laboratory infections.
(e) Median 1932-1938.
(f) Plus an epidemic of an unknown number of cases.
(g) Retrospective rates based on the period 1934-1938.

Infant Mortality (deaths of infants under 1 year of age, per 1,000 live births).

SWITZERLAND

	Yearly rates	I J	II F	III M	IV A	V M	VI J	VII —	VIII J	IX A	X S	XI O	XII N	XIII D
1928-1938 Max.	*54*	*65*	*70*	*58*	*61*	*56*	*53*		*49*	*54*	*49*	*52*	*55*	*56*
1928-1938 Med.	*48*	*57*	*52*	*53*	*51*	*48*	*48*		*43*	*42*	*42*	*44*	*46*	*54*
1928-1938 Min.	*43*	*43*	*43*	*44*	*44*	*40*	*44*		*37*	*36*	*36*	*37*	*40*	*40*
1937	47	56	50	52	50	**50**	46		46	43	42	44	40	40
1938	43	43	43	44	44	42	47		40	38	39	37	44	51
1939	43	48	47	45	42	42	40		38	36	35	37	50	53
1940	46	**63**	58	50	49	48	48		38	37	37	40	41	39
1941	41	44	**61**	50	43	39	37		41	36	38	37	36	41
1942	38	42	42	43	33	36	33		37	32	36	36	44	48
1943 *(a)*	40	57	42	36	38	37	38		39	39	34	36	36	44

TOWNS WITH MORE THAN 30,000 INHABITANTS

1930-1938 Max.	*46*	*60*	*64*	*57*	*62*	*46*	*54*		*51*	*42*	*44*	*47*	*54*	*59*
1930-1938 Med.	*42*	*47*	*44*	*52*	*48*	*38*	*46*		*38*	*35*	*34*	*36*	*40*	*47*
1930-1938 Min.	*38*	*39*	*37*	*32*	*30*	*30*	*26*		*28*	*24*	*24*	*24*	*31*	*32*
1937	39	43	37	**57**	**62**	37	26		38	29	**44**	32	31	32
1938	38	**50**	41	36	30	36	**46**		35	**42**	29	24	**49**	42
1939	36	32	43	36	31	**48**	31		33	35	30	37	41	33
1940	37	49	47	50	40	30	40		25	31	30	39	30	35
1941	36	42	49	**54**	38	37	31		34	33	36	28	27	35
1942	35	34	40	38	26	29	40		32	35	23	33	43	49

General Mortality (rates per 1,000 inhabitants on a yearly basis).

SWITZERLAND

	Yearly rates	I J	II F	III M	IV A	V M	VI J	VII —	VIII J	IX A	X S	XI O	XII N	XIII D
1928-1938 Max.	*12.5*	*14.8*	*17.4*	*17.7*	*14.7*	*12.7*	*11.7*		*11.3*	*10.4*	*10.1*	*11.2*	*11.8*	*12.5*
1928-1938 Med.	*11.6*	*13.1*	*13.3*	*13.9*	*13.2*	*12.1*	*11.2*		*10.4*	*9.8*	*9.7*	*10.3*	*11.0*	*12.2*
1928-1938 Min.	*11.3*	*12.6*	*12.6*	*12.4*	*12.0*	*10.8*	*10.1*		*10.0*	*9.5*	*9.4*	*10.0*	*10.7*	*11.4*
1937	11.3	13.0	13.2	13.1	12.4	11.6	10.1		10.2	9.6	**10.0**	10.2	11.0	12.1
1938	11.6	13.0	12.6	14.3	13.4	**12.3**	11.2		**10.4**	9.7	9.5	10.1	11.2	**12.3**
1939	11.8	13.4	**16.3**	13.8	12.1	11.5	11.0		10.0	**9.9**	9.3	10.5	11.1	**12.9**
1940	12.0	**17.7**	16.1	**14.3**	12.7	11.1	10.4		9.8	9.5	9.6	10.1	10.7	11.8
1941	11.1	13.4	12.9	12.2	12.0	11.1	10.8		10.1	9.5	9.8	9.8	10.9	11.3
1942	11.0	12.3	13.4	12.7	10.9	10.9	9.9		9.5	9.5	9.0	9.7	10.9	**12.7**
1943 *(a)*	11.0	13.2	12.2	11.7	11.2	10.6	10.3		10.0	9.5	9.5	10.2	11.3	12.1

TOWNS WITH MORE THAN 30,000 INHABITANTS

1930-1938 Max.	*11.0*	*13.2*	*14.9*	*16.1*	*11.9*	*11.0*	*10.7*		*10.3*	*9.7*	*10.1*	*10.5*	*11.3*	*11.8*
1930-1938 Med.	*10.7*	*12.0*	*12.1*	*11.7*	*11.2*	*10.6*	*10.2*		*9.7*	*9.4*	*9.3*	*9.8*	*10.4*	*11.1*
1930-1938 Min.	*10.4*	*11.0*	*11.2*	*11.2*	*10.9*	*9.3*	*9.1*		*9.0*	*8.9*	*8.7*	*9.8*	*9.8*	*10.7*
1937	10.4	11.6	11.6	11.7	11.1	9.8	9.1		9.7	9.3	9.6	9.8	**11.1**	11.0
1938	10.7	12.0	11.5	**13.2**	**11.7**	**10.9**	9.7		9.2	**9.6**	9.1	9.8	10.5	11.1
1939	10.9	**12.4**	**15.7**	12.1	10.5	10.8	9.8		9.2	**9.6**	8.9	**10.4**	10.0	**11.7**
1940	**11.0**	**14.3**	**14.0**	12.7	11.2	**10.9**	10.0		9.0	9.3	9.5	9.9	10.3	10.7
1941	10.6	**13.3**	11.6	11.9	11.3	9.8	9.9		9.5	9.2	9.8	9.4	10.6	10.7
1942	10.4	11.1	13.1	11.7	10.3	10.3	9.5		9.5	9.3	9.0	9.3	10.5	11.3
1943 *(a)*	10.5	**12.2**	11.0	10.7	10.3	10.0	**10.7**		9.4	**9.5**	9.0	10.1	**10.9**	**11.8**

Tuberculosis Mortality (rates per 100,000 inhabitants on a yearly basis).

	Switzerland					Towns with more than 30,000 inhabitants				
	Yearly rates	Quarterly I	II	III	IV	Yearly rates	Quarterly I	II	III	IV
1928-1938 Max.	*135*	*148*	*161*	*125*	*111*	*135*	*153*	*154*	*121*	*122*
1928-1938 Med.	*106*	*118*	*117*	*101*	*92*	*105*	*114*	*117*	*100*	*84*
1928-1938 Min.	*82*	*90*	*96*	*76*	*68*	*79*	*83*	*87*	*65*	*62*
1937	87	91	102	80	75	80	83	93	75	69
1938	82	90	96	76	68	79	101	87	65	62
1939	80	85	92	73	71	74	72	92	63	70
1940	78	89	84	69	71	74	81	83	65	67
1941	79	80	89	73	75	76	74	82	69	77
1942	83	86	95	82	68	78	83	78	88	66
1943 *(a)*	76	83	89	67	66	74	85	82	64	67
1944 *(a)*		87					84			

Communicable Diseases : Cases notified in Switzerland.

	Median 1928-1938	1937	1938	1939	1940	1941	1943	1943
Typhoid fever	*103 (e)*	74	115	67	77	70	117	114
Paratyphoid fevers	*92 (e)*	**153**	99	88	115	87	**286**	123
Dysentery	*3*	2	**6**	1	1	**5**	**1,067**	**1,504**
Diphtheria	*2,265*	772	716	751	662*(f)*	1,114	1,800	**3,655**
Scarlet fever	*3,268*	**4,363**	**5,268**	**4,547**	**4,601**	3,311	2,879	2,625
Cer.-sp. meningitis	*43*	32	33	49	**710**	**250**	**159**	**96**
Poliomyelitis	*229*	**1,492**	286	**521**	237	**1,479**	**669**	170
Typhus fever	*0*	—	—	—	—	2*(b)*	—	—
Smallpox	*0*	—	—	—	—	—	—	—
Notified cases	*6,003*	6,888	6,523	6,024	6,403	6,318	6,977	8,287
Composite epidemic index	100	114.7	108.7	100.3	106.7	105.2	116.2	138.0

Germany

Examination of the health situation in Germany is rendered very difficult owing to variations in the population on which statistics are based. The following study is concerned exclusively with the territory of 1937 (Altreich) in order to avoid statistical variations resulting from the addition of populations differing from the original population as regards both composition according to age and sex, and degree of urbanisation and its consequences with regard to health conditions. Notwithstanding this, the German population on which the statistics are based has nevertheless undergone consi-

derable changes which, for obvious reasons, have not been satisfactorily indicated in official figures now available. The population has undergone mobilisation, progressive but to an extent without precedent, to supply the requirements not only of the army but also of the labour services. As it happens, mortality statistics deal only with the civilian population. The officially published figures show no decrease in the number of the civilian population, a decrease which must nevertheless have occurred. Statistics for the large cities are further vitiated by the decrease in population which, since the end of 1941, must have taken place in a growing number of such cities as a result of bombardments and evacuations. It is difficult to take at their face value the official figures given for the population of the large towns, which show a decrease of but 3% in 1943 as compared to 1939 ! Figures relating to individual towns only show reductions in population greatly inferior to those which might have been expected from official German declarations on the ravages of air raids and with a considerable lag after these raids.

Birth rates and general mortality rates given in the accompanying tables have been taken from official German sources. They would appear to be definitely below the actual facts when it is realised that the populations on which they are based have not been reduced sufficiently to account for mobilisation and, furthermore, that the rates do not include deaths in the forces, or civilian deaths due to enemy action, or those of foreign workers prior to 1942.

As they officially stand, and without taking into account the above reservations, it can be seen that, during 1939 and 1940, there was a large rise in *births* and in the natural increase in the population, *general mortality*, from 1939 to 1942, only slightly exceeding the pre-war median (*cf.* graph, page 581.)

The excess of births would appear to have been rather higher in the large towns than in the rest of the country. Infant mortality, which does not suffer from the same sources of error as birth and general mortality rates because it does not take population figures into account, remained low up to the middle of 1942. Since then, a tendency for it to rise has been observed. Mortality in the large towns, however, has remained low, up to and including 1943, approaching even the minimum recorded in the years preceding the war. General mortality rates officially recorded from 1929 up to 1943 exceed, almost consistently, the upper quartile of the cor-

responding series before the war, in large towns as well as in the whole of the Reich. It should be noted, however, that these rates were—and still are—low, given the unfavourable age composition of the present civilian German population.

Mortality from *tuberculosis*, which was clearly decreasing in large towns, particularly up to 1939, shows a slight increase since then, culminating in 1942. Even the rate for that year, however, is below the median rate observed during the 1929-1938 period. The intensification of industrial work would amply account for this slight rise in mortality from tuberculosis, without there being any need to adduce a change in the nutritional condition of the population. It should be noted that the German medical Press reports no change in the health situation from this cause, thus contrasting with that of most occupied countries. Some investigations carried out in 1941 with regard to the nutritional condition, height and weight of school-children in large towns (such as Leipzig) even demonstrated that at the time the physical condition of these children was better than ever before.[1] In order to prevent possible deficiencies, a systematic distribution of vitamins, both to school-children and to newly-born babies, was maintained from the beginning of the war.

For most *contagious diseases*, an increase in the number of cases notified began from the time of the pre-war mobilisation and has definitely expanded since the outbreak of hostilities. This is particularly the case as regards scarlet fever and diphtheria, for which record figures were notified in 1942 and 1943. The increase in the number of cases of meningitis preceded the war, and after 1940 an improvement as regards the epidemic outbreak was observed. Typhoid and paratyphoid fevers showed an appreciable rise from 1942. Dysentery, already abnormally high before the war, remained high during the war. Though the German Army on the eastern front has been severely affected by the serious forms of bacillary dysentery, it would appear that, among the German civilian population, there has been an increase only in the very mild forms due to the Sonne bacillus.

It would also appear that the increase in the endemic of common infectious diseases in Germany is essentially due to the mingling

[1] Koch, E. W. (1942) : " Längen und Gewichte der Leipziger Kinder im Kriegsjahr 1941 " ; *Öffentl. Gesundheitsdienst*, **7**, H. 2, 609-618.

of populations and the extension of communal living, not only among adults, but also among adolescents and children, consequent on the industrial and military mobilisation. In the centres of war industry, the arrival of rural elements, either from Germany itself, but even more from the countries of Eastern Europe, where the population is for the most part susceptible to common infectious diseases, is the principal cause of the increase in contagious diseases which has been recorded. The arrival from Eastern Europe of prisoners-of-war and of deported civilian workers has also been responsible, to a large extent, for the dissemination of typhus fever among the civilian German population since 1941.

Some pseudo-epidemics occurred in the neighbourhood of camps of Russian prisoners, Polish workers, etc., and among the hospital personnel called upon to deal with them or with wounded Germans returning from the Russian front. German troops returning from the east of Europe, passing through Germany on their way to rest quarters in the occupied territories of the west and north, in some cases brought the exanthematic virus with them and occasioned sporadic infections. In Germany and in Western Europe the disease has not spread, the population on the whole containing few carriers of body lice. The same factor prevented the spread of trench fever, an infection caused by *Rickettsia quintana*, from which troops on the eastern front suffered as well as from typhus fever.

From the figures and indications available, it appears that, on the whole, the health situation in Germany is favourable; that though extensive movements of the population have provoked an increase in common contagious diseases, this is a temporary phenomenon, unaccompanied by any serious increase in fatality from those diseases.

These remarks apply only to the German population itself, and not to the mass of some 12 million foreigners, prisoners and civilian workers at present in Germany, for whom living conditions, from the health point of view—housing, nutrition, working conditions and medical care—are very inferior to those of the German population. This foreign population, moreover, does not form the subject of any published health statistics. Information concerning them (clandestine correspondence, accounts given by escaped and repatriated persons and their condition, deaths reported to the Red Cross) shows that their health situation is definitely bad.

GERMANY (Altreich, 1937 territory).

Austria annexed : March 12th, 1938 ; Sudetenland annexed : October 1st, 1938. Bohemia and Moravia occupied : March 1939 ; Poland invaded : September 1st, 1939. Denmark and Norway occupied : April 9th, 1940 ; Belgium, Luxemburg and the Netherlands invaded : May 10th, 1940 ; France invaded : May 17th, 1940. Yugoslavia and Greece invaded : April 6th, 1941 ; U.S.S.R. invaded : June 22nd, 1941. Bombardments of large German towns, increasing in number and gravity, by the Allied air forces, beginning at the end of 1941. Number of foreign workers at the beginning of 1943 (including *employed* prisoners of war) : 6,500,000 *(a)* (constantly increasing).

Civilian **Population** used for calculating the rates.

GERMANY *(g)*

C.	VI.1933 *(b)* :	65,218,000	O. N. E.	VI.1940 *(g) (h)* :	73,134,000
E. S.	VI.1938 *(e)* :	68,340,000	O. N. E.	VI.1941 *(g) (h)* :	73,134,000
O. N. E.	V. 1939 *(f)(g)* :	69,460,000	E. S.	VI.1942 *(g) (p)* :	73,000,000

TOWNS OF MORE THAN 100,000 INHABITANTS

O. N. E.	VI.1938 :	21,178,000	O. N. E.	VI.1941 :	21,886,000
O. N. E.	VI.1939 :	21,295,000	O. N. E.	VI.1942 :	22,175,000
O. N. E.	VI.1940 :	21,873,000	O. N. E.	VI.1943:	21,461,000 *(k)*

Natural Movement of Population.

Year	GERMANY Births ‰ inhab.	General mortality ‰ inhab.	Increase (+) or decrease (−) ‰ inhab.	TOWNS OF MORE THAN 100,000 INHABITANTS *(k)* Births ‰ inhab.	General mortality ‰ inhab.	Increase (+) or decrease (−) ‰ inhab.
1928-1938 Max.	*19.6*	*12.6*	*+8.0*	*16.1*	*11.5*	*+4.7*
1928-1938 Med.	*18.0*	*11.6*	*+7.0*	*13.6*	*10.6*	*+3.0*
1928-1938 Min.	*14.7*	*10.8*	*+3.5*	*10.8*	*10.0*	*+0.8*
1937	18.8	11.7	+7.1	**15.4**	11.3	+4.1
1938	**19.6**	11.6	+8.0	**16.1**	**11.4**	+4.7
1939 *(g)*	**20.3**	**12.3**	+8.0	**17.0**	**12.1**	+4.9
1940 *(g)*	**20.0**	**12.7**	+7.3	**17.3**	**12.4**	+4.9
1941 *(g)*	18.6	**12.0**	+6.6	**16.8**	**11.9**	+4.9
1942 *(g)*	14.9	**12.0**	+2.9	13.9	**11.7**	+2.2
1942 *(g)* (I.I-30.IX)	15.3	12.1	+3.2			
1943 *(g) (i) (id.)*	16.3	11.9	+4.4	12.5 *(k)*	10.8 *(k)*	+1.7

C. = Census.
E. S. = Estimate of the Epidemiological Intelligence Service of the L.o.N.
O. N. E. = Official national estimates *(Reichsgesundheitsblatt)*.

(a) *Cf.* E. M. Kulischer : *The Displacement of Population in Europe*, I.L.O., Montreal, 1943, page 160.

(b) Excluding the Saar Territory (828,000 inhabitants. E. XII.1933).

(e) 1933 and Saar Territory (842,000 inhabitants. E. VI.1939).

(f) Including Memel (142,000 inhabitants). Source : *Wirtschaft und Statistik*, 1940, page 519.

(g) From 1939 inclusive, population figures for Germany have been taken as a basis for calculating tuberculosis mortality rates only. Birth, infant mortality, and general mortality rates have been taken from *Wirtschaft und Statistik*. Owing to the fact that general mortality rates do not include deaths in the Services nor those of civilians due to enemy action, there was a fall in these rates. According to *Wirtschaft und Statistik*, this fall is probably offset by the inclusion (since 1942) in the total deaths, of deaths of foreign workers. This compensation, however, is probably insufficient, and the rates should be increased by 0.1, 0.2, 0.2 and 0.3‰ for the years 1940 to 1943 inclusive. Nevertheless, general mortality rates would appear, even thus corrected, to be lower than the real ones.

(h) According to the *Reichsgesundheitsblatt, Statistische Sonderbeilage zur Nr. 46 vom 17. November, 1943.*

For footnotes (i) to (s), see page 637.

Infant Mortality (deaths of infants under 1 year of age, per 1,000 live births).

GERMANY

	Yearly rates	I J	II F	III M	IV A	V M	VI J	VII —	VIII J	IX A	X S	XI O	XII N	XIII D
1932-1938(o) *Max.*	*97*		*91*			*79*				*76*			*76*	
Med.	*76*		*77*			*69*				*59*			*65*	
Min.	*60*		*62*			*62*				*56*			*57*	
1937	64		77			64				56			57	
1938	60		62			62				59			57	
1939	60		68			61				55			57	
1940 *(j)* ...	63		74			65				56			59	
1941	63		75			63				55			59	
1942*(i)(n)* .	70		68			68				63			73	
1943*(i)(n)* .			73			65				**70**				

TOWNS OF MORE THAN 100,000 INHABITANTS *(m)*

1928-1938 *Max.*	*92*	*108*	*116*	*120*	*99*	*91*	*96*	*76*	*78*	*85*	*87*	*89*	*88*	*93*
Med.	*71*	*78*	*82*	*83*	*77*	*78*	*65*	*65*	*58*	*62*	*57*	*65*	*70*	*77*
Min.	*58*	*61*	*58*	*65*	*74*	*61*	*56*	*54*	*53*	*50*	*49*	*53*	*53*	*61*
1937	61	71	78	71	67	61	58	54	54	55	51	53	55	61
1938	58	61	58	65	62	61	62	55	56	57	49	56	53	65
1939	60	71	64	64	62	58	58	58	53	58	53	57	57	61
1940	62	68	72	73	60	59	64	64	54	58	51	59	62	67
1941	59	74	72	74	68	51	53	56	53	52	46	52	59	66
1942 *(n)* ...	66	70	77	79	70	63	59	56	53	62	60	69	69	72
1943 *(k)(n)*	62	67	57	59	60	60	55	52	63	63	54	67	68	86

General Mortality (rates per 1,000 inhabitants on a yearly basis).

GERMANY

	Yearly rates	I J	II F	III M	IV A	V M	VI J	VII —	VIII J	IX A	X S	XI O	XII N	XIII D
1930-1938(o) *Max.*	*12.6*		*14.0*			*12,2*				*10.8*			*13.4*	
Med.	*11.6*		*12.4*			*11,5*				*9.9*			*11.0*	
Min.	*10.8*		*11.8*			*10,7*				*9.4*			*10.4*	
1937	11.7		**14,0**			11.7				10.1			**11.5**	
1938	11.6		12,4			**12.2**				**10.8**			**11.9**	
1939 *(g)* ...	**12.3**		**14,4**			**12.4**				**10.4**			**12.1**	
1940 *(g)* ...	**12.7**		**15,9**			**13.2**				**10.3**			**11.5**	
1941 *(g)* ...	**12.0**		**14,6**			**12.0**				**10.2**			11.3	
1942 *(g)* ...	**12.6**		13,6			**12.0**				**10.6**			**11.7**	
1943 *(g)(i)*			12,9			**11.9**				**10.8**				

TOWNS OF MORE THAN 100,000 INHABITANTS *(m)*

1928-1938 *Max.*	*11.5*	*14.7*	*18.4*	*16.0*	*12.2*	*12.7*	*11.2*	*10.5*	*10.5*	*9.6*	*10.3*	*11.0*	*11.6*	*15.1*
Med.	*10.6*	*12.4*	*12.0*	*12.1*	*11.8*	*10.9*	*10.5*	*9.5*	*9.2*	*9.1*	*9.2*	*9.8*	*10.6*	*11.4*
Min.	*10.0*	*10.7*	*11.1*	*11.3*	*10.7*	*10.2*	*9.5*	*8.9*	*8.6*	*8.3*	*8.7*	*9.2*	*9.6*	*10.3*
1937	11.3	**14.0**	**13.6**	**12.4**	**12.0**	11.3	10.8	9.8	**9.6**	9.1	**10.1**	**10.7**	**11.6**	**12.0**
1938	**11.4**	**12.9**	11.9	**12.7**	**12.0**	**12.7**	**11.2**	**10.2**	**10.5**	**9.6**	**10.3**	**10.6**	**11.2**	**13.0**
1939 *(n)* ...	**12.1**	**14.1**	**15.5**	**14.3**	**12.8**	**12.2**	**11.9**	**10.6**	**9.9**	**10.3**	**10.1**	**11.4**	**11.6**	**12.7**
1940 *(n)* ...	**12.4**	**16.6**	**16.5**	**15.2**	**14.2**	**12.9**	**11.7**	**10.5**	**9.7**	**9.9**	**10.4**	**10.8**	**11.2**	**12.0**
1941 *(n)* ...	**11.9**	**14.1**	**15.5**	**13.6**	**12.4**	**11.7**	**11.1**	**10.7**	**9.6**	**9.4**	**10.1**	10.3	**11.6**	**12.0**
1942 *(n)* ...	**11.7**	**13.7**	**14.5**	**13.7**	**12.7**	**12.1**	**11.3**	**10.5**	**10.3**	**10.3**	**10.6**	**10.7**	11.0	11.6
1943 *(k)(n)*	10.8	**13.1**	12.0	**13.8**	**13.1**	**12.0**	10.1	9.5	**9.6**	8.4	8.4	9.4	10.1	11.9

Tuberculosis Mortality (rates per 100,000 inhabitants on a yearly basis).

GERMANY *(g)*

Deaths	1928-1938			1937	1938	1939	1940	1941	1942 *(p)*
	Max.	*Med.*	*Min.*						
(a) Recorded by the Registrar	*88*	*73*	*62*	69	62	...	...	...	...
(b) Notified to the sanitary authorities *(q)*	...	...	...	56	50	50	57	60	62

TOWNS OF MORE THAN 100,000 INHABITANTS *(m)*

	Yearly rates	I J	II F	III M	IV A	V M	VI J	VII —	VIII J	IX A	X S	XI O	XII N	XIII D
1928-1938 *Max.*	*92*		*107*			*99*				*81*			*82*	
1928-1938 *Med.*	*77*		*80*			*79*				*69*			*71*	
1928-1938 *Min.*	*63*		*67*			*68*				*56*			*59*	
1937	69		78			77				61			62	
1938	63		67			68				56			59	
1939 *(n)*....	61		69			65				51			58	
1940 *(n)*....	68		76			74				58			63	
1941 *(n)*....	72		80			78				66			64	
1942 *(n)*....	76		82			85				67			70	
1943 *(k) (n)*	70		85			81				57			56	
1944 *(k) (n)*			83											

Communicable Diseases : Cases notified in GERMANY (Altreich).

	M dian 1928-1938	1937	1938	1939	1940	1941	1942	1943
Typhoid fever	*3,051(s)*	3,051	2,941	2,679	3,036	2,655	**4,885**	**6,861**
Paratyphoid fevers.	*2,725(s)*	**3,755**	3,211	2,434	2,930	**3,735**	**5,102**	**3,666**
Dysentery	*3,472*	**7,545**	**5,191**	**6,135**	**12,705**	**8,734**	**12,442**	**5,183**
Diphtheria	*76,877*	**145,446**	**149,490**	143,585	138,397	**173,161**	**237,037**	**238,409**
Scarlet fever	*111,545*	**117,480**	114,219	**129,794**	**136,567**	**251,969**	**325,206**	**248,678**
Cer.-sp. meningitis .	*962*	**1,532**	**1,816**	**4,903**	**5,413**	**3,248**	**1,877**	**1,761**
Poliomyelitis	*1,710*	**2,737**	**5,717**	**3,740**	1,534	**4,076**	**3,411**	2,450
Typhus fever	*1*	—	—	—	**6**	**395**	**2,400**	**1,668***(r)*
Smallpox	—	—	—	—	—	—	1	1
Notified cases	*200,343*	281,546	282,585	293,270	300,588	447,973	592,361	508,677
Composite epidemic index	100	140.5	141.1	146.4	150.0	223.6	295.7	253.9

For footnotes (a) to (h), see page 635.

(i) For 1943 (and 1942 for infant mortality rates) *Wirtschaft und Statistik* gives rates only for a *Great Reich*, including Austria, Sudetenland, Memel, Danzig, but not incorporated Polish territories and Eupen-Malmédy.

(j) From May 1940, rates referring to Germany are calculated on the basis of the number of births within a 12-months period.

(k) As from 1943, official statistics *(Reichsgesundheitsblatt)* take into account evacuations of the civilian population of towns, consequent on air bombardments, but, it would appear, to an insufficient extent and with a time-lag of several months. This being so, the figure given for population should be reduced by at least 10 % ; birth, general mortality, and tuberculosis mortality rates should, consequently, be increased by at least 10 %. Moreover, the figures given concern the resident population only ; the figure for inhabitants not domiciled (including particularly foreign workers) is equal to 10-15 % of the figure for the German resident population.

(m) Towns of the 1937 territory. For the years 1939 to 1943, figures for deaths and for population are taken from the *Reichsgesundheitsblatt*.

(n) Excluding deaths of persons in the Services and those due to enemy action.

(o) Annual rates taken in the period 1928-1938.

(p) 1942 population estimated at approximately 73,000,000. For the years 1940 to 1942, population figures corresponding to the rates given by *Wirtschaft und Statistik* would be respectively 69,900,000, 70,300,000, and 70,800,000.

(q) Rates based on deaths notified to the sanitary authorities are about 20 °/₀₀ less than those based on statistics of causes of death recorded by the Registrar ; moreover, for the reasons given in footnote *(g)* above, a corrective increase should be applied to tuberculosis mortality rates from 1940 on.

(r) 3.1-27.VI.1943.

(s) Median 1932-1938.

Austria

The *birth rate* had been particularly low in Austria during the years preceding the annexation of the country by Germany in 1938.

After the "Anschluss", the birth rate rose sharply, reaching in 1940 a rate of 21.9 $^{0}/_{00}$, which was higher than had been recorded in any year since 1924.

In 1942, the rate fell once more to 17.2 (the 1928 level). In Vienna, where the birth rate had been the lowest in the world in 1937, there was a still sharper relative rise, the rate having almost tripled by 1940, though later it decreased somewhat.

General mortality also rose between 1938 and 1940, and subsequently remained high ; this rise was due, in the first place, to the increase in the number of infants, amongst whom the death rate is normally high and, secondly, to the drafting away from the civilian population, as a result of mobilisation, of the young men, among whom the death rate is low. It is impossible, with regard to the increase in the death rate, to arrive at any numerical estimate of the effects of discriminatory administrative measures taken in respect of certain categories of citizens—Jews, the inmates of asylums, etc.

Before the "Anschluss", not counting migrations, the population of Vienna was very noticeably decreasing : in the country as a whole the decrease was very slight. Since then, the population has been more or less stationary in Vienna and has increased in Austria as a whole, so far, at all events, as can be judged by comparing the birth rate and the general *civil* death rate.

The absence of data as to the death rate among Austrians mobilised in the Wehrmacht and in auxiliary military organisations renders it impossible to ascertain whether the population has in fact decreased or increased during the war.

The *infant mortality* rate has varied but little during the war-years in Austria as a whole or in Vienna ; in the latter, however, it rose in 1942 and 1943, though remaining below the average pre-war level.

In Vienna as in Germany, the increased demands on workers of all kinds may have influenced mortality, especially that caused by *tuberculosis*. In spite of some irregularities from year to year, the rate for the period 1940-1943 averaged 130 per 100,000, as compared with 107 per 100,000 from 1937 to 1939—an increase of 22%.

Figures are available for *communicable diseases* for the whole of Austria throughout the war period. As compared with the

pre-war period 1928-1938, they show a slight increase in the prevalence of diphtheria, a more significant increase in scarlet fever and a marked rise in cerebrospinal meningitis at the time of the mobilisation in 1939, which slowly subsided in the course of the following years. As a result of the campaign on the eastern front, sporadic secondary cases of typhus fever occurred in all parts of the country, particularly in Vienna, as from the end of 1941. No true secondary epidemic has apparently taken place.

There have been no reports of food shortages, or of malnutrition, and the observed death rates do not suggest that there was any.

Both the nutritional and sanitary situation would therefore appear to be normal.

AUSTRIA

Annexed by Germany : March 12th, 1938.

Civilian **Population** used in calculating the rates.

AUSTRIA		VIENNA	
C. V.1939 :	6,650,306 6,972,269 *(a)*	C. VI.1934 :	1,874,000
S. E. VI.1940 :	6,700,000 7,030,000 *(a)*	O. N. E. VI.1939 :	2,087,000
S. E. VI.1941 :	7,080,000 *(a)*	O. N. E. VI.1940 :	1,924,000
S. E. VI.1942 :	7,110,000 *(a)*	O. N. E. VI.1941 :	1,927,000
S. E. VI.1943 :	7,100,000 *(a)*	O. N. E. VI.1942 :	1,927,000
		O. N. E. VI.1943 :	1,874,000

Natural Movement of Population.

Year	AUSTRIA Births °/₀₀ inhab.	General mortality *(f)* °/₀₀ inhab.	Increase (+) or decrease (−) °/₀₀ inhab.	VIENNA Births °/₀₀ inhab.	General mortality °/₀₀ inhab.	Increase (+) or decrease (−) °/₀₀ inhab.
1928-1938 *Max.*	*17.6*	*14.6*	*+3.3*	*10.7*	*14.4*	*−3.1*
Med.	*14.3*	*13.6*	*+1.1*	*6.7*	*13.0*	*−6.0*
Min.	*12.9*	*12.7*	*−0.5*	*5.4*	*12.0*	*−7.6*
1937.....	12.9	13.4	−0.5	5.4	13.0	−7.6
1938.....	14.1	14.3	−0.2	6.7	13.8	−7.1
1939.....	20.9	15.3	+5.6	13.8	14.7	−0.9
1940.....	21.8 21.9 *(a)*	15.0 14.9 *(a)*	+6.8 +7.0 *(a)*	15.3	15.9	−0.6
1941.....	20.2 *(a)*	14.0 *(a)*	+6.2 *(a)*	15.2	14.9	+0.3
1942.....	17.2 *(a)*	13.3 *(a)*	+3.9 *(a)*	13.2	14.7	−1.5
1943.....	18.0 *(a)*	13.8 *(a)*	+4.2 *(a)*	14.8	14.5	+0.3

C. = Census.
O. N. E. = Official national estimate.
S. E. = Estimate of the Epidemiological Intelligence Service of the L.o.N. on the basis of actual figures and official rates.

For footnotes, see page 640.

Infant Mortality (deaths of infants under 1 year of age, per 1,000 live births).

VIENNA

	Yearly rates	I J	II F	III M	IV A	V M	VI J	VII —	VIII J	IX A	X S	XI O	XII N	XIII D
1928-1938 Max.	*88*	*110*	*115*	*122*	*114*	*84*	*83*	M	*82*	*82*	*79*	*70*	*93*	*102*
1928-1938 Med.	*71*	*76*	*77*	*81*	*80*	*73*	*60*	M	*62*	*64*	*64*	*59*	*66*	*69*
1928-1938 Min.	*51*	*44*	*57*	*53*	*59*	*47*	*46*	M	*40*	*45*	*40*	*38*	*55*	*58*
1937	71	76	58	53	**89**	76	**83**	M	61	51	48	38	**71**	69
1938	51	44	57	57	67	47	46	M	40	50	40	51	55	58
1939	44	59	54	46	51	48	43	48	36	38	32	39	45	39
1940	54	50	44	58	66	67	60	51	46	46	50	52	53	62
1941	53	74	77	68	74	48	47	43	46	41	40	43	40	57
1942	63	54	71	59	**89**	**83**	68	60	63	55	41	59	48	77
1943	62	83	82	63	67	66	64	54	62	51	46	50	60	63

Infant Mortality (deaths of infants under 1 year of age, per 1,000 live births).

AUSTRIA

	Yearly rates	Quarterly I	II	III	IV
1928-1938 Max.	*118*	...	...	...	...
1928-1938 Med.	*99*	...	...	...	...
1928-1938 Min.	*83*	...	...	...	...
1937	92	85	89	77	80
1938	83	88	86	68	74
1939	69	101	78	57	65
1940	72	...	...	...	...
1940 *(a)*....	73	71	86	63	69
1941 *(a)*....	69	92	71	54	63
1942 *(a)*....	72	68	79	66	76
1943 *(a)*....	78	89	73	70	80

General Mortality (rates per 1,000 inhabitants on a yearly basis). *(f)*

AUSTRIA

	Yearly rates	Quarterly I	II	III	IV
1928-1938 Max.	*14.6*	...	...	...	...
1928-1938 Med.	*13.6*	...	...	...	...
1928-1938 Min.	*12.7*	...	...	...	...
1937	13.4	15.6	13.3	11.4	13.2
1938	**14.3**	15.7	15.1	12.1	14.3
1939	**15.3**	19.2	14.7	12.3	15.0
1940	**15.0**	19.0	15.2	12.0	13.6
1940 *(a)*....	**14.9**	19.0	15.2	12.0	13.5
1941 *(a)*....	14.0	17.8	13.6	11.4	13.2
1942 *(a)*....	13.3	15.4	13.4	11.3	13.0
1943 *(a)*....	13.8	15.3	13.6	11.7	14.4

General Mortality (rates per 1,000 inhabitants on a yearly basis). *(f)*

VIENNA

	Yearly rates	I J	II F	III M	IV A	V M	VI J	VII —	VIII J	IX A	X S	XI O	XII N	XIII D
1928-1938 Max.	*14.4*	*18.2*	*22.7*	*18.8*	*15.8*	*14.4*	*13.4*	M	*12.4*	*11.9*	*12.1*	*13.8*	*14.6*	*16.1*
1928-1938 Med.	*13.0*	*14.8*	*15.2*	*15.2*	*13.9*	*13.3*	*12.2*	M	*10.8*	*10.6*	*10.9*	*12.7*	*12.8*	*14.2*
1928-1938 Min.	*12.0*	*13.9*	*13.5*	*12.7*	*12.6*	*11.6*	*11.1*	M	*10.3*	*9.4*	*9.9*	*11.4*	*12.0*	*12.2*
1937	13.0	15.6	**17.0**	14.7	13.4	12.8	11.4	M	10.7	10.1	11.3	12.4	**13.5**	14.2
1938	**13.8**	15.8	14.8	16.1	15.0	**13.9**	**12.6**	M	11.2	**11.1**	**11.8**	**13.1**	**14.6**	**16.1**
1939	**14.7**	**19.8**	**20.3**	15.8	15.2	**15.6**	**14.1**	**13.0**	11.8	**12.1**	**11.8**	**13.2**	**14.1**	14.5
1940	**15.9**	**18.2**	**20.4**	**22.5**	**20.4**	**17.4**	**14.4**	**14.0**	**12.3**	**12.3**	**12.2**	**13.2**	**14.3**	**15.0**
1941	**14.9**	**17.0**	**21.1**	17.0	15.2	**14.4**	**13.8**	**12.8**	11.8	**11.6**	**11.8**	**13.3**	**14.5**	**15.9**
1942	**14.7**	15.8	**19.5**	17.1	**17.3**	**16.5**	**14.7**	**13.5**	**12.8**	**11.7**	**11.7**	12.9	13.1	**15.0**
1943	**14.5**	**16.1**	16.7	16.4	**17.2**	**15.5**	**14.5**	**12.6**	**12.6**	**11.3**	**11.5**	**13.7**	**15.0**	**15.1**

(a) " Reichsgaue der Ostmark " (since 1942 called " Alpen- und Donau-Reichsgaue ") — *i.e.* Austrian territory plus a few Sudeten districts incorporated in the Reichsgaue Niederdonau and Oberdonau.

(b) 25 weeks.

(c) Median based on the period 1932-1938.

(f) As from September 1st, 1939, excluding military deaths (Wehrmacht).

Tuberculosis Mortality (rates per 1,000 inhabitants on a yearly basis).

VIENNA

	Yearly rates	I J	II F	III M	IV A	V M	VI J	VII —	VIII J	IX A	X S	XI O	XII N	XIII D
1928-1938 *Max.*	*188*		*213*			*219*		M		*159*			*161*	
Med.	*142*		*155*			*163*		M		*124*			*128*	
Min.	*102*		*105*			*111*		M		*86*			*88*	
1937	107		126			112		M		96			95	
1938	102		105			111		M		90			102	
1939	113		121			124		M		96			112	
1940	132		146			153		M		116			115	
1941	118		125			128		M		104			115	
1942	133		139			150		M		123			121	
1943	136		153			155		M		122			115	
1944			161											

Communicable Diseases: Cases notified in AUSTRIA.

	M dian 1928-1938	1937	1938	1939	1940	1941	1942	1943
Typhoid fever *(e)*	*748*	660	748	**1,071**	769	729	846	**1,121**
Paratyphoid fevers *(e)*	*724*	836	**870**	755	**1,067**	675	814	**1,123**
Dysentery	*106*	93	72	92	**164**	**259**	**543**	**570**
Diphtheria	*19,494*	19,494	16,800	19,137	15,910	14,255	15,534	**22,444**
Scarlet fever	*10,243*	**12,048**	**12,659**	**14,756**	7,872	**12,933**	**19,222**	**28,878**
Cer.-sp. meningitis	*42*	42	**51**	**1,021**	**654**	**346**	**238**	**234**
Poliomyelitis	*205*	**805**	501	270	234	**869**	265	195
Typhus fever	—	—	—	—	—	18	106	37 *(b)*
Smallpox	—	—	—	—	—	—	—	—
Notified cases	*31,562*	33,978	31,701	37,102	26,670	30,084	37,568	54,602
Composite epidemic index	100	107.7	100.4	117.6	84.5	95.3	119.0	173.0

CZECHO-SLOVAKIA

Since the Munich agreements at the end of September 1938, which transferred the Sudetenland to Germany, and the invasion and seizure of Bohemia and Moravia on March 18th, 1939, the declaration of independence by Slovakia on March 23rd, the seizure by Hungary of Southern Slovakia and of Sub-Carpathian Ruthenia (Subcarpathia), statistical data for Czecho-Slovakia as such are no longer available, and statistics relate only to certain component parts of that country.

From the health, as well as from other points of view, there were marked differences in these parts. Bohemia and Moravia, as well as Czech Silesia, being more urbanised and industrialised, had a lower mortality from most causes and also a lower birth rate. Slovakia, being more agricultural, had higher birth and death

rates. Subcarpathia, a more isolated and backward rural mountainous area, had still higher birth and death rates. It was, moreover, the only part of Czecho-Slovakia where typhus fever was endemic.

As a " Protectorate ", Bohemia and Moravia had to participate in the German war effort, both in the military and economic fields. The civilian population covered by the statistics lost a large number of able-bodied young men with normally low mortality, a fact which may explain the slight rise in *general mortality* in 1940 and 1941, and again in 1942 and 1943. A rise of 10% in the number of deaths recorded at Prague also took place between 1939 and 1942.

Infant mortality, which in " Bohemia and Moravia " was slightly declining, remained stationary in 1939 and 1940 and increased in 1941 and 1942.

Tuberculosis mortality rose by 25% between 1939 and 1942 in the " Protectorate ", and by 37% in Prague.

In the " Protectorate " a gradual rise took place between 1939 and 1942 in mortality, not only from tuberculosis, but also from diseases of childhood (from 53 to 64 per 100,000) and from diarrhœa and enteritis (28 to 34 per 100,000) ; deaths from all communicable diseases also increased (56 to 64 per 100,000). A rise of 50% in the number of deaths from diphtheria in 1942 partly accounts for this increase.

The *birth rate*, which had fallen sharply during the critical pre-war years, has gradually risen from 14.3‰ in 1938 to 20.8‰ in 1943. This would mean a definite rise in the natural increase were it not for military losses, which cannot be calculated.

In Slovakia there has been a slight rise in general mortality from 1938 to 1942. The birth rate has also risen slightly, so that the apparent natural increase has undergone no change.

Separate vital statistics for the Sudetenland are not available since 1940.

While the available figures do not permit us to assess the part played by shortage of food in the changes in mortality observed in Bohemia and Moravia, particularly in Prague, information concerning food rationing in these areas indicates that, in the towns, the amounts of food available in the rations, and of high-class protein particularly, are insufficient, and it is not surprising that a steady fall in the weight of adults in these towns should have taken place. The situation is evidently more favourable in the food-growing countryside.

CZECHO-SLOVAKIA

Loss of Sudetenland, ceded to Germany by the Munich agreement (September 30th, 1938); Tešin (Cieszyn, Teschen), taken by Poland.

Bohemia and Moravia invaded and a German protectorate declared (March 18th, 1939). Slovakia secedes and declares itself independent (March 23rd, 1939).

Subcarpathian Ruthenia (Subcarpathia) and a southern strip of Slovakia (Northern Territory) annexed by Hungary (November 1938, April 4th, 1939).

Population.

Czecho-Slovakia	E.	VIII.1938:	15,263,000
"Sudetenland"	C.	V.1939:	3,408,000
"Bohemia and Moravia"	E.	XII.1940:	7,485,000
Tešin (Teschen)	E.	XII.1938:	241,700
Slovakia	C.	XII.1940:	2,655,000
Southern Slovakia (Hungarian "Northern Territory")	E.	XII.1941:	1,026,000
Subcarpathian Ruthenia (Hungarian "Subcarpathia")	C.	XII.1930:	725,000

	1931-35	1936	1937	1938	1939	1940	1941	1942	1943
Births:									
Czecho-Slovakia	19.6	17.4	17.2	16.8(r)	.	.	.	.	.
"Bohemia and Moravia"	16.7(r)	14.6	14.4	14.3	14.7	16.7	17.2	18.3	20.8
"Slovakia"	25.6	23.6	23.3	22.8(r)	.	24.2	24.1	24.3	...
"Sudetenland"	16.4(r)	14.4	14.2	15.7(r)	22.0	24.0	.	.	.
Natural Movement:									
Czecho-Slovakia	+5.8	+4.1	+3.9	+4.0(r)	.	.	.	.	.
"Bohemia and Moravia"	+3.7	+1.8	+1.7	+1.8	+1.7	+3.3	+3.8	+4.4	+7.1
"Slovakia"	+10.5	+9.3	+9.0	+9.0	.	+9.6	+9.4	+9.1	...
"Sudetenland"	+3.1	+1.4	+1.0	+1.5	+8.1	+9.7	.	.	.
General Mortality:									
Czecho-Slovakia	13.8	13.3	13.3	12.8(r)	.	.	.	.	.
"Bohemia and Moravia"	13.0(r)	12.8	12,7	12.5	13.0	13.4	13.4	13.9	13.7
Prague	.	11.2	11.2	...	11.4	11.8	12.0	12.5	...
"Slovakia"	15.1	14.3	14.3	13.8(r)	.	14.6	14.7	15.2	...
"Sudetenland"	13.3(r)	13.0	13.2	14.2	13.9	14.3	.	.	.
Infant Mortality:									
Czecho-Slovakia	130	124	122	121(r)	.	.	.	.	.
"Bohemia and Moravia"	.	.	.	.	95	94	99	98	...
"Slovakia"	.	.	.	.	.	156	135	...	...
Tuberculosis Mortality:									
Czecho-Slovakia	146	129	124	.	.	.	.	.	.
"Bohemia and Moravia"	137(p q)	122(p q)	...	123	124	135	141	156	...
Prague	138	126	129	122	123	132	158	169	...
"Slovakia"	159 (q)	135 (q)	...	...	...	...	...	...	...

C. = Census. E. = Estimate.

(a) 8 months for "Bohemia and Moravia".
(b) 28 weeks for "Slovakia".
(e) 8 months for Bohemia and 10 months for Moravia.
(f) 3 whole months and 14 weeks spread over the year for "Slovakia".
(g) 8 months for "Bohemia and Moravia", 11 months for "Slovakia".
(h) 11 months for "Slovakia".
(i) 45 weeks for "Slovakia".
(j) 31 weeks for "Slovakia".
(k) 29 weeks for "Slovakia".
(l) 26 weeks for "Slovakia".
(m) 9 months for Bohemia and 10 months for Moravia.
(n) 31 weeks for "Slovakia".
(o) Excluding "Bohemia and Moravia".
(p) Bohemia, Moravia and Silesia.
(q) Pre-1938 boundaries.
(r) Approximately.

The *epidemic situation* has been characterised by an unusually high incidence of both diphtheria and scarlet fever. The increase has been rapid from 1940 onwards. It has been greatest in the Sudeten-land, somewhat less in Bohemia and still less in Moravia, but negligible in both Slovakia and Subcarpathia. Cerebrospinal meningitis showed a marked rise in 1939, particularly in Slovakia. It has been steadily decreasing since. Poliomyelitis, which used to be comparatively rare, also became epidemic in 1939, chiefly in Bohemia. Typhus fever endemicity in Subcarpathia has increased markedly since 1940. In Slovakia, where typhus fever was exceptional, sporadic cases occurred in 1941 and 1942 and there was an epidemic of some 700 cases in 1943. Figures for Bohemia and Moravia, both of which used to be free from the disease, are not available.

The epidemic trend in Czecho-Slovakia has followed closely that of Germany, which is not surprising in view of the increased communications and chances of contact between the populations of the two countries.

Communicable Diseases : Cases notified in CZECHO-SLOVAKIA.

	Median 1928-1938	1937	1938	1939	1940	1941	1942	1943
Typhoid fever	*6,338*	**7,461**	5,603	3,766 (*a*)	4,434	4,345	4,477(*b*)	5,549
Paratyphoid fevers .	*217*	217	**238**	221 (*e*)	**463**	**565**	**420** (*f*)	**540**
Dysentery	*678*	**3,059**	639	30 (*g*)	**2,426** (*h*)	317 (*i*)	973 (*j*)	**1,565**
Diphtheria	*26,141*	**31,487**	25,304	22,358 (*a*)	25,541	31,050	**48,959**(*k*)	**53,678**
Scarlet fever	*23,188*	23,975	21,057	19,137 (*a*)	20,516	**37,343**	**55,703**(*b*)	**59,818**
Cer.-sp. meningitis .	*157*	**206**	**487**	**1,371** (*a*)	**1,403**	**772**	**326** (*l*)	**284**
Poliomyelitis	*152*	213	72	**1,289**(*m*)	**976**	**618**	**441**(*n*)	**1,087**
Typhus fever	*126*	92	28	23	95	**257**	**348**(*o*)	**1,264** (*o*)
Smallpox	*0*	0	0	**1**	0	0	0	0
Notified cases	*56,997*	66,710	53,428	48,196	55,854	75,267	111,647	123,785
Composite epidemic index	100	117.0	93.7	84.6	98.0	132.1	195.9	217.2

HUNGARY

The fact that, from 1942 onwards, available vital statistics refer to greater Hungary and no longer to the territory defined by the Treaty of Trianon makes it difficult to form definite conclusions from Hungarian statistics. Indeed, the *general mortality* rate for 1941 was lower than any previously recorded for the country (*cf.* graph, page 659). The abrupt rise in 1942 may well be the result of

the incorporation in the statistics of a mass of population, representing an increase of 58% over that of "Trianon Hungary", annexed from Slovakia, Ruthenia, Northern Transylvania and northern Yugoslavia, where the standard of living and health conditions are lower than those of Trianon Hungary.

It is well known that these conditions tend in particular to cause a high rate of *infant mortality*, and figures for 1942 and 1943 show a rise in infant mortality over the rates for 1940 and 1941 even greater than the increase observed for general mortality.

The apparent increase in the death rates of the country is therefore of doubtful significance.

The same observation probably applies also to the apparent increase of *tuberculosis mortality* in 1942.

Although this explanation is probably the true one, it does not entirely account for the rise observed in 1942, since as regards the general and infant death rates and the tuberculosis death rate, the rise in that year affected not only the country as a whole, but also the city of Budapest. There must therefore have been unfavourable circumstances affecting the whole of Hungary.

The tuberculosis mortality rate in Budapest rose from 112 in 1939 to 147 in 1943, an increase of 30%.

Since the war, Hungary has remained remarkably free of *epidemic disease*, the only exceptional feature observed being the rise in meningitis in 1940 and, since the beginning of the Russian campaign, the marked increase in the number of sporadic cases of typhus fever throughout the country, and the increase in the typhus endemic in Ruthenia. Owing perhaps to large-scale enforcement of compulsory vaccination, Hungary escaped the diphtheria pandemic which has swept over Europe during the last three years.

As a whole, the sanitary situation can be considered as satisfactory. As to the food situation, it must be recalled that Hungary, as an agricultural country with a wide range of production, is far more than self-supporting.

HUNGARY *(a) (e)*

Following the annexations carried out on November 2nd, 1938, April 4th, 1939, August 30th, 1940, and in April 1941, the January 1941 census gave to " Greater Hungary " a population of 14,670,000 inhabitants, of whom there were 9,314,000 in the territory defined by the Treaty of Trianon.

Yugoslavia invaded : April 6th, 1941.

Participation in the war against the U.S.S.R. : June 22nd, 1941.

Civilian **Population** used for calculating the rates.

HUNGARY *(a) (e)*			BUDAPEST		
C.	XII.1930 :	8,688,000	C.	XII.1930 :	1,006,180
O. N. E.	XII.1939 :	9,129,000	E. S.	VI.1939 :	1,101,800
E. S.	VI.1940 :	9,140,000	E. S.	VI.1940 :	1,113,600
C.	I.1941 :	9,314,300	C.	I.1941 :	1,162,820
E. S.	VI.1942 :	14,750,000	O. N. E.	XII.1941 :	1,165,700
O. N. E.	XII.1942 :	14,843,000	O. N. E.	XII.1942 :	1,167,500

Natural Movement of Population.

Year	HUNGARY *(a)* Births °/₀₀ inhab.	General mortality °/₀₀ inhab.	Increase (+) or decrease (−) °/₀₀ inhab.	BUDAPEST Births °/₀₀ inhab.	General mortality °/₀₀ inhab.	Increase (+) or decrease (−) °/₀₀ inhab.
1928-1938 Max.	*26.4*	*17.9*	*+9.9*	*13.3*	*13.7*	*+0.5*
1928-1938 Med.	*22.0*	*15.2*	*+7.1*	*11.1*	*11.8*	*−0.9*
1928-1938 Min.	*20.1*	*14.2*	*+5.5*	*10.4*	*11.1*	*−1.8*
1937	20.2	14.2	+6.0	10.4	11.6	−1.2
1938	20.1	14.4	+5.7	10.4	11.3	−0.9
1939	19.6	13.7	+5.9	10.3	11.3	−1.0
1940	20.0	14.3	+5.7	11.4	12.5	−1.1
1941	18.5	13.2	+5.3	12.2	11.7	+0.5
1942	20.8	15.3	+5.5	**13.9**	**13.0**	+0.9
1942(1.I-30.IX)	21.3	15.1	+6.2	**14.1**	12.7	+1.4
1943(1.I-30.IX)	19.4	13.9	+5.5	**14.5**	12.5	+2.0

C. = Census.
E. S. = Estimate of the Epidemiological Intelligence Service of the L. o. N.
O. N. E. = Official national estimate.

(a) The territory defined by the Treaty of Trianon (1920-1938) was taken into consideration for demographic statistics up to 1941 inclusive ; from 1942 inclusive, statistics refer to the enlarged territory.

(b) January-November.

(e) Through successive annexations, the population of Hungary was increased from 9,314,300 in January 1941 in the territory defined by the Treaty of Trianon (1920) to 14,843,000 in December 1942 (14,669,600 in January 1941). The censuses of January and October 1941 give the following population figures for these different territories :

1. " Northern territory "—*i.e.*, part of Slovakia and Ruthenia—attributed to Hungary by the first arbitral sentence of Vienna, November 2nd, 1938 : 1,058,300 inhabitants.
2. Another part of Slovakia and the remainder of Ruthenian territory, incorporated April 4th, 1939 : 697,800 inhabitants.
3. Transylvania (about two-thirds of it), awarded by the second arbitral sentence of Vienna, August 30th, 1940 : 2,573,200 inhabitants.
4. The " Southern Territories " (more particularly Batchka), formerly Yugoslav, occupied in April 1941 : 1,026,000 inhabitants.

(f) Figures reduced in the same proportion as that of the population of the former territory (1920-1937) to the population of the enlarged territory, in order to maintain comparability.

(g) January-October. *(h)* January-August and November-December.

Infant Mortality (deaths of infants under 1 year of age, per 1,000 live births).

HUNGARY *(a)*

	Yearly rates	I J	II F	III M	IV A	V M	VI J	VII —	VIII J	IX A	X S	XI O	XII N	XIII D
1928-1938 *Max.*	*184*	*192*	*183*	*177*	*176*	*182*	*177*	M	*223*	*247*	*277*	*264*	*178*	*199*
Med.	*152*	*138*	*137*	*151*	*143*	*140*	*133*	M	*173*	*175*	*148*	*157*	*146*	*148*
Min.	*131*	*117*	*126*	*128*	*120*	*137*	*117*	M	*128*	*146*	*119*	*127*	*108*	*116*
1937	134	137	137	128	127	139	117	M	149	159	143	132	119	125
1938	131	134	142	151	120	137	122	M	133	172	130	134	108	116
1939	121	118	126	132	150	114	125	M	128	132	121	122	106	119
1940	134		157			150		M		111			115	
1941	117		138			123		M		102			108	
1942	151		119			136		M		182			167	
1943	...		135			118		M		140			...	

BUDAPEST

1928-1938 *Max.*	*161*	*189*	*164*	*148*	*129*	*160*	*125*	*120*	*166*	*244*	*243*	*210*	*147*	*154*
Med.	*116*	*100*	*116*	*117*	*110*	*99*	*82*	*74*	*116*	*99*	*103*	*91*	*92*	*111*
Min.	*84*	*76*	*79*	*86*	*89*	*85*	*68*	*55*	*83*	*68*	*73*	*81*	*65*	*95*
1937	111	**124**	**143**	**148**	98	**115**	82	**120**	108	99	90	81	108	104
1938	84	93	96	98	89	86	81	55	83	78	79	89	81	106
1939	92	105	86	105	107	76	84	M	84	92	110	100	77	82
1940	107		120			**116**		M		95			95	
1941	100		105			102		M		93			101	
1942	117		98			100		M		**145**			**128**	
1943	...		98			85		M		111			...	

General Mortality (rates per 1,000 inhabitants on a yearly basis).

HUNGARY *(a)*

	Yearly rates	I J	II F	III M	IV A	V M	VI J	VII —	VIII J	IX A	X S	XI O	XII N	XIII D
1928-1938 *Max.*	*17.9*	*18.7*	*22.2*	*20.9*	*19.8*	*18.1*	*16.8*	M	*17.6*	*18.4*	*20.0*	*20.0*	*17.3*	*17.5*
Med.	*15.2*	*15.8*	*15.8*	*17.2*	*16.6*	*15.2*	*13.8*	M	*14.6*	*14.9*	*14.3*	*14.7*	*14.3*	*14.9*
Min.	*14.2*	*13.6*	*15.0*	*15.4*	*15.1*	*13.9*	*13.4*	M	*12.9*	*13.2*	*12.6*	*12.7*	*12.0*	*13.3*
1937	14.2	15.0	15.7	15.5	15.1	14.8	13.4	M	13.4	13.5	13.1	14.0	12.6	14.1
1938	14.4	16.3	15.8	16.6	16.4	15.2	13.5	M	13.1	14.1	12.8	12.7	12.0	13.3
1939	13.7	**18.0**	16.5	15.5	14.9	12.7	12.4	M	11.9	11.6	11.6	12.4	11.9	12.7
1940	14.3		17.8			15.2		M		12.2			12.3	
1941	13.2		15.8			13.2		M		11.4			12.5	
1942	15.3		15.9			14.9		M		14.5			**15.9**	
1943	...		16.1			13.4		M		12.3			...	

BUDAPEST

1928-1938 *Max.*	*13.7*	*14.8*	*22.0*	*20.3*	*15.2*	*14.1*	*13.1*	*12.1*	*11.6*	*11.9*	*12.3*	*12.4*	*12.8*	*14.5*
Med.	*11.8*	*13.4*	*13.6*	*14.2*	*13.1*	*13.0*	*11.5*	*10.4*	*10.2*	*11.4*	*10.5*	*11.4*	*11.5*	*12.3*
Min.	*11.1*	*11.6*	*12.8*	*12.1*	*11.8*	*10.9*	*10.0*	*10.0*	*9.1*	*8.8*	*9.2*	*9.8*	*10.9*	*11.2*
1937	11.6	13.6	15.1	14.2	11.8	12.6	10.9	10.1	10.2	10.1	9.5	10.4	11.6	11.8
1938	11.3	13.1	12.9	12.1	13.1	13.1	10.5	10.3	9.2	9.5	9.8	9.8	11.2	11.9
1939	11.3	**16.5**	12.4	12.6	11.9	10.7	10.3	M	9.4	8.6	9.5	11.4	10.6	11.6
1940	12.5		**15.6**			12.4		M		10.3			11.6	
1941	11.7		14.0			11.2		M		9.9			11.7	
1942	**13.0**		13.9			12.7		M		**11.6**			**14.0**	
1943	...		14.6			12.1		M		10.7			...	

Tuberculosis Mortality (rates per 100,000 inhabitants on a yearly basis).

	Hungary *(a)*					Budapest				
	Yearly rates	Quarterly I	II	III	IV	Yearly rates	Quarterly I	II	III	IV
1928-1938 Max.	*225*	*248*	*281*	*187*	*170*	*214*	*249*	*269*	*175*	*163*
1928-1938 Med.	*170*	*193*	*204*	*128*	*127*	*149*	*184*	*172*	*116*	*118*
1928-1938 Min.	*142*	*160*	*172*	*113*	*104*	*118*	*140*	*135*	*96*	*97*
1937	149	165	179	118	115	124	152	140	108	98
1938	142	160	172	113	104	118	141	140	96	97
1939	138	172	154	102	100	112	150	127	83	89
1940	135	155	170	110	105	120	139	143	94	102
1941	138	158	173	117	107	126	144	145	114	101
1942	158	176	197	132	127	142 *(g)*	154	171	114	...
1943	147 *(b)*	169	175	121	...	147 *(h)*	160	162	...	...
1944							172	157		

Communicable Diseases : Cases notified in Hungary.

	Median 1934-1938	1937	1938	1939	1940	1941	1942	1943
Typhoid and paratyphoid fevers	*10,144*	10,399	7,741	7,117	5,599	7,969	**12,789**	10,085
Dysentery	*5,308*	5,308	**6,441**	4,197	5,388	3,948	**8,587**	4,592
Diphtheria	*8,148*	8,148	6,266	6,397	4,927	5,049	6,676	8,259
Scarlet fever	*12,931*	11,652	12,931	**13,991**	9,360	**14,178**	**24,101**	**20,344**
Cer.-sp. meningitis	*72*	72	**78**	**395**	**3,516**	**1,776**	**1,000**	**942**
Poliomyelitis	*305*	160	**340**	**693**	**903**	**1,116**	**999**	**555**
Typhus fever	*22*	22	5	**57**	**92**	**621**	**891**	**991**
Smallpox	—	—	—	—	—	—	—	—
Notified cases	*36,930*	35,761	33,802	32,847	29,785	34,657	55,043	45,768
(*f*)	*36,930*	35,761	33,160(*f*)	27,230(*f*)	19,211(*f*)	16,012(*f*)	22,789(*f*)	19,314(*f*)
Composite epidemic index	100	96.8	89.8 (*f*)	73.7 (*f*)	52.0 (*f*)	43.4 (*f*)	61.7 (*f*)	52.3 (*f*)

Eastern Europe.

Poland

There are very few official figures relating to Poland available since the time of her invasion in September 1939. Invasion was accompanied by mass migration movements eastward by Poles fleeing from the German invading armies. Partition of the country between Germany and the U.S.S.R. at the end of 1939 prevented many Poles from the western provinces from returning home. They had consequently to live under extremely unfavourable conditions in over-congested towns and villages of the poorest parts of Poland.

Further migrations took place when the German-occupied area was split into three elements : parts were simply incorporated in pre-war German territory (Polish Corridor, Suvalki district, Polish Silesia), another section, consisting mainly of the departments of Lodz, Kalisz, Posnan, formed the so-called " Wartheland ", given over to German colonisation, and the third, the so-called " Generalgouvernement ", extended eastward up to the Vistula and the San. Large numbers of Polish landowners, bourgeois, and Jews were forced to leave Wartheland for the " Generalgouvernement ", where the living conditions were decidedly bad. They were particularly so in the ghetto of Warsaw, which was formed into a special administrative unit and subjected to special restrictions of all kinds [1]. The only available statistics of mortality or communicable diseases refer to the larger towns of Wartheland, but as this information does not cover the Polish or the Jewish inhabitants, and the corresponding population figures are not precisely known, its value is very small.

These figures, and also various reports and studies which have appeared in the German medical Press, show that the prevalence of *typhus fever* in both western Poland (Wartheland) and central Poland (Generalgouvernement) increased considerably as from the first winter of the German occupation [1]. It was particularly widespread among the Jews herded in the ghettos. No data are available for the Russian-occupied area. According to unofficial information available, typhus fever, which was endemic before the war over a large part of this area, continued to prevail after the German invasion, the German army of occupation having to take special measures, as in Russia, to protect themselves. Notwithstanding these measures, the German army suffered markedly from typhus fever, and also from " Wolhynian fever " (five-day fever, trench fever) also louse-borne and due to *Rickettsia quintana*. The war was accompanied by a rise in the prevalence of water-borne diseases ; typhoid caused a severe epidemic with some 30,000 cases in Warsaw during the siege in September 1939, following upon the cutting-off of the water

[1] Eventually the Jewish population concentrated there was exterminated in pursuance of the official policy of eradication (" Ausrottung ") of the Jews.

[2] Biraud, Y. (1943) : " The Present Menace of Typhus Fever in Europe and the Means of combating it." *Bull. of the Health Organisation*, L.o.N., vol. **10**, N° 1, pages 7-10.

supply. It has remained common in Warsaw and elsewhere, the morbidity being about $1^0/_{00}$ of the population ($2^0/_{00}$ in Wartheland, $1^0/_{00}$ in the " Generalgouvernement ")[1]. Dysentery of the Flexner, and even of the Shiga, type was encountered by the German eastern army, in Poland as well as in Russia. In western Poland it seems that the incidence of diphtheria and scarlet fever remained below that recorded in Germany.

From available evidence, it seems that the effects of communicable diseases on the Polish population as a whole were of far less importance than the effects of food restrictions. These effects are difficult to analyse, as conditions varied enormously from place to place and also as between the different ethnic groups. In Poland, as elsewhere, in spite of requisitions, most of the peasant food producers and the inhabitants of villages generally managed to nourish themselves fairly well. Inhabitants of towns, particularly of the larger towns, fared worse; the poorer classes and those who had lost their fortune through destruction or seizure of their property suffered considerably, because prices prevailing on the black market were beyond their means. Closure of the ghettos prevented the inmates from gaining access to these foodstuffs and, as their official rations were considerably below those of the other Polish citizens, famine played considerable havoc among them, particularly in Warsaw—there the situation was probably worse even than in Athens. According to BOURNE [2], in May 1941 the number of deaths in the Warsaw ghetto was 5,000— *i.e.* $120^0/_{00}$ per annum, a twelvefold increase over the pre-war rate.

The rationing system applied in Poland also differentiates between Poles and Germans [3]. Rations for Germans are considerably better than those for Poles in the same category—in some cases double. According to BOURNE (*loc. cit.*, page 48), in Lwow, the weekly bread ration was $4^1/_2$ pounds (2 kg.) for a German, $2^1/_4$ (1 kg.) for a Pole, and a little over 1 pound ($^1/_2$ kg.) for a Jew. A number of foodstuffs were reserved exclusively for Germans, neither Poles nor Jews being entitled to buy them. (This information appears from official notices

[1] MAYER (1943), *Öffentl. Gesundheitsdienst* 9 : page A377.

[2] BOURNE, G. H. (1943) : *Starvation in Europe*. Allen & Unwin, London, page 49.

[3] This differentiation is also made in Germany to the detriment of Polish workers.

in German local papers : *Ostdeutscher Beobachter*, *Thorner Freiheit*, *Litzmannstädter Zeitung* [1].

The same principle of discrimination against the Poles as compared with the Germans is also found in the working conditions applied to the former in Germany. Many official regulations were published in the *Reichsarbeitsblatt* (German Labour Gazette) fixing longer working-hours, lower salaries, and suppressing holidays where Polish workers are concerned.

Owing to her military losses during the German invasion of 1939, and the Russian counter-invasion, mass emigration of Poles into the U.S.S.R. as from June 1941, the large-scale deportation of both male and female Polish labour into Germany as from 1942, the results of direct and indirect attempts by the German authorities to " eradicate " the Jewish elements, the large number of executions, and the consequences of semi-starvation in towns, Poland ranks among the countries most severely affected by the war.

Two other causes of loss of population must be added—the considerable reduction in births resulting from the keeping in captivity of a great number of men of military age and the prohibition of marriage for a large proportion of others.

The effects of a heightened epidemic level have been comparatively trifling when compared with these other causes of enfeeblement of the nation.

Baltic States

No official demographic or epidemiological statistics have been available for the Baltic States since their incorporation in the U.S.S.R. in 1940, as the policy of the Union since 1937, apparently for military reasons, has been to forbid the publication or issue of figures relating to births, deaths, or diseases. Occupation by Germany in 1941 did not result in any change in this respect. Information about these countries is therefore limited to private communications and reports from local newspapers. Before the war the Baltic States were, generally speaking, self-supporting with regard to the main foodstuffs, Lithuania and Latvia even exporting cereals, and the

[1] Quoted by Bourne, *loc. cit.*

population of these two countries was generally well fed. This situation has not materially altered, in spite of the fact that the German army of occupation drew its supplies from local produce. The peasants, however, showed a tendency to restrict their sales in the towns, with a resulting shortage of food there, though the urban population represents but a very small proportion of the total population of the countries. It is estimated that, even in towns, the official rations at the end of 1943 of 1,060 calories per person per day were supplemented by potatoes and other foods, so as to bring actual consumption to above the 2,000-calorie mark.

The only reports relating to unusual prevalence of disease concern typhus fever, which broke out in sporadic form during the winter of 1941/42, when the German army suffered from it, authorities having recourse to the closing of public places of worship and of entertainment.

The absence of major epidemics and severe food shortages in the Baltic countries—at least until the renewal of military operations there in 1944—does not mean that the population were spared the ravages of war.

First, during the winter of 1939/40, tens of thousands of Balts of German origin had to submit to " repatriation " (some 50,000 from Latvia alone). In 1941, up to the German invasion of June, tens of thousands were transported into the interior of the Soviet Union.

German occupation in its turn led to the incorporation of the stronger men in the Wehrmacht (particularly Estonians and Letts), the " eradication " of Jews (some 50,000), the transfer to Germany for forced labour of many other inhabitants, followed, since the spring of 1944, by hundreds of thousands of refugees.

Here again, losses due to fighting swell those due to deportations and executions.

Union of Soviet Socialist Republics

Since April 1937 no vital statistics or epidemiological data have been issued by the U.S.S.R., and information regarding that country is limited to newspaper and unofficial sources. The sufferings endured by the millions of village and town dwellers, who were transported from their homes eastward into outlying provinces during the retreat of the Russian forces in the summer and autumn of 1941

and 1942 in the carrying-out of the " scorched earth " policy, can only be conjectured. It was of course fortunate that this exodus took place during the warm season of the year, when the prevalence of typhus fever is normally at its lowest. The same remark applies equally to the large-scale movements of the retreating and invading armies during those two years. Protection against water-borne diseases, which are most prevalent precisely in summer, is more easily secured than protection against louse-borne typhus.

German publications give some idea of the prevalence of typhus fever in their forces, and in the Russian population from whom the infection was contracted, during the winter of 1941/42. Among the Germans the disease caused a number of small epidemics, while among the Russian population it was endemo-sporadic, as it had been in pre-war days.[1] In occupied Russia, the disease is known to have been strictly under control during the winter of 1941/42. A large proportion of the Russian population must have benefited from immunity acquired during the pandemic of 1918-1923, when cases were estimated at 25 millions.[2]

Relapsing fever, of which there were also millions of cases after the first world war, has since then gradually receded, and caused no epidemic—at any rate not in the areas occupied by German troops.

The nutritional situation in the U.S.S.R. is better known than the health situation. It is estimated that the occupation by the Germans of the most fertile black-earth region of the Ukraine, not to speak of territories farther north, represented a loss of some 30% of its cereal production. It also meant a considerable loss in sugar, meat and milk production. In spite of strenuous efforts in putting new land under cultivation, and owing in part to the lack of the man-power absorbed by the army and war industries, these losses could not entirely be made good. The food stringency which resulted is evidenced by the important grain shipments from North America to the U.S.S.R. under lend-lease agreements at a time when shipping space was scarce, losses by sinking heavy, and Russia

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[1] *Cf.* Biraud, Y., & Deutschman, S. (1936): " Typhus and Other Exanthematic Rickettsia Infections ", *Epidemiological Report*, L.o.N., **15**, pages 4-7.

[2] Tarassevitch (1922): " Report to the Health Committee of the League of Nations ", *E.I. 2.*

in dire need of war material. The need for grain must have been acute if within a certain period half the tonnage sent to the U.S.S.R. consisted of grain !

In the German-occupied area the situation was no better, in spite of the greatly reduced population, since " the main economic purpose of the (Russian) campaign was to obtain for Germany as much food and oil as possible." [1] From what remained of the destroyed harvest of 1941 and 1942, the German invading army had to be fed and exports made to Germany. This must have caused great hardship among the remaining Russian population.

In gauging the demographic situation resulting from the war in the U.S.S.R., account must be taken of the tremendous losses incurred in the field by the Russian armies in a ceaseless struggle extending over three years on a front of unprecedented length.

The large numbers of civilians executed in the areas occupied by the Germans, particularly of Jews in the large Ukrainian towns, must also be reckoned with.

South-Eastern and Southern Europe.

Roumania

Changes in the population of Roumania as a result of the annexation in 1940 of part of her territory by Hungary (Northern Transylvania), the U.S.S.R. (Bessarabia and Bukovina), and Bulgaria (Southern Dobrudja), with a resulting movement of refugees, many of them settling in Bucarest, make the population figures on which rates are to be based rather uncertain and the rates to a certain extent unreliable. Such as they are, they have remained practically stable during the last six years for the country as a whole. The apparent increase in *general mortality* in Bucarest during 1940 and 1941 may be, in part at least, accounted for by the increased population. Recognition of this increase in 1942 has resulted in an abrupt and hardly credible fall in mortality, from 21.8 to 15.3 ‰ (*cf.* graph, page 659).

[1] Goering, H. [*Instructions for directing the Economy of newly occupied Eastern Regions*], Berlin, June 1941 ; cited by Bourne, *loc. cit.*

Even in peace-time, *tuberculosis mortality* showed large fluctuations from year to year, which makes the interpretation of changes that have occurred during the war difficult. There would appear to be a steady increase from 1939 to 1942, in both the country as a whole and in the capital. It is possible that this rise may be the result of harder conditions of work and economic stringency affecting individuals with limited incomes. It can scarcely be ascribed to a general food shortage in the country, which is one of the greatest food producers and exporters in Europe. It should be borne in mind that rationing of certain articles of food, introduced to prevent undue speculation, was suppressed in the autumn of 1943 at a time when the surplus of food was such that the price of certain articles on the black market fell below the price fixed by the Government for the rationed foods.

Infant mortality has remained close to the average of pre-war years. Fluctuations in the rates for Bucarest have been considerable, but, except in 1942, they did not exceed the median for the 1928-1938 period.

The absence of any large-scale *epidemic* among the civilian population, in spite of conscription and migration, is a remarkable achievement. The only disease which showed an abnormal prevalence was typhus fever, the incidence of which rose noticeably in 1942-1943 in the endemic areas of Bessarabia and the adjoining territories ; it also spread in the form of sporadic cases and minor epidemics to the country as a whole.

Although the health situation in the country—where a high infant and general mortality prevailed in normal times—cannot be said to be really satisfactory, there seems to be no doubt that, notwithstanding difficulties resulting from migrations and the war, epidemics have been kept well under control, and no widespread food shortage has occurred.

Roumania

From 19,900,000 on the July 1st, 1939, the population fell to 13,300,000 on September 7th, 1940, after the following territories were ceded :

1. Bessarabia and Northern Bukovina to the U.S.S.R., June 28th, 1940 (about 3,700,000 inhabitants) ;
2. Two-thirds of Transylvania to Hungary, August 30th, 1940 (about 2,600,000 inhabitants) ;
3. Southern Dobrudja to Bulgaria, Sept. 7th, 1940 (about 320,000 inhabitants).

In June 1941, Roumania joined Germany in the invasion of the U.S.S.R. Recuperation of the territories of Bessarabia and Northern Bukovina increased the number of the population at the end of 1941 to 16,800,000.

Civilian **Population** used for calculating the rates.

Roumania *(a)*			Bucharest *(b)*		
C.	XII.1930 :	18,057,000	C.	XII.1930 :	631,288
O. N. E.	1.VII.1939 :	19,933,802	O. N. E.	1.VII.1939 :	649,564
O. N. E.	7.IX.1940 :	13,299,185	O. N. E.	1.VII.1940 :	659,610
O. N. E.	1.VII.1941 :	16,742,723	O. N. E.	1.VII.1941 :	659,070
O. N. E.	1.VII.1942 :	16,843,539	O. N. E.	1.VII.1942 :	991,400

Natural Movement of Population.

Year	Roumania: Births ‰ inhab.	Roumania: General mortality ‰ inhab.	Roumania: Increase (+) or decrease (−) ‰ inhab.	Bucharest: Births ‰ inhab.	Bucharest: General mortality ‰ inhab.	Bucharest: Increase (+) or decrease (−) ‰ inhab.
1928-1938 *Max.*	*35.9*	*21.7*	*+15.8*	*23.0*	*18.1*	*+5.5*
1928-1938 *Med.*	*32.4*	*20.2*	*+12.7*	*20.9*	*16.7*	*+4.3*
1928-1938 *Min.*	*29.6*	*18.7*	*+ 9.6*	*17.3*	*14.9*	*+2.4*
1937	30.8	19.3	+11.5	**22.4**	16.9	+5.5
1938	29.6	19.2	+10.4	**23.0**	**18.1**	+4.9
1939	28.3	18.6	+ 9.7	**22.4**	17.9	+4.5
1940 *(f)*.....	26.5	19.2	+ 7.3	20.7	**20.7**	0.0
1941 *(f)*.....	25.7	19.1	+ 6.6	20.7	**21.8**	−1.1
1942	24.4	19.5	+ 4.9	13.8 *(b)*	15.3 *(b)*	−1.5

C. = Census.
E. S. = Estimate of the Epidemiological Intelligence Service of the L. o. N.
O. N. E. = Official national estimate.

(a) Population figures are taken from the official Roumanian organ *Buletinul demografic al României*, 1939-1943. Rates of births, general mortality, and infant mortality published for Roumania are taken from the same source. Owing to extensive displacements of population as a result of the war and of territorial transfers, population figures and rates given are only approximate from the beginning of 1940, particularly as concerns the years 1940 and 1941.

(b) The numbers of births and deaths given in official statistics at Bucharest show a slight increase for the second half of 1940 and for the year 1941, which can be explained by the influx of refugees from Bessarabia and Bukovina, Transylvania and Dobrudja. This increase is not, however, on a scale comparable with the increase in official population figures between July 1st, 1941 (659,000) and September 1st, 1941 (993,000). The sustained population figures recorded from 1939 to the middle of 1941 are obviously abnormal in view of movements of refugees ; inversely the decrease in the rates for 1942 appears to be solely due to the change in the official basic population.

(d) Deaths.
(e) Monthly rates based on the years 1930-1938.
(f) Rates only approximate, owing to changes in the population (see notes *(a)* and *(b)*).
(g) Rates only approximate, owing to territorial changes.
(h) Monthly rates based on the years 1932-1938.
(i) Provisional data.

Infant Mortality (deaths of infants under 1 year of age, per 1,000 live births).

ROUMANIA

	Yearly rates	I J	II F	III M	IV A	V M	VI J	VII —	VIII J	IX A	X S	XI O	XII N	XIII D
1928-1938 *Max(h)*	*197*	*297*	*304*	*227*	*186*	*183*	*205*		*234*	*223*	*196*	*201*	*226*	*286*
1928-1938 *Med( )*	*182*	*180*	*160*	*176*	*174*	*163*	*155*		*179*	*202*	*172*	*166*	*181*	*245*
1928-1938 *Min(h)*	*174*	*138*	*155*	*157*	*166*	*146*	*138*		*137*	*157*	*149*	*139*	*162*	*196*
1937	178	195	186	176	174	156	138		188	191	**176**	**182**	176	196
1938	183	180	159	**190**	**185**	**183**	155		179	205	174	162	182	**259**
1939	176	198	**210**	**213**	**208**	149	139		159	170	149	150	164	228
1940 *(g)* ...	**189**	170	188	**219**	**200**	**174**	**162**		160	204	**176**	**183**	**206**	**279**
1941 *(g)* ...	171	205	182	**190**	174	155	130		133	167	161	155	175	213
1942	183	178	171	**195**	**230**	**181**	**162**		158	161	155	177	**207**	**263**

BUCHAREST

1928-1938 *Max.*	*205*	*167*	*181*	*166*	*211*	*212*	*252*		*312*	*282*	*224*	*212*	*230*	*214*
1928-1938 *Med.*	*177*	*131*	*144*	*151*	*158*	*150*	*190*		*248*	*201*	*163*	*167*	*148*	*166*
1928-1938 *Min.*	*155*	*116*	*113*	*124*	*116*	*96*	*161*		*198*	*171*	*87*	*103*	*113*	*116*
1937	155	133	153	148	158	96	147		234	171	139	157	148	164
1938	156	127	127	**158**	**211**	**173**	178		248	186	87	103	113	166
1939	129	120	112	116	141	105	108		204	154	123	109	117	138
1940	170	135	129	143	157	158	135		206	**277**	**201**	**172**	141	**197**
1941	160	**151**	146	144	175	121	132		227	**228**	164	134	143	132
1942	179	120	134	**186**	213	157	160		**282**	**253**	165	157	144	181

General Mortality (rates per 1,000 inhabitants on a yearly basis).

ROUMANIA

	Yearly rates	I J	II F	III M	IV A	V M	VI J	VII —	VIII J	IX A	X S	XI O	XII N	XIII D
1928-1938 *Max(e)*	*21.7*	*24.9*	*37.4*	*24.3*	*24.5*	*20.5*	*20.9*		*24.3*	*24.0*	*22.6*	*24.0*	*25.4*	*22.9*
1928-1938 *Med(e)*	*20.2*	*19.7*	*21.2*	*21.0*	*21.2*	*18.5*	*16.9*		*18.3*	*19.7*	*18.4*	*18.4*	*19.1*	*20.5*
1928-1938 *Min(e)*	*18.7*	*17.4*	*19.7*	*19.6*	*20.0*	*17.7*	*15.8*		*15.1*	*16.1*	*17.1*	*16.3*	*16.8*	*19.7*
1937	19.3	21.6	**21.5**	20.6	20.0	17.7	15.8		**18.9**	19.7	18.4	**20.6**	18.7	19.7
1938	19.2	19.7	19.7	21.7	**21.4**	**19.5**	16.5		17.7	20.1	18.0	17.9	18.6	**20.7**
1939	18.6	19.4	**21.6**	**25.4**	**24.9**	17.1	15.2		15.9	16.8	15.8	16.8	16.9	17.8
1940 *(f)*	19.2	21.0	**22.3**	**24.9**	**22.5**	18.8	15.7		15.1	17.7	17.4	17.7	17.7	20.0
1941 *(f)*	19.1	20.7	20.5	18.2	17.5	16.0	15.2		16.3	18.8	**21.1**	18.6	19.7	**22.6**
1942	19.5	20.9	20.7	21.5	20.9	18.0	15.8		15.8	17.8	18.5	20.4	**21.0**	**22.5**

BUCHAREST

1928-1938 *Max(e)*	*18.1*	*19.5*	*21.3*	*21.1*	*25.5*	*20.2*	*20.3*		*23.9*	*19.7*	*17.6*	*18.0*	*19.2*	*20.5*
1928-1938 *Med(e)*	*16.7*	*17.0*	*18.0*	*17.8*	*17.6*	*17.0*	*17.0*		*17.9*	*17.5*	*16.4*	*17.1*	*16.6*	*17.3*
1928-1938 *Min(e)*	*14.9*	*14.3*	*15.2*	*15.8*	*15.8*	*13.7*	*14.0*		*16.5*	*14.3*	*14.1*	*13.6*	*13.1*	*14.1*
1937	16.9	**19.3**	**20.5**	**19.0**	**18.8**	**17.6**	17.0		**20.2**	17.7	16.5	**18.0**	**17.5**	17.3
1938	**18.1**	**19.5**	**18.4**	**21.1**	**25.5**	**20.2**	**20.3**		**21.6**	**19.0**	15.7	17.1	16.6	**20.5**
1939	17.9	**18.7**	18.3	**19.8**	**20.1**	17.4	16.6		18.7	17.5	16.3	17.0	16.7	**17.7**
1940	**20.7**	**21.4**	**20.1**	**22.4**	**22.7**	**20.9**	18.2		19.0	**21.8**	**20.2**	**18.5**	**21.4**	**21.8**
1941	**21.8**	**27.3**	**22.1**	**21.8**	**21.3**	**18.2**	17.2		**21.9**	**22.3**	**22.4**	**22.0**	**21.0**	**23.7**
1942 *(b)* ...	15.3	15.1	15.7	17.4	16.3	14.2	13.4		16.2	16.2	14.4	13.3	15.6	16.3

Tuberculosis Mortality (rates per 100,000 inhabitants on a yearly basis).

	Roumania					Bucharest				
	Yearly rates	Quarterly I	II	III	IV	Yearly rates	Quarterly I	II	III	IV
1932-1938 Max.	*181*	*223*	*232*	*154*	*145*	*284*	*345*	*357*	*262*	*255*
1932-1938 Med.	*175*	*192*	*211*	*148*	*142*	*259*	*297*	*311*	*208*	*212*
1932-1938 Min.	*166*	*179*	*202*	*140*	*134*	*242*	*278*	*278*	*181*	*191*
1937	**178**	**206**	**220**	148	142	**283**	**345**	**357**	220	212
1938	168	191	209	140	134	**284**	**307**	**325**	**262**	**243**
1939	162	190	202	126	130	264	313	320	214	211
1940 *(f)*	177	195	209	146	140	**305**	**338**	**380**	**256**	**247**
1941 *(f)*	**181**	**213**	**222**	**155**	131	**311**	**401**	**334**	**269**	**243**
1942 *(f)*	**191**	**211**	**245**	**162**	**148**	204	243	250	167	158
1943 *(f)*		**217**								

Communicable Diseases: Cases notified in Roumania.

	Median 1928-1938	1937	1938	1939	1940	1941	1942 *(i)*	1943 *(i)*
Typhoid and paratyphoid fevers	*6,512*	**7,802**	6,512	4,347	2,442	2,599	4,539	4,394
Dysentery	*6,037*	**6,897**	6,189	2,733	2,539	2,843	**3,075**	1,469
Diphtheria	*4,337*	2,889	2,290	2,279	1,839	1,103	1,612	1,879
Scarlet fever	*21,851*	21,851	20,925	26,675	15,428	8,084	12,781	24,082
Cer.-sp. meningitis	*71*	**85**	**90**	**69**	**226**	**113**	**308**	**389**
Poliomyelitis	*137*	163	150	122	154	120	96	124
Typhus fever	*1,857*	**4,976**	2,254	1,014	1,378	1,906	**4,124**	**8,362**
Smallpox	*6*	15	1(*d*)	1(*d*)	1(*d*)	—	30	—
Notified cases	*40,808*	44,678	38,411	37,240	24,007	16,768	31,565	41,699
Composite epidemic index	100	109.5	94.1	91.3	58.8	41.1	77.4	102.2

Bulgaria

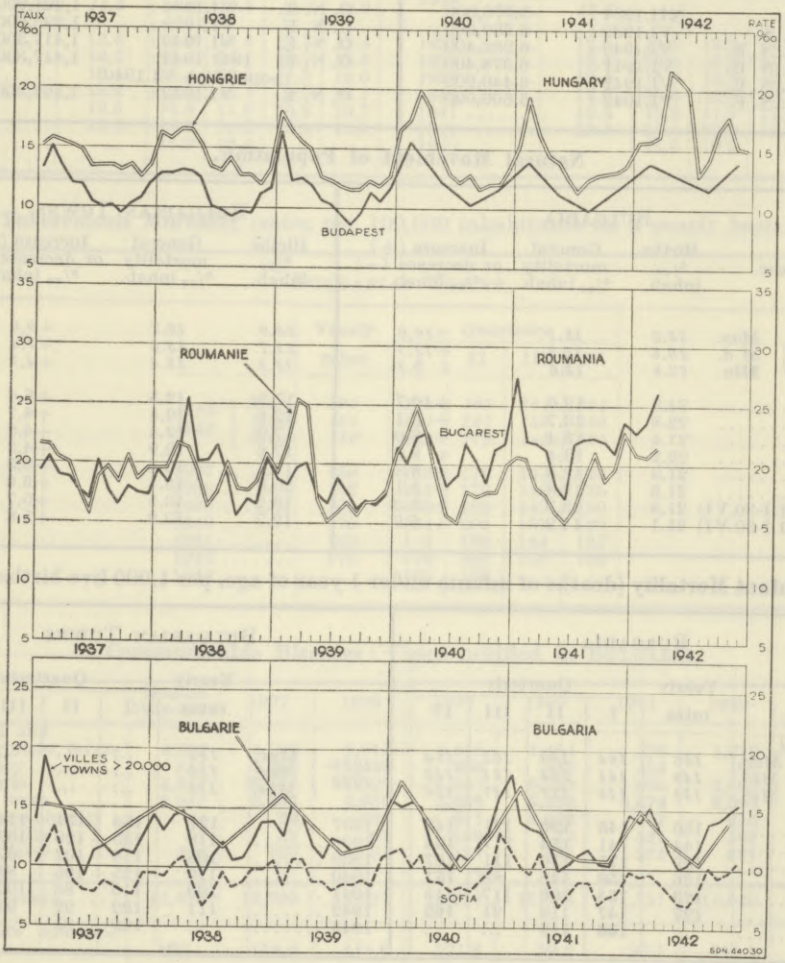
General mortality, which was slowly declining in the whole of Bulgaria and also in Bulgarian towns has risen slightly since 1941.

The rise was slightly greater with regard to *tuberculosis mortality* in Bulgarian towns.

Infant mortality rates have remained below pre-war averages —those for 1941 and 1942 for the whole of Bulgaria were below pre-war minima, those for Bulgarian towns have remained below the pre-war minimum since 1939.

This general fall in the death rate has counterbalanced the slight decline in the *birth rate*, so that the natural increase in the population has remained unchanged and comparatively high.

General Mortality, per 1,000 Inhabitants, in Bulgaria, Hungary and Roumania, 1937-1942.



N. B. — For war years, rates are approximate.

Bulgaria

In April and May 1941, Bulgaria joined in the invasion, and then the occupation, of northern Greece (Thrace) and of part of Yugoslavia (Macedonia).

Civilian **Population** used for calculating the rates.

Bulgaria *(a)*			Bulgarian Towns *(a)*		
C.	XII.1934 :	6,078,000	O. N. E.	VI.1934 :	1,291,900
E. S.	VI.1939 :	6,280,000	O. N. E.	VI.1939 :	1,395,600
O. N. E.	VI.1940 :	6,332,400	O. N. E.	VI.1940 :	1,417,300
O. N. E.	VI.1941 :	6,378,400	O. N. E.	1941-1942 : (population VI.1940)	1,417,300
O. N. E.	VI.1942 :	6,440,000			
O. N. E.	VI.1943 :	6,500,000	O. N. E.	VI.1943 :	1,462,900

Natural Movement of Population.

Year	Bulgaria: Births ‰ inhab.	Bulgaria: General mortality ‰ inhab.	Bulgaria: Increase (+) or decrease (−) ‰ inhab.	Bulgarian Towns: Births ‰ inhab.	Bulgarian Towns: General mortality ‰ inhab.	Bulgarian Towns: Increase (+) or decrease (−) ‰ inhab.
1928-1938 Max.	*33.2*	*18.1*	*+16.0*	*23.9*	*16.7*	*+9.6*
1928-1938 M d.	*29.5*	*15.6*	*+12.5*	*21.7*	*13.5*	*+7.4*
1928-1938 Min.	*22.8*	*13.6*	*+ 9.1*	*17.1*	*12.4*	*+4.7*
1937	24.3	13.6	+10.7	17.9	12.5	+5.4
1938	22.8	13.7	+ 9.1	17.1	12.4	+4.7
1939	21.4	13.4	+ 8.0	17.2	12.4	+4.8
1940	22.2	13.4	+ 8.8	17.2	12.9	+4.3
1941	21.3	12.5	+ 8.8	17.7	12.4	+5.3
1942	21.9	12.8	+ 9.1	20.0	13.4	+6.6
1942 (1.1-30.VI)	21.8	13.5	+ 8.3	19.2	13.5	+5.7
1943 (1.I-30.VI)	21.1	14.2	+ 6.9	19.7	13.9	+5.8

Infant Mortality (deaths of infants under 1 year of age, per 1,000 live births).

	Bulgaria: Yearly rates	Bulgaria: Quarterly I	II	III	IV	Bulgarian Towns: Yearly rates	Quarterly 1	II	III	IV
(b) 1928-1938 Max.	*156*	*184*	*190*	*182*	*164*	*152*	...	...	...	...
1928-1938 Med.	*149*	*141*	*154*	*121*	*142*	*129*	...	...	...	...
1928-1938 Min.	*131*	*118*	*117*	*117*	*128*	*118*	...	...	...	...
1937	150	**145**	**190**	121	142	127	154	131	114	110
1938	144	141	**158**	117	**164**	118	135	120	107	135
1939	139	**175**	136	111	127	105	118	102	97	103
1940	136	**156**	146	88	**157**	112	125	105	86	134
1941	123	**154**	106	117	113	100	110	86	105	98
1942	127	**147**	116	91	**163**	111	132	92	94	133
1943	...	**166**	119	...	...	...	129	81	...	...

C. = Census.
E. S. = Estimate of the Epidemiological Intelligence Service of the L. o. N.
O. N. E. = Official national estimate.

(a) Bulgaria and Bulgarian towns according to the territory defined in the Treaty of Neuilly (excluding Southern Dobrudja and other annexed territories).

(b) Quarterly rates based on the period 1934-1938.

For footnotes (e) and (f), see page 661.

General Mortality (rates per 1,000 inhabitants on a yearly basis).

	Bulgaria					Bulgarian Towns *(e)*				
	Yearly rates	Quarterly				Yearly rates	Quarterly			
		I	II	III	IV		I	II	III	IV
1928-1938 *Max.*	*18.1*	*22.9*	*18.6*	*16.3*	*19.9*	*16.7*	...	...	...	...
1928-1938 *Med.*	*15.5*	*17.3*	*14.7*	*13.4*	*13.8*	*13.5*	...	...	...	...
1928-1938 *Min.*	*13.6*	*14.0*	*10.8*	*9.4*	*12.9*	*12.4*	...	...	...	...
1937	13.6	14.9	14.4	11.7	13.4	12.5	14.6	12.2	11.4	11.9
1938	13.7	15.4	14.1	11.7	13.8	12.4	12.8	13.8	11.2	12.6
1939	13.4	16.4	13.8	11.3	12.0	12.4	13.8	12.6	11.4	11.8
1940	13.4	17.3	13.7	9.9	12.7	12.9	15.6	12.5	10.3	13.2
1941	12.5	16.8	11.9	10.8	10.7	12.4	15.2	11.7	11.4	11.2
1942	12.8	14.8	12.2	10.4	13.9	13.4	15.1	12.0	11.7	15.0
1943	...	15.9	12.4	...	...	...	15.6	12.3	...	...

Tuberculosis Mortality (rates per 100,000 inhabitants on a yearly basis).

Bulgarian Towns

	Yearly rates	Quarterly			
		I	II	III	IV
1928-1938 *Max.*	*309*	*352*	*368*	*261*	*254*
1928-1938 *Med.*	*185*	*211*	*221*	*159*	*150*
1928-1938 *Min.*	*150*	*152*	*170*	*143*	*125*
1937	150	152	172	147	128
1938	152	152	172	148	135
1939	162	180	192	147	130
1940	170	184	200	167	129
1941	163	1 4	188	144	121
1942	176	178	201	156	169
1943	...	180	188	...	...

Communicable Diseases : Cases notified in Bulgaria.

	Median 1928-1938	1937	1938	1939	1940	1941	1942	1943
Typhoid and paratyphoid fevers	*4,509*	4,509	2,911	2,418	1,464	1,496	1,238	779
Dysentery	*513*	**1,955**	**1,466**	**890**	**612**	453	158	57
Diphtheria	*4,355*	**9,211**	5,728	5,367	3.732	1,935	3,606	5,300
Scarlet fever	*2.957*	**3,835**	**4,056**	**5,699**	**5,288**	**6,678**	**8,594**	**5,171**
Cer.-sp. meningitis .	*34*	**53**	**202**	**671**	**1,239**	**497**	**215**	**203**
Poliomyelitis	*39*	39	**67**	**120**	**72**	**421**	**167**	**175**
Typhus fever	*216*	160	91	129	127	**271**	**671**	**1,118**
Smallpox	—	—	—	4	—	—	—	—
Notified cases	*12,623*	19,762	14,521	15,298	12,534	11,751	14,649	12,803
Composite epidemic index	100	156.6	115.0	121.2	99.3	93.1	116.1	101.4

For footnotes (a) and (b), see page 660.

(e) For want of comparable retrospective data, quarterly rates do not indicate whether the upper quartile has been exceeded.

(f) The territory defined by the Treaty of Neuilly (November 27th, 1919), which comprised 6,078,000 inhabitants on December 31st, 1934, and about 6,381,000 at the end of 1940, has been modified as follows : September 7th, 1940, addition of Southern Dobrudja (319,000 inhabitants) ; May 1941, occupation and annexation of Greek (Thrace) and Yugoslav (Macedonia) territories (about 1,300,000 inhabitants).

The only abnormal *epidemic* phenomenon was the increased sporadic incidence of typhus fever from 1941 onwards. There has been no large-scale or lasting epidemic. Meningitis, as in other countries, showed an unusually high incidence in 1940, followed by a gradual decline. The scarlet-fever level, which was high before the war, has remained so without the disease reaching epidemic proportions. Diphtheria prevalence was normal.

Bulgaria being an agricultural country and having participated only to a very limited extent in active warfare, there has been no reason for serious food shortages. Although food rationing was introduced in the towns, shortage of food did not affect the bulk of the population, apart perhaps from the low-income groups in the larger towns, as a result of the rise in the cost of living.

Both the nutritional and the sanitary situation can therefore be considered as satisfactory.

Greece

Greece, which sustained a hard winter campaign when attacked by Italy in October 1940 and German invasion in April and May 1941, is probably the country which has, as a whole, suffered more than any other European country from actual famine during recent years. What was left of food stores were seized by the army of occupation for its subsistence, and even for export (olive oil), so that there was an acute shortage in the autumn of 1941 and in the winter of 1941/42. The weather during this winter was unusually severe, and in the large towns and in many of the smaller islands the shortage became acute and starvation widespread. Indeed, children and adults died of hunger by thousands in the streets of Athens. The lack of food was so complete that death occurred rapidly, before symptoms of vitamin or other deficiencies such as famine œdema could develop. Motor-lorries picked up people dead of hunger daily in the streets, the number was so large that individual burials were not possible and mass interment in common graves was resorted to. With milder weather, and imports of relief supplies through International and national Red Cross action, the situation improved somewhat and acute starvation was gradually replaced by chronic forms of famine diseases. In spite of outside efforts the food situation has remained bad and malnutrition in all its forms is rife, particularly in the towns.

In these, actual shortage of food has been greatly accentuated in 1944 by the effects of inflation, which has reached levels comparable with those obtaining in Germany in 1923. Peasants have little incentive to bring their produce to market when the millions of drachmæ they receive in exchange can buy very little and rapidly depreciate. Obviously, not all town dwellers can have recourse to barter to obtain agricultural products.

According to an official statement by the Greek Government, the number of people who died from starvation, from April 1941 to the end of 1943, in Athens, Piræus and 30 provincial capitals, was 104.612. The excess of mortality due to under-nourishment during that period was reckoned at 270,000.

These figures will not surprise those acquainted with the fact that only a minor part of the food requirements of Greece were grown in the country. Wines, fruit, tobacco, olives are grown for export, being traded against corn and other essential foodstuffs, which trading was not possible under war conditions. The country has also had to support a large army of occupation in addition to its own population.

Furthermore, the country suffered losses not only as the result of military operations but also through the executions carried out by the occupying armies, which were estimated in May 1944 at 70,000, and in consequence of a reduction in the number of births, amounting to some 300,000 in three years.

GREECE

Communicable Diseases : Cases notified in GREECE.

	Median 1928-1938	1937	1938	1939	1940	1941	1942	1943
Typhoid and paratyphoid fevers	*3,701*	**6,266**	**4,896**	**5,001**	3,275	3,476	3,934 (*a*)	3,901 (*a*)
Dysentery	*607* (*b*)	255	**673**	571	256	103	217 (*a*)	310 (*a*)
Diphtheria	*981*	990	981	1,033	924	984	764 (*a*)	543 (*a*)
Scarlet fever	*2,220*	573	578	391	181	191	105 (*a*)	95 (*a*)
Cer.-sp. meningitis	*198*	**440**	**479**	**389**	**374**	**511**	**894** (*a*)	**630** (*a*)
Poliomyelitis	*26* (*g*)	**50**	49	22	20	6	29 (*a*)	10 (*a*)
Typhus fever	*63*	73	60	**102**	**89**	35	**688**	**510**
Smallpox	*3*	3	—	—	—	—	—	830 (*f*)
Notified cases	*7,796*	8,647	7,716	7,509	5,119	5,757 (*e*)	7,386 (*e*)	7,626 (*e*)
Composite epidemic index	100	110.9	99.0	96.3	65.7	73.8 (*e*)	94.7 (*e*)	97.9 (*e*)

(*a*) Excluding the districts of Drama, Kavalla, Serres (Macedonia), Hevros, Rhodope (Thrace), Cephalonia, Corfu, Zante (Ionia).

(*b*) Median 1930-1938 only.

(*e*) These figures were adjusted to take account of the occupation of Greek territories since April 1941.

(*f*) With 187 deaths in the district of Hevros.

(*g*) Median 1932-1938 only.

Notwithstanding these conditions, there was no abnormal prevalence of *epidemic disease* except a rise in typhus fever during 1942 and 1943, and the second half of 1943 saw the introduction of smallpox from Turkey by gypsies in the department of Hevros—830 cases with 187 deaths recorded in that department, and a somewhat smaller number during the first half of 1944. The disease, however, was restricted to a certain well-defined zone.

Turkey

Turkey, as a neutral Power situated on the periphery of Europe, need not be included in the present review from the nutritional standpoint, as she did not suffer from the shortage of food due to actual warfare or to requisitioning, nor was there any excessive drain on the country's food resources through exportation [1]. It is true, however, that Turkey has suffered from the blockade and the inadequacy of shipping.

From the *epidemic* point of view, however, Turkey cannot be ignored, in view of her strategic position. During the war, suppression of most of the sea-borne traffic through the Dardanelles and the considerable restriction of land traffic in the Balkans have limited the importance of that position. But the re-opening of communications makes the epidemic situation in Turkey of considerable importance to Europe.

Two points of interest should be considered. First, the high endemo-sporadic incidence of typhus fever in recent years throughout the country, which has culminated in what amounts to an epidemic in 1943. This epidemic showed little sign of abatement in the early months of 1944. There were 810 cases, with 100 deaths on the European territory, most of them in Istanbul. The disease was scattered throughout the country. Secondly, smallpox broke out in 1942 and reached very large proportions in 1943. Over 2,000 cases, with 372 deaths, took place on European territory, out of 12,400 cases recorded for the country as a whole. The disease was still spreading during the first months of 1944; although unequally distributed, it was to be found in nearly every vilayet. The disease is of the severe type and highly infectious. It has been carried by gypsies into Greece, where it produced a secondary epidemic.

[1] The sending of shiploads of grain to Greece for relief purposes must, however, be mentioned in this connection.

Cholera has been absent from the country for over 20 years. The same is also the case as regards plague [1].

TURKEY

Communicable Diseases : Cases notified in TURKEY.

	Median 1928-1938	1937	1938	1939	1940	1941	1942	1943
Typhoid fever	*3,349*	**5,815**	3,349	3,211	3,091	3,139	3,812	3,293
Paratyphoid fevers .	*214*	**277**	125	174	221	198	167	153
Dysentery	*407*	322	383	304	341	**598**	**638**	**650**
Diphtheria	*1,204*	1,188	1,087	1,038	633	936	869	846
Scarlet fever	*917*	765	796	607	472	604	341	338
Cer.-sp. meningitis .	*521*	**968**	**739**	**819**	669	476	616	400
Typhus fever	*259*	**667**	**450**	**463**	**816**	**950**	**873**	**4,143**
Smallpox	*188*	36	**641**	431	544	7	**1.871**	**12,370**
Notified cases	*6,071*	10,038	7,570	7,047	6,787	6,908	9,192	22,193
Composite epidemic index	100	165.3	124.7	116.1	111.8	113.8	151.4	365.6

YUGOSLAVIA

Vital statistics available for Yugoslavia before the war were comparatively limited. Since the invasion of that country in April 1941, they are practically nil.

We are in a better position with regard to epidemiological statistics. Before the war they were issued weekly from the Central Hygiene Institute in Belgrade and gave considerable geographical detail. Since invasion and the partition of the country, regular information has been unobtainable for Serbia proper, but has become available for Croatia, while for the south-eastern part of Yugoslavia occupied by Bulgaria, it has been embodied in Bulgarian reports.

From the information available, it appears that typhus fever has constituted the most serious health problem in that country. The disease naturally manifested itself in detention camps, where sanitation was defective, as was the case in the immediate neighbourhood of Belgrade in March 1943. It has prevailed sporadically in Macedonia and all Serbia. It has assumed truly epidemic proportions in Croatia, owing to the particularly troubled state of that area. Already in 1943 the weekly number of cases reached 300 to 350. During the winter of 1943/44 the epidemic redoubled in violence, as many as 800 cases being recorded in a single week in May 1944. The total

[1] The rumour which found its way into the Press concerning 5,000 cases of plague in Adrianople (Edirne) was quite unfounded.

number of cases notified during the first half of that year exceeded 6,500—that is ten times the figure normally recorded for the whole year for all Yugoslavia.

The incidence of typhoid fever was also unusually high.

The quantities allocated by official ration cards in Croatia are an indication that the people there are seriously short of food—particularly in the towns. Bread is definitely scarce, this situation being due to the fact that, even in normal times, Croatia is hardly self-supporting for food and, in addition, it has now to contend with foreign requisitions and the effects of abnormally small sowings as a result of disturbed conditions, for at least two successive years. It is therefore obvious that active warfare in this region decreases the number of producers and increases the number of consumers, while destroying harvests and stores.

In Serbia the situation appears to be better, although far from satisfactory. Official rations in 1942 do not provide more than 1,000 calories a day and not more than 30 g. of protein, a grossly deficient diet. Famine œdema has been reported in Serbian towns.

From the very incomplete information at hand, it appears that, while the nutritional and health situation in Serbia is definitely unsatisfactory, it is really serious and becoming worse in Croatia. The population is suffering real hardships, particularly in the more arid areas of Herzegovina and Montenegro.

In an appraisal of the losses sustained by the Yugoslav population, account must be taken not only of the losses in the field in April 1941, but of those incurred during the later guerilla warfare, with its resulting reprisals and counter-reprisals, and also of the massacres of the Serbian population in the western part of the country shortly after the foreign occupation began [1].

Italy

No significant change has taken place during the war years in the *mortality rates* for Italy or for Rome, the slight rise which occurred for both during the year 1942 being due to an unusually high prevalence of winter influenza-like infections in January and February (*cf.* graph, page 581).

[1] The number of victims of these massacres is estimated at 700,000.

Tuberculosis mortality remained practically unaffected until 1942, when it showed an abrupt increase of 25% for the Kingdom as a whole.

In 1941 *infant mortality* showed a rise. This transitory increase did not continue in 1942. Too much significance should not be attached to fluctuations in infant mortality in a warm country. The excess in 1941 occurred for the most part during the summer months.

The *epidemic situation* until the end of 1942 was not out of the ordinary,[1] although the incidence of typhoid was high and that for meningitis and poliomyelitis unusually so. Perhaps the most striking event was the outbreak of typhus fever, which took place in Naples from October 1943 to January 1944. The number of cases aggregated some 500.[2] A small outbreak of smallpox of some 50 cases also occurred in Naples and Taranto at the end of April 1944, after the practical disappearance of the disease for several years.

Up to 1942 inclusive, the available mortality statistics and figures for epidemic diseases did not indicate any really abnormal development as regards the civilian population. The birth rate, it is true, fell sharply as from 1941, but there was still a fairly high natural increase in the population. It is unfortunate that figures are lacking for the very period during which the sufferings of the population must have been greatest—that is to say, during the invasion period, which was accompanied by civil strife, increasing rigour of the German occupation, spontaneous or forced migration before the advancing battle front, the breakdown of communications and consequently of the food-rationing system in the cities and particularly in Rome. It is therefore impossible to ascertain to what extent the nutritional and health situation has deteriorated since the middle of 1943.

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[1] Unfortunately, regular and detailed statistics on communicable diseases for Italy are no longer available, since 1943.

[2] The fact that the people were living herded together in caves and the destruction of the water supply during the last days of German occupation created ideal conditions for the spread of the disease. Typhus fever was repeatedly imported from Tunisia during the Italo-German evacuation of that country.

ITALY

Declared war on Great Britain and France : June 10th, 1940 ; attacked Greece : October 28th, 1940. Sicily invaded by the Anglo-Americans : July 10th, 1943 ; the mainland : September 3rd, 1943.

Civilian **Population** used for calculating the rates.

ITALY			ROME		
C.	1V.1936 :	42,444,600	C.	1V.1936 :	1,173,034
O. N. E.	VI.1939 :	44 213,000	E. S.	V1.1939 :	1,303,433
O. N. E.	V1.1940 :	44.667,000	O. N. E.	V1.1940 :	1,348 671
O. N. E.	VI.1941 :	45,065 000	O. N. E.	VI.1941 :	1.391 950
O. N. E.	V1.1942 :	45,515,000	O. N. E.	V1.1942 :	1,448,989
O. N. E.	V1.1943 *(i)* :	45,809,000	O. N. E.	V.1943 :	1,464,600

Natural Movement of Population.

Year	ITALY Births ‰ inhab.	ITALY General mortality ‰ inhab.	ITALY Increase (+) or decrease (−) ‰ inhab.	ROME Births ‰ inhab.	ROME General mortality ‰ inhab.	ROME Increase (+) or decrease (−) ‰ inhab.
		(e)				
1928-1938 Max.	*26.7*	*16.5*	*+12.6*	*23.8*	*14.6*	*+12.0*
1928-1938 Med.	*23.8*	*14.1*	*+ 9.6*	*21.9*	*11.8*	*+ 9.9*
1928-1938 Min.	*22.4*	*13.3*	*+ 8.6*	*20.8*	*11.0*	*+ 8.3*
1937	22.9	14.2	+ 8.7	21.9	11.6	+10.3
1938	23.7	14.1	+ 9.6	23.6	11.8	+11.8
1939	23.5	13.4	+10.1	23.2	10.7	+12.5
1940	23.4	13.6	+ 9.8	22.9	10.8	+12.1
1941	20.8	13.8	+ 7.0	21.4	10.7	+10.7
1942	20.2	14.1	+ 6.1	20.2	11.7	+ 8.5
1942 (1.1-30.VI)	20.6	14.4	+ 6.2	21.3 *(l)*	**13.1** *(l)*	+ 8.2 *(l)*
1943 (1.1-30.VI)	20.3 *(h)*	14.0	+ 6.3	19.4 *(m)*	**12.7** *(m)*	+ 6.7 *(m)*

C. = Census.

E. S. = Estimate of the Epidemiological Intelligence Service of the L. o. N.

O. N. E. = Official national estimate.

(a) Monthly rates based on the period 1934-1938.
(b) Excluding—from November 1940—deaths occurring in war zones.
(d) Deaths.
(e) Excluding deaths in the Services, except those in Africa and Spain from 1935 to 1939.
(f) Monthly rates based on the period 1930-1938.
(g) Quarterly rates based on the period 1934-1938.
(h) Provisional figures.
(i) At the beginning of 1943, about 350,000 civilian Italian workers were in Germany.
(j) Median 1932-1938.
(k) Epidemic in Naples, X.1943-20.I.1944.
(l) 1.I.-30.IV.1942.
(m) 1.I.-30.IV.1943.

Infant Mortality (deaths of infants under 1 year of age, per 1,000 live births).

ITALY

	Yearly rates	I J	II F	III M	IV A	V M	VI J	VII —	VIII J	IX A	X S	XI O	XII N	XIII D
1928-1938 *Max(a)*	*125*	*124*	*118*	*101*	*91*	*92*	*148*		*183*	*164*	*97*	*90*	*93*	*115*
1928-1938 *Med(a)*	*106*	*91*	*97*	*90*	*83*	*83*	*116*		*160*	*137*	*94*	*88*	*82*	*94*
1928-1938 *Min(a)*	*99*	*75*	*89*	*88*	*78*	*73*	*101*		*151*	*126*	*89*	*82*	*80*	*93*
1937	109	**124**	**118**	**92**	78	83	**148**		**178**	137	**95**	83	82	94
1938	106	91	89	88	83	82	**112**		**183**	**164**	**97**	**90**	**93**	**112**
1939	97	**101**	96	90	78	73	92		143	126	**103**	87	79	96
1940 *(b)*....	103		**103**			**96**				126			90	
1941 *(b)*....	**116**		**115**			88				**145**			90	
1942 *(b)*....	108		**104**			**96**				**141**			**98**	

ROME

1928-1938 *Max.*	*88*	*126*	*126*	*85*	*93*	*89*	*169*		*159*	*114*	*88*	*73*	*78*	*114*
1928-1938 *Med.*	*77*	*81*	*75*	*81*	*73*	*67*	*89*		*117*	*73*	*55*	*61*	*64*	*74*
1928-1938 *Min.*	*69*	*49*	*57*	*58*	*58*	*46*	*57*		*84*	*61*	*43*	*48*	*53*	*56*
1937	83	**126**	**96**	58	58	55	**169**		**135**	75	51	61	53	68
1938	79	80	67	**85**	70	**79**	94		117	86	58	**72**	58	79
1939	68	88	77	77	72	55	58		80	65	60	59	56	60
1940	77	78	76	68	78	62	82		111	80	58	64	**76**	**92**
1941	...	78	64	73	64	...	...		...	...	...	...	...	...

General Mortality (rates per 1,000 inhabitants on a yearly basis). *(e)*

ITALY

	Yearly rates	I J	II F	III M	IV A	V M	VI J	VII —	VIII J	IX A	X S	XI O	XII N	XIII D
1928-1938 *Max.*	*16.5*	*21.3*	*28.4*	*23.5*	*15.9*	*14.4*	*14.9*		*18.1*	*17.9*	*14.8*	*14.3*	*14.2*	*18.0*
1928-1938 *Med.*	*14.1*	*16.7*	*16.8*	*15.4*	*13.5*	*12.5*	*13.0*		*14.4*	*13.9*	*11.9*	*12.7*	*13.1*	*14.9*
1928-1938 *Min.*	*13.3*	*13.8*	*15.2*	*14.0*	*12.6*	*11.6*	*11.7*		*13.2*	*12.2*	*11.2*	*11.2*	*12.1*	*13.5*
1937	14.2	**21.3**	16.8	14.0	12.6	12.4	13.2		14.7	13.0	11.9	12.0	12.7	15.5
1938	14.1	**18.5**	16.0	15.1	13.9	**12.9**	12.4		14.4	13.9	11.3	11.4	12.8	14.7
1939	13.4	16.6	16.6	16.1	13.1	11.4	11.3		13.1	12.3	11.5	11.7	12.4	14.3
1940	13.6	17.5	15.9	14.9	14.3	12.1	11.7		12.7	12.4	11.4	11.1	12.1	**16.7**
1941	13.8	17.7	**18.4**	15.0	12.3	11.4	11.8		13.4	13.3	12.0	11.6	13.7	15.2
1942	14.1	**18.6**	**18.1**	13.6	12.4	11.7	12.0		13.5	13.0	12.6	13.0	**14.7**	15.9
1943	...	16.9	15.9	14.6	13.1	11.6	12.0		...	...	...	...	...	...

ROME

1928-1938 *Max.*	*14.6*	*20.3*	*17.0*	*14.0*	*13.7*	*13.3*	*12.7*		*14.8*	*12.4*	*10.5*	*10.5*	*11.8*	*19.7*
1928-1938 *Med.*	*11.8*	*15.5*	*14.7*	*13.3*	*11.4*	*10.2*	*11.3*		*11.6*	*9.8*	*9.1*	*9.8*	*10.5*	*12.7*
1928-1938 *Min.*	*11.0*	*12.0*	*11.9*	*10.8*	*10.8*	*9.6*	*10.1*		*10.7*	*8.5*	*8.6*	*9.1*	*10.0*	*11.7*
1937	11.6	**20.3**	13.1	10.8	11.1	9.9	**12.7**		11.5	9.2	8.8	9.5	10.0	12.7
1938	11.8	**17.1**	14.5	**14.0**	**12.2**	**11.6**	11.2		11.6	9.8	8.6	9.1	10.2	11.8
1939	10.7	14.7	13.7	13.1	10.9	9.3	9.4		10.8	9.1	8.0	9.1	9.9	11.1
1940	10.8	13.6	12.8	11.5	11.9	9.5	9.8		9.2	8.8	8.1	8.9	10.0	**15.4**
1941	10.7	14.1	11.5	10.9	9.8	9.4	10.0		10.9	10.0	9.2	9.2	10.8	12.4
1942	11.7	15.7	15.0	11.3	10.3	10.7	10.5		11.7	10.0	**9.4**	**10.7**	**12.3**	**13.0**

Tuberculosis Mortality (rates per 100,000 inhabitants on a yearly basis).

	Italy					Rome				
	Yearly rates	Quarterly (g) I	II	III	IV	Yearly rates	Quarterly I	II	III	IV
1928-1938 Max.	*128*	*99*	*99*	*89*	*84*	*160*	*185*	*174*	*156*	*155*
1928-1938 Med.	*99*	*92*	*94*	*83*	*82*	*141*	*146*	*142*	*135*	*135*
1928-1938 Min.	*80*	*85*	*84*	*77*	*74*	*125*	*127*	*129*	*108*	*111*
1937	85	92	91	81	78	128	**156**	135	108	114
1938	80	85	84	77	74	125	136	129	125	111
1939	76	84	80	74	67	118	134	123	103	112
1940	75	77	81	71	69	119	118	124	108	126
1941	81	80	85	79	...	...	128	...	...	...
1942	102	...	...	...	...	...	...	...	...	...

Communicable Diseases: Cases notified in Italy.

	Median 1928-1938	1937	1938	1939	1940	1941	1942	1943
Typhoid and paratyphoid fevers	*31,467 (j)*	31,467	**35.724**	25,143	25,300	**43,011**	**45,402**	...
Dysentery	*945 (j)*	**2,044**	**997**	**1.328**	**1.940**	**1.667**	**2.299**	...
Diphtheria	*25,732*	**28,541**	**27.017**	**28,101**	26.218	21.221	25.530	...
Scarlet fever	*18,180*	15,881	13,629	12,045	15,220	12,737	12,655	...
Cer.-sp. meningitis	*606*	**1,037**	**1.286**	**1.330**	**2,783**	**3,836**	**2,327**	...
Poliomyelitis	*839*	**2,307**	**1.923**	**5,030**	**2,027**	**2,467**	1,570	...
Typhus fever	*3 (d)*	2 (d)	—	1 (d)	1 (d)	2 (d)	3 (d)	460 (k)
Smallpox	*2 (d)*	1 (d)	—	—	—	1 (d)	1 (d)	...
Notified cases	*77,774*	81,280	80,576	72,978	73,48.	84,942	89,787	...
Composite epidemic index	100	104.5	103.6	93.8	94.5	109.2	115.4	...

Chapter III.

From this broad survey emerges the fact that the epidemic situation in Europe is, on the whole, favourable and, with the possible exception of typhus fever in certain parts of Eastern and Southern Europe, has not been influenced so far by the deterioration of the resistance of the population which might have been expected from the widespread malnutrition. The spread of epidemic diseases appears to have been due far more to migrations, intranational and international, with the resulting increase in contact infection, than to the nutritional situation.

After first briefly reviewing the general demographic situation, one may therefore consider separately, first, the epidemic or sanitary situation and, secondly, the general health and nutritional situation of the peoples of Europe. These two situations call for different forms of relief—medical and nutritional.

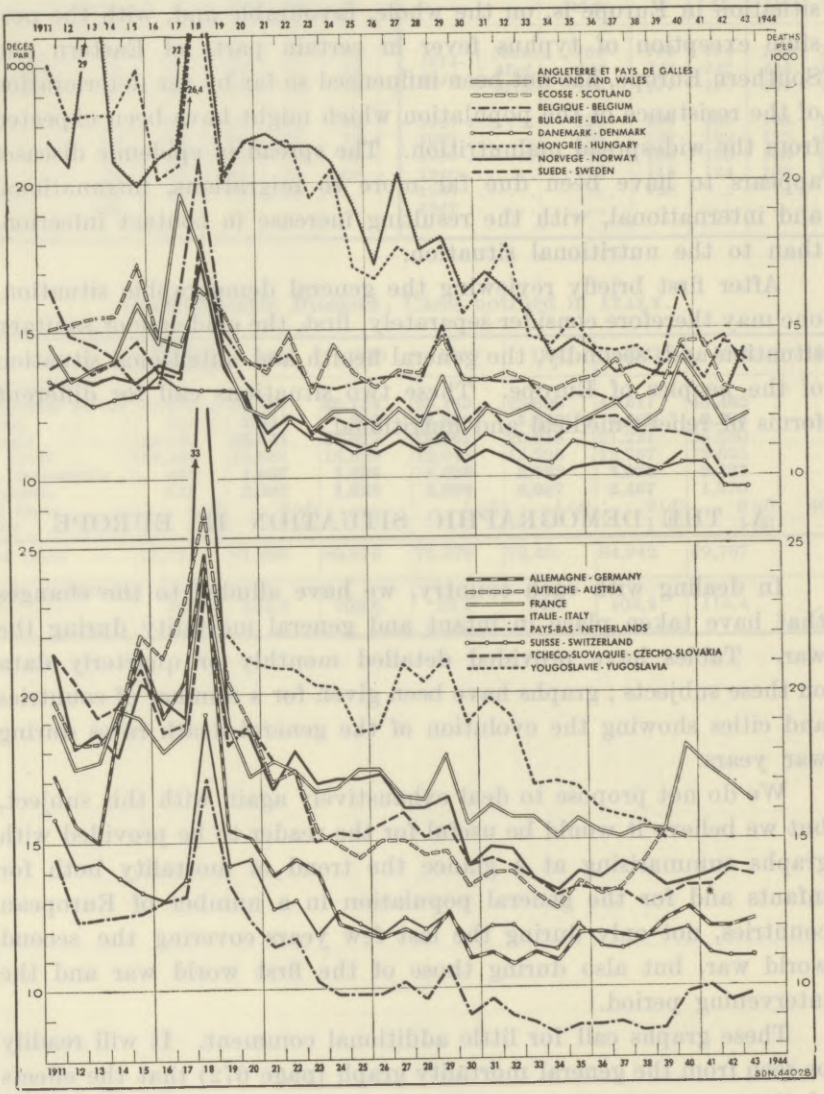
A. THE DEMOGRAPHIC SITUATION IN EUROPE

In dealing with each country, we have alluded to the changes that have taken place in infant and general mortality during the war. Tables have provided detailed monthly or quarterly data on these subjects ; graphs have been given for a number of countries and cities showing the evolution of the general death rates during war years.

We do not propose to deal exhaustively again with this subject, but we believe it would be useful for the reader to be provided with graphs summarising at a glance the trend of mortality both for infants and for the general population in a number of European countries, not only during the last few years covering the second world war, but also during those of the first world war and the intervening period.

These graphs call for little additional comment. It will readily be seen from the general mortality graph (page 672) that the effects of the war on civilian mortality have been comparatively small in belligerent countries, except those which have suffered from occupation, and have been negligible in neutral countries. As a rule, infant mortality has been still less affected (*cf.* page 673).

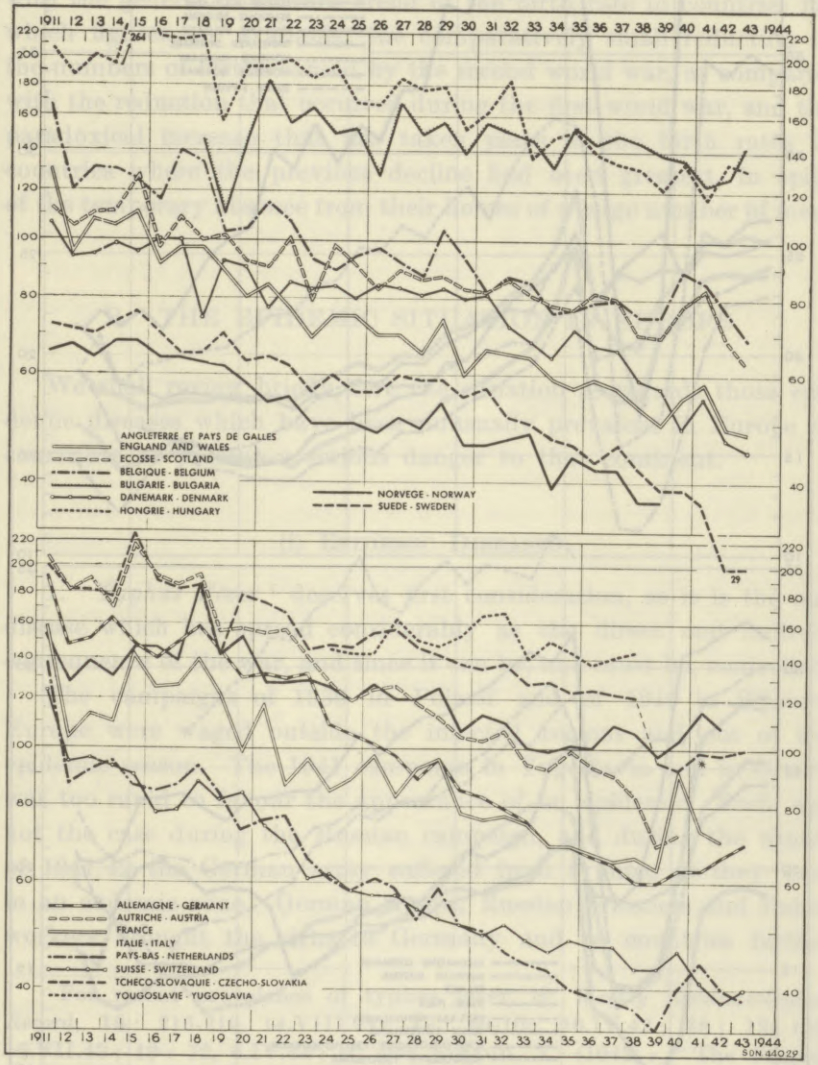
General Mortality, per 1,000 Inhabitants, in Various Countries of Europe, 1911-1943.



* "Bohemia and Moravia".

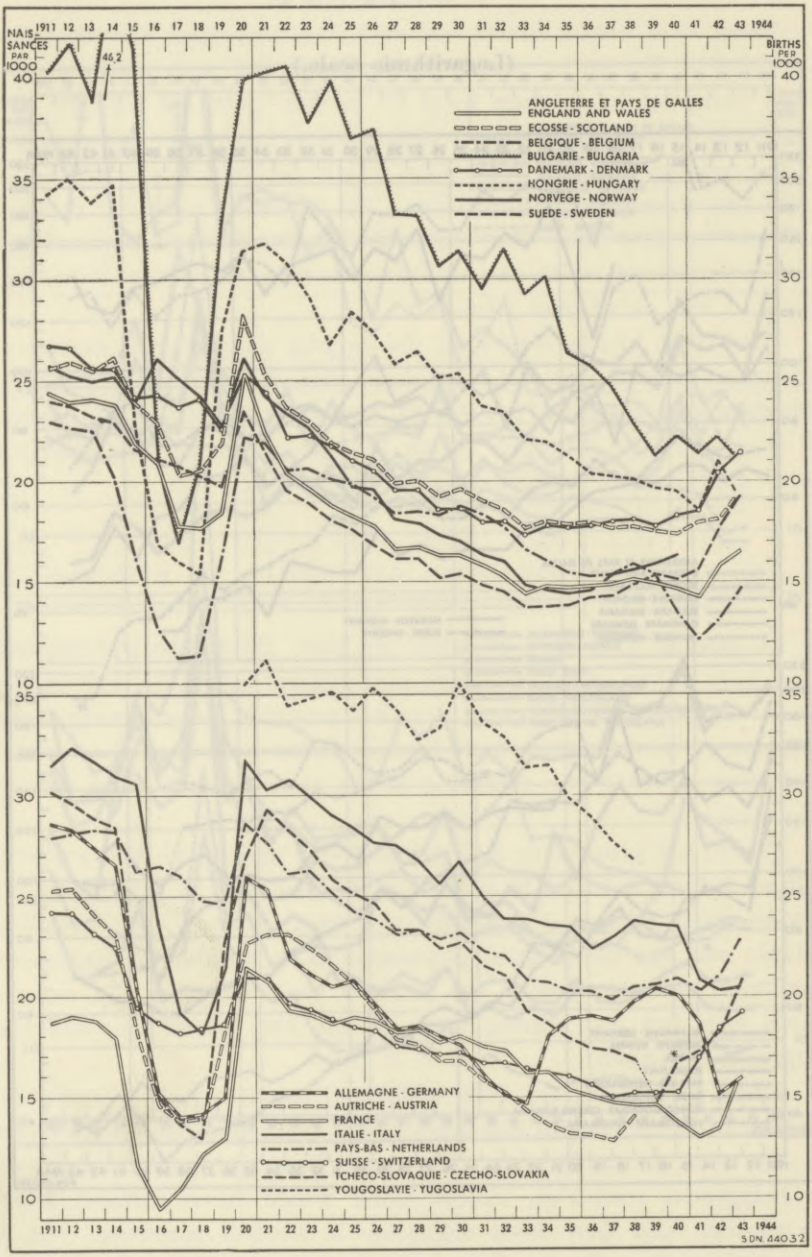
Infant Mortality, per 1,000 Live Births, in Various Countries of Europe, 1911-1943.

(Logarithmic scale.)



* "Bohemia and Moravia".

Birth Rates, per 1,000 Inhabitants, in Various Countries of Europe, 1911-1943.



(2) " Bohemia and Moravia ".

It must be emphasised that the graphs unfortunately do not cover those countries which, like Poland, Greece, Yugoslavia, and perhaps the U.S.S.R., have suffered most acutely from the consequences of the war.

Natality deserves special treatment, and we cannot do the subject justice in this article. The graph on page 674 shows, together with the general downward trend of the birth rate in countries for which figures are available, the comparatively small reduction in the numbers of births caused by the second world war, as compared with the reduction that occurred during the first world war, and the paradoxical increase that has taken place in the birth rates in countries where the previous decline had been greatest, in spite of the temporary absence from their homes of a large number of men.

B. THE EPIDEMIC SITUATION IN EUROPE

We shall review briefly here the situation as regards those epidemic diseases which have been unusually prevalent in Europe or constitute at present a serious danger to that continent.

(i) Epidemic Diseases

1. *Typhus Fever* [1] deserves first consideration, as it is the one disease which has spread considerably as the direct and indirect consequence of the war, and since it can be, and must be, controlled.

The campaigns of 1939 in Poland and of 1940 in Western Europe were waged outside the infected regions and out of the epidemic season. The 1941 campaign in Yugoslavia and in Greece was too rapid to favour the appearance of an epidemic. Such was not the case during the Russian campaign, and during the winter of 1941/42 the German army suffered from typhus, as they were in an endemic zone. German troops, Russian prisoners and Polish workers brought the virus to Germany and to countries farther

[1] For recent prevalence of typhus fever, *cf. Weekly Epidemiological Record*, **16**: 216-219, 14.VIII.41; **17**: 99-105, 16.IV.42; **18**: 124-126, 15.VII.43; **19**: 72, 6.IV.44; *cf.* also Biraud, Y. (1943): "The Present Menace of Typhus Fever in Europe ..." *Bull. Health. Organ.* L.o.N.: **10**, pages 7-14; a map of the incidence of typhus is given on pages 8 and 9 of this article.

west, and it is owing to the rarity of the body-louse in these countries that only sporadic cases occurred and no true secondary epidemics. Where louse infestation was more common, in the Balkans and Hungary, contact with the eastern front resulted in the dissemination of the disease. In Poland the general destitution favoured infestation by lice and consequently the spread of typhus fever, and also the revival of old, apparently cured, infections.

This latter process caused the appearance of the disease among Russian and Polish workers in Germany.

Systematic delousing prevented epidemics arising from these cases. At present the prevalence of the disease is fairly high in its endemic haunts in Poland and Roumania, epidemic in most of Croatia and sporadic in Serbia, Bulgaria, Hungary and Spain. Its prevalence in the U.S.S.R. is not known, but it is probably not excessive, since it has not checked the Russian war effort.

Moreover, it should be remembered that, since the discovery of the vector of typhus fever, only beaten and disorganised armies are unable to apply the delousing methods which suffice to control the spread of the disease. In forecasting the possibilities of the future extension of the disease, it should be realised that, while after the first world war refugees and prisoners-of-war migrated from highly infected to uninfected territories, the reverse will probably be the case after this war ; thus the chances of large epidemic outbreaks will be reduced to practically nil, the more so if the homeward migrations do not take place during the winter season—*i.e.*, the seasonal peak of the disease in endemic areas.

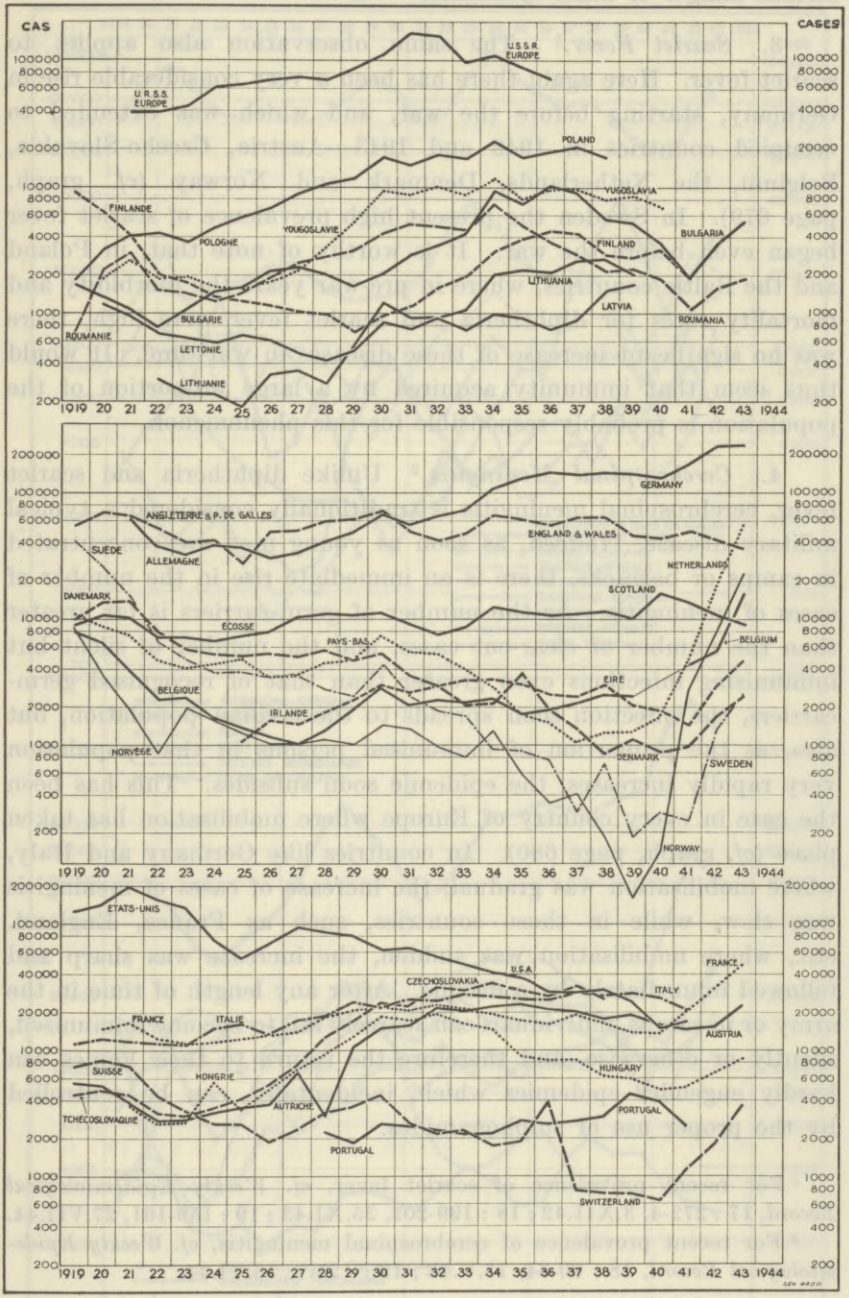
Any systematic delousing carried out as a prophylactic measure against typhus fever will at the same time be effective against the spread of *Wolhynian fever* (the trench fever of the first world war on the western front) caused by *Rickettsia quintana* and which, like typhus fever, is louse-borne.

2. *Diphtheria* [1] is the disease which has shown by far the greatest increase during the war years (*cf.* graph, page 677), particularly in Germany, and also in Czecho-Slovakia, France, Belgium, the Netherlands, and Norway. The increase was greatest in those countries where the disease had in recent years reached the lowest level, *viz.*, the Netherlands and Norway, and where in consequence

[1] For recent prevalence of diphtheria in Europe, *cf. Weekly Epidemiological Record*, 18 : 15-20, 28.I.43 ; 19 : 153-5, 20.VII.44.

Cases of Diphtheria notified in Various Countries of Europe, 1919-1943.

(Logarithmic scale.)



the proportion of receptive individuals was greatest. As the majority of adults throughout Europe are immune to diphtheria, there is no serious danger of mass epidemics.

3. *Scarlet Fever.*[1] The same observation also applies to scarlet fever. Here again there has been a very considerable rise in Germany, starting before the war, and which was extended to occupied countries in 1942 and 1943—Austria, Czecho-Slovakia, Belgium, the Netherlands, Denmark and Norway (*cf.* graph, page 679). In Sweden the present high prevalence of scarlet fever began even before the war. It is worthy of note that, in Poland and the Baltic countries, where in pre-war years the morbidity and mortality rates for diphtheria and scarlet fever were high, there was no significant increase of these diseases in war-time. It would thus seem that immunity acquired by a large proportion of the population is probably responsible for this phenomenon.

4. *Cerebrospinal Meningitis.*[2] Unlike diphtheria and scarlet fever, cerebrospinal meningitis is traditionally considered a typical military disease. Indeed, as soon as young men are concentrated in camps or barracks, there is an immediate rise in the number of cases of meningitis. As the number of germ-carriers is far greater than the number of clear-cut cases, and the number of silent but immunising infections even greater than that of recognised germ-carriers, the infection soon spreads to the civilian population, but also, as the proportion of immunised persons in that population very rapidly increases, the epidemic soon subsides. This has been the case in every country of Europe where mobilisation has taken place (*cf.* graph, page 680). In countries like Germany and Italy, where mobilisation was gradual, the increase of cases of meningitis was slow, while in those countries, such as France, England, etc., where mobilisation was sudden, the increase was sharp and followed immediately by a decline. After any length of time in the army or in camps, individuals can scarcely fail to become immunised, silently or otherwise, and therefore the return to their homes can hardly engender epidemics which, incidentally, can be controlled by the proper use of sulphonamides.

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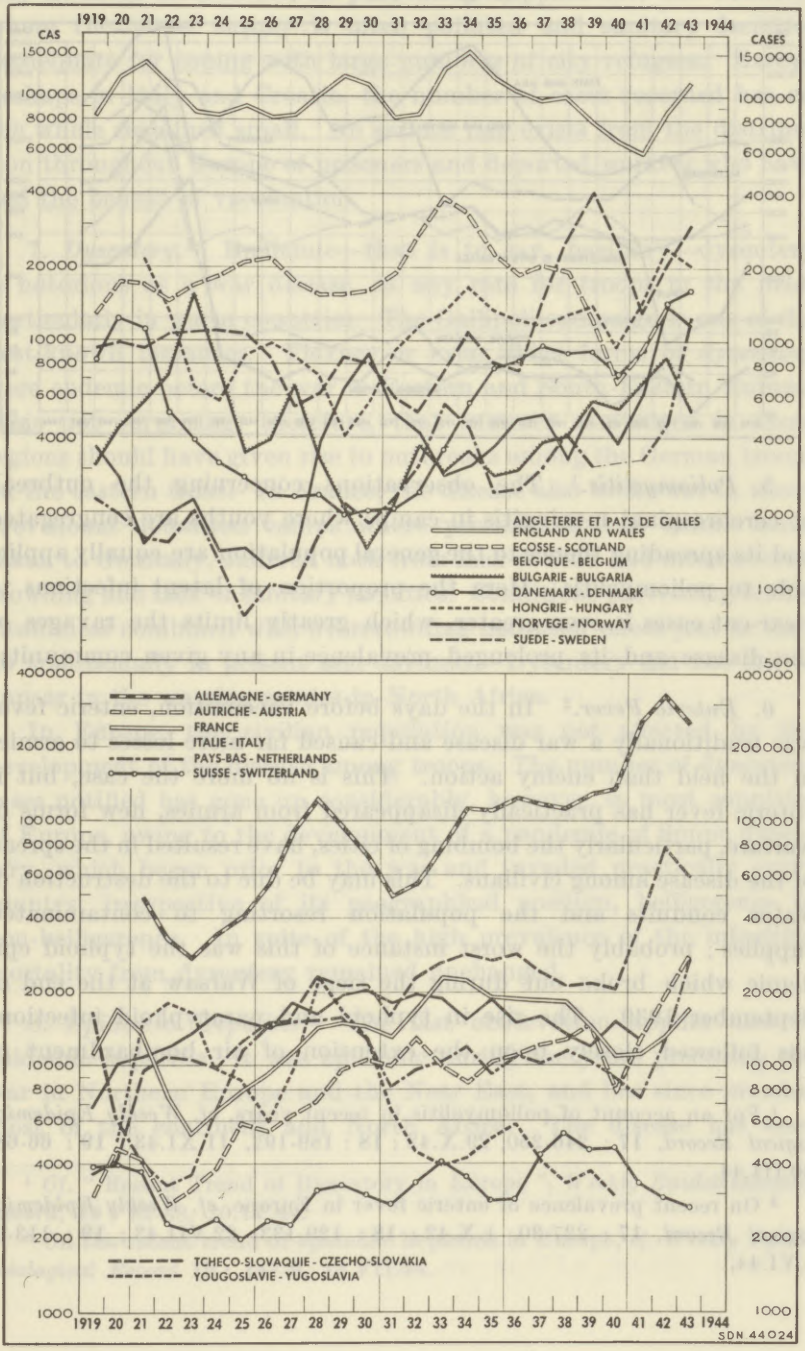
[1] For recent prevalence of scarlet fever, *cf. Weekly Epidemiological Record*, **17** : 272-4, 3.XII.42 ; **18** : 199-202, 25.XI.43 ; **19** : 159-161, 27.VII.44.

[2] For recent prevalence of cerebrospinal meningitis, *cf. Weekly Epidemiological Record*, **18** : 80-84, 13.V.43 ; **19** : 125-7, 15.VI.44.

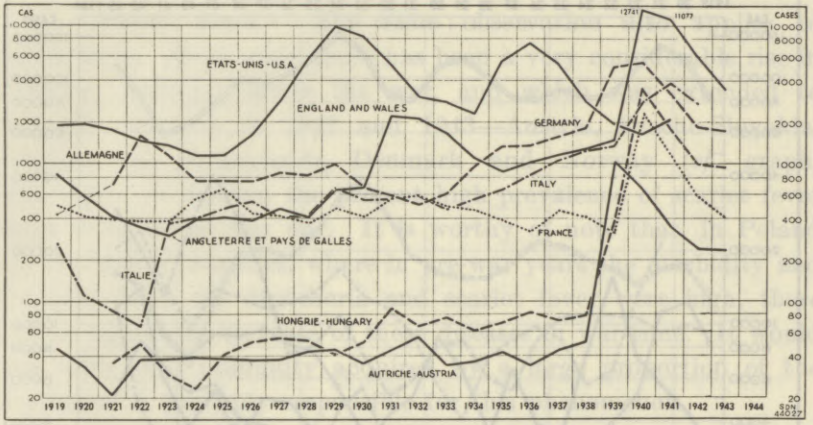
Cases of Scarlet Fever notified in Various Countries of Europe, 1919-1943.

(Logarithmic scale).



Cases of Cerebrospinal Meningitis notified in Various Countries of Europe, 1919-1943.

(Logarithmic scale.)



5. *Poliomyelitis.*[1] The observations concerning the outbreak of cerebrospinal meningitis in camps where youths are congregated and its spreading thence to the general population are equally applicable to poliomyelitis. Here the proportion of latent infections to clear-cut cases is even greater, which greatly limits the ravages of the disease and its prolonged prevalence in any given community.

6. *Enteric Fever.*[2] In the days before vaccination, enteric fever was traditionally a war disease and caused far more losses to armies in the field than enemy action. This is no more the case, but if enteric fever has practically disappeared from armies, new forms of warfare, particularly the bombing of cities, have resulted in the spread of the disease among civilians. This may be due to the destruction of water conduits and the population resorting to contaminated supplies ; probably the worst instance of this was the typhoid epidemic which broke out during the siege of Warsaw at the end of September 1939. The rise in typhoid and paratyphoid infections has followed closely upon the extension of air bombardment of

[1] For an account of poliomyelitis in recent years, *cf. Weekly Epidemiological Record*, **17** : 246-250, 29.X.42 ; **18** : 189-192, 11.XI.43 ; **19** : 66-68, 30.III.44.

[2] On recent prevalence of enteric fever in Europe, *cf. Weekly Epidemiological Record*, **17** : 227-30, 1.X.42 ; **18** : 129-133, 22.VII.43 ; **19** : 113-6, 1.VI.44.

cities in England in 1940 and 1941, and in Germany from 1942 onwards. Bombardments may also have an indirect effect in sending city dwellers accustomed to pure water supplies into the country, where the water supply is often polluted and sanitary facilities inadequate for coping with large numbers of city refugees. Except perhaps in Italy and Croatia, the number of cases recorded has on the whole remained small. No serious risk exists from the distribution throughout Europe of prisoners and deported workers who have had the benefit of vaccination.

7. *Dysentery.*[1] Epidemic—that is to say, bacillary—dysentery is notorious as a war disease, at any rate for troops in the field, particularly in warm countries. The Gallipoli campaign is one of the best-known instances. Flexner or even Shiga forms of dysentery were endemic before the war in Eastern and South-Eastern Europe. It is not surprising, therefore, that summer campaigns in those regions should have given rise to outbreaks among the German troops on the eastern front. In France, the disease also broke out in many provisional detention camps where prisoners-of-war, before being taken to Germany, suffered both from lack of food and intense overcrowding and lack of sanitary facilities. Conditions preventing bodily cleanliness combined with overcrowding had their effect just as they do traditionally in prisons and asylums. Dysentery did not fail to appear in the warring armies in North Africa.

In Europe, the civilian population was not affected by the development of the disease among troops. The number of dysentery cases notified has gone up considerably, however, in most countries of Europe, owing to the development of a pandemic of Sonne dysentery, which began prior to the war and invaded practically every country, irrespective of its geographical position, belligerence or non-belligerence. In spite of the high prevalence of the infection, mortality from dysentery remained unchanged.

8. *Epidemic Hepatitis.*[2] The last observation applies also to epidemic hepatitis ; it was epidemic during the years preceding the war in Northern Europe and the Near East, and has since invaded most of the continent and North Africa. The disease has been

[1] *Cf.* " Recent Trend of Dysentery in Europe ", *Weekly Epidemiological Record,* **19** : 82-88, 20.IV.44.

[2] On the recent trend of epidemic hepatitis in Europe, *cf. Weekly Epidemiological Record* **19** : 147-9, 13.VII.44.

particularly widespread among troops, especially during the Greek and Libyan campaigns. The fact that it is a droplet infection with a long incubation, and that a large number of cases escape diagnosis makes prevention very difficult. It is fortunate therefore that, in spite of the high prevalence of the disease, fatality has been very low.

9. *Epidemic Influenza* [1] deserves but a brief reference. There is a widespread opinion that epidemic influenza is a "war disease", in spite of the fact that, of the dozen pandemics of the disease which have prevailed in Europe during the last century and a-half, only two occurred during wars (1918 and 1943/44) and even these were not limited to belligerent countries. The fact that an influenza wave swept over the United States and all of Western Europe and Germany, from November 1943 to February 1944 (*cf.* graph, page 683), makes a recurrence of any serious outbreak improbable within the next year or two.

10. *Smallpox.*[2] In spite of the existence of smallpox in its severe form on what may be termed the fringes of Europe—that is to say, in North Africa, the Near East and Turkey, whence the disease invaded a Greek district in 1943—and in the Iberian peninsula, the Continent proper remained practically free from the disease, owing to the generalised practice of vaccination (*cf.* graph, page 684). The fact that this practice was not general in Eastern Europe and some of the Balkan countries prior to the first world war was largely responsible for the epidemics that prevailed there at its close.

11. *Cholera.* It is gratifying that cholera, which from 1913 to 1923 caused serious epidemics in Eastern and South-Eastern Europe, has not been reintroduced to the European continent since that date, or into North Africa.

12. *Yellow Fever.* It is even more satisfactory to note the fact that yellow fever has not been brought to the shores of the Mediterranean and the Near East, in spite of the greatly developed air and road traffic from 1941 to 1943 across the African continent,

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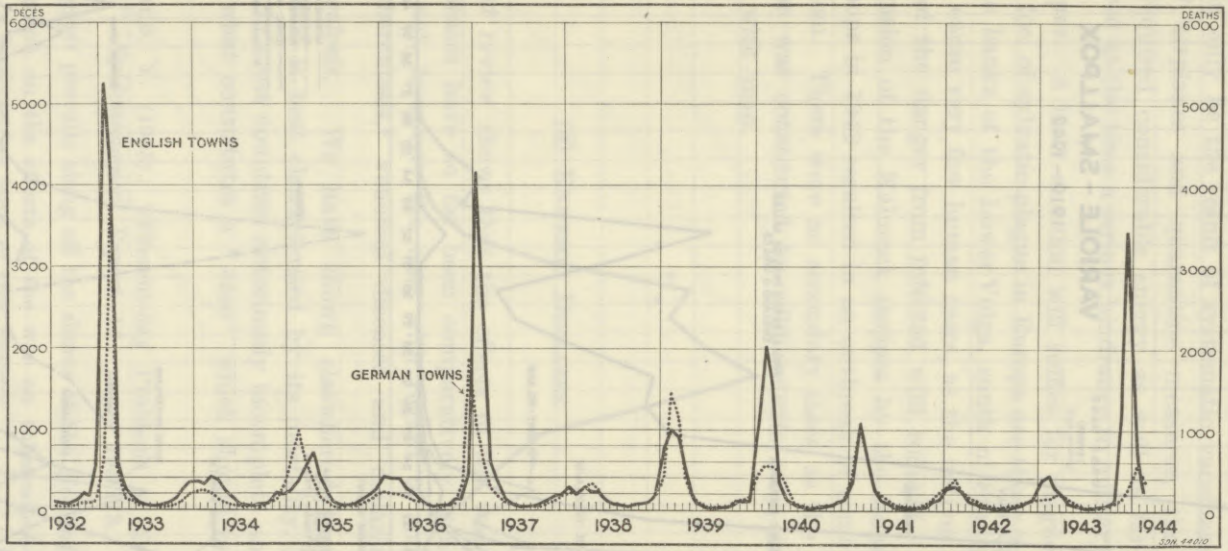
[1] For influenza during the war years, *cf. Weekly Epidemiological Record* 18 : 212-215, 16.XII.43 ; 19 : 118-122, 8.VI.44.

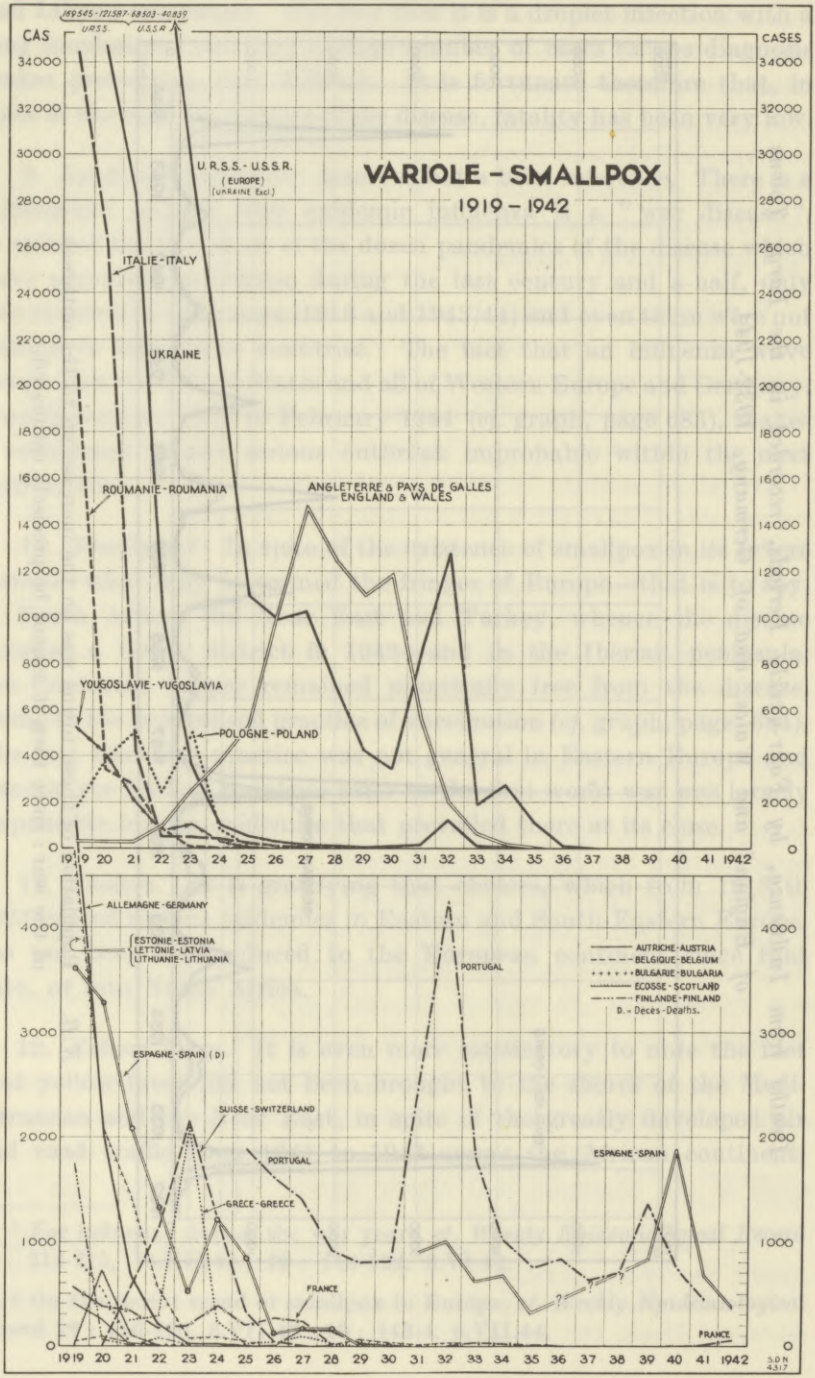
[2] On the recent trend of smallpox in Europe, *cf. Weekly Epidemiological Record* 18 : 116-120, 8.VII.43 ; 19 : 143-4, 6.VII.44.

Deaths from Influenza, by Four-weekly Periods, recorded in the Large Towns of England and Wales, and of Germany, 1932-1944.



N.B. — Population of these towns was on the increase up to the outbreak of the war : since then it cannot be precisely estimated.

Cases of Smallpox notified in Various Countries of Europe, 1919-1942.



from the endemic areas of West Africa [1] and the Sudanese belt to Egypt and other highly infectible [2] areas of North Africa.

This can only be the result of systematic vaccination, disinsectisation of airplanes and quarantine measures, the application of which required considerable energy as well as far-sightedness, as it involved at the time a serious hindrance to military operations.

13. *Plague.* A brief mention will suffice for plague. The only permanent foci of sylvatic plague in Europe are those in the steppes on both the banks of the Lower Volga, south of Stalingrad. Normally they cause very few human cases, as the natives of this area are aware of the danger from infected wild rodents.

The invasion of the Kalmuck steppes by the Roumanian and German troops in 1942 resulted in an outbreak of some forty cases amongst them. There were no secondary cases, as the reservoir of the infection was constituted by wild rodents only, and not rats associating with man.

* * *

(ii) Endemic Diseases

This brief review shows that the effects of the war as regards *epidemic* diseases have so far been comparatively small.

Such is not, however, the case with regard to some *endemic* diseases, tuberculosis,[3] venereal diseases and malaria.

1. *Tuberculosis.* We have shown elsewhere [4] that tuberculosis prevalence is best characterised by its mortality, since notifications are in most countries notoriously incomplete and opinions differ as to what constitutes a " case " which should be reported.

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[1] *Cf.* Biraud, Y. (1935) : " Present-day Problems of Yellow Fever Epidemiology ". *Epidemiological Report,* **14** : pages 103-173.

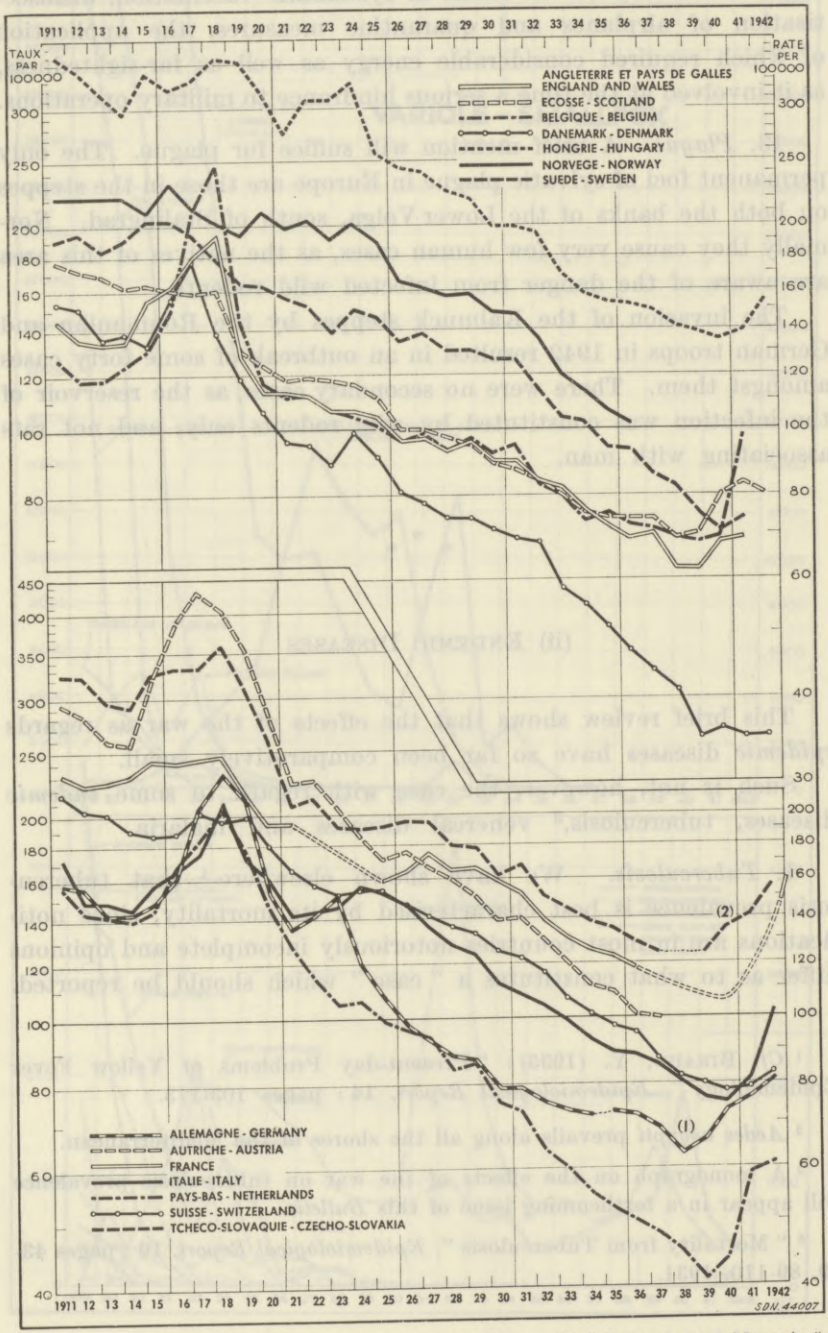
[2] *Aëdes aegypti* prevails along all the shores of the Mediterranean.

[3] A monograph on the effects of the war on tuberculosis prevalence will appear in a forthcoming issue of this *Bulletin.*

[4] " Mortality from Tuberculosis ". *Epidemiological Report,* **10** : pages 43-59, 89-110, 1931.

Deaths from Tuberculosis, per 100,000 Inhabitants, in Various Countries of Europe, 1911-1942.

(Logarithmic scale.)



(1) Based on deaths notified to the sanitary police. (2) " Bohemia and Moravia ".

Tuberculosis mortality is a sensitive index of prevailing social and economic conditions, which influence it—above all, conditions of nutrition and work.[1]

It was to be expected that the war, influencing as it does both of these factors, should have resulted in a significant increase in tuberculosis mortality. Such has indeed been the case.

In appraising the significance of this increase, one should consider not so much the actual difference in rates per 100,000 as the *percentage* of rise, since the level reached by the death rates in different countries before the war was far from uniform.

The increase due to longer hours of work has been apparent in all countries at war, particularly in industrial cities. It has taken place even in cities such as Detroit (Michigan), where war industries were very active prior to the entry of the United States in the war and where there was no food shortage.

This " industrial " increase of tuberculosis is apparent in England and Wales, Scotland and Germany. Food shortage, which affects in present circumstances a far larger proportion of the population than does industrial fatigue, influences far more strongly the tuberculosis death rates in occupied countries.

We have seen that this " nutritional " increase in tuberculosis mortality was particularly marked in Belgium, in France, and in the Netherlands. It was less but still noticeable in "Bohemia and Moravia", Ireland, Italy, Spain and even in Hungary (in Budapest), which shows that not only can tuberculosis due to malnutrition make itself felt when food shortage is general, but even when it is limited to certain groups of the population such as town dwellers, or the poorer classes, or certain racial groups against whom economic discrimination is practised.

Although no routine figures are available, information from welfare organisations indicates that tuberculosis mortality has also risen markedly in Greece, Yugoslavia and Poland and, in this latter country, particularly in the towns.

On the other hand, the pre-war downward trend of the disease has continued unchecked in Denmark, Sweden and Switzerland, where neither food shortage, nor industrial over-activity existed (*cf.* graph, page 686).

[1] On the factors of tuberculosis mortality, see BIRAUD, Y. (1930) : " La mortalité tuberculeuse et son évolution — Aperçu géographique et épidémiologique ". *Rev. de Phtisiol.* **11**, pages 37-63.

Finally, reference must be made to the camps of prisoners-of-war, political deportees and forced labour in Germany. There over-exertion and food insufficiency combine their effects with dire results, as is apparent from the excessive number of deaths taking place on the spot and the large numbers of hopeless cases among the prisoners released as permanently unfit for labour, and accordingly sent home, where they constitute sources of infection.

In view of the great concern displayed concerning the future consequences of the war increase of tuberculosis, it must be borne in mind that, in most cases, the tuberculosis death rates, in spite of their recent rise, are still well below not only those levels reached during the first world war but even the period immediately following it. As in all countries of Central and Western Europe the rates subsequently went steadily down, the present war rise has brought them to what they were 10 or at most 15 years ago.

It can reasonably be expected that elimination of the causes of the present rise will result in a spontaneous fall in the rates. This fall was extremely rapid in 1919/20, and again after the period of inflation in Austria and Germany in 1924. The fact that the armament against tuberculosis has been greatly improved in all countries since that time should contribute to the speedy return of the pre-war trend of the tuberculosis curve.

Undoubtedly, the economic rehabilitation of European countries is the most effective way of bringing about a reduction in tuberculosis. Nevertheless, it would seem indicated to give special attention to the millions of foreign prisoners and labourers held in Germany, in view of the particular conditions of exposure and debilitation to which they have been subjected. Systematic diagnostic examination, particularly with radiophotography, would be desirable in their case. This might be carried out before their demobilisation, on reaching their own countries if these are properly equipped for the task, or it might be done in Germany itself in the case of those who would not be likely to undergo such examination at home. Treatment itself would take place in their home countries, for obvious psychological reasons.

2. *Venereal Diseases.* Venereal diseases have spread considerably since the beginning of the war, not only in belligerent countries—France, England, the United States—but also in neutral countries which have taken mobilisation measures, such as Sweden and Switzerland.

Promiscuous sexual intercourse, which is the obvious cause of this increase in venereal morbidity, is itself the result of the forced disruption of family ties by mobilisation, deportation, internment and, in some countries such as France, Greece, Poland, and Yugoslavia, the life of outlawry which large numbers of men are forced to lead as partisan soldiers. In addition to the obvious psychological cravings in the wives, protracted absence of their husbands often causes financial difficulties which are a further incentive to unfaithfulness. Special enquiries have indeed shown that the increase in venereal diseases is not confined to the mobilised men or women.

In Germany, the fact that a survey [1] among German civilians did not reveal an increase of venereal diseases in 1940, as compared with the surveys of 1934, does not signify that such an increase has not taken place in the mobilised part of the population—the younger part, more liable to exposure both at home and in occupied areas. Systematic organisation and control of brothels by German military authorities in these areas constitute an unreliable protection against contamination. Probably the most dangerous sources of contamination in, and for, Europe are the camps of foreign conscripted labourers in Germany. They include not only millions of men deprived of nearly every form of healthy recreation, but large numbers of women, including many professional prostitutes.[2] Promiscuous intercourse between male and female foreign labourers was not only possible but in many camps deliberately fostered. Although figures are not available, the moral and physical consequences of this policy can easily be imagined and probably constitute one of the most urgent health problems to be dealt with during the armistice period.

It would obviously be preferable from every point of view to discover infected men and women on the spot, and to give them, in special camps, the benefit of a first course of treatment in the case of syphilis, or of an effective sulphonamide cure in the case of gonorrhœa, rather than to let them return and contaminate their

[1] Werr, F., & Gottschalk, H. (1943): "Ergebnisse und Zukunftaufgaben der Geschlechtskrankenstatistik im Deutschen Reich". *Öffentl. Gesundheitsdienst.* 9, pp. B 20-B 31, B 42-B 50.

[2] They constituted a majority of the women who answered the call of the Vichy Government for volunteer workers for Germany in 1942.

families, with the consequence that it would later be necessary trace and treat a much larger number of cases.

Another reason for the adoption of this policy is the fact that in many countries the organisation for combating venereal disease, particularly in rural areas, is far from adequate to accomplish this task.

3. *Malaria.* Malaria deserves special mention as a disease of which the prevalence is favoured by war conditions. It is true that we possess very little accurate information about its recent development, but experience of the first world war and its aftermath, coupled with our knowledge of recent events, lead us to expect, as a result of the present war, a marked extension of malaria and a rise of morbidity and mortality from that disease in endemic areas.

Movements of non-immune persons through malaria-infected areas, the arrival of plasmodium carriers in malaria-free but readily infected regions, are probably the main danger.[1]

The interruption of anti-larval measures because of the mobilisation of the specialised staff, the systematic flooding, for military reasons, of drained or reclaimed areas, such as took place in the Pontine marshes and in the Netherlands, the dearth of physicians available for the civilian population, especially in troubled areas such as Croatia, the shortage or even lack of quinine owing to the Japanese occupation of the cinchona-producing areas (Java), are all contributory factors to an extension of malaria. Malnutrition, which is breaking down acquired resistance to the disease, may also lead to an increase in its prevalence, or even to the revival of extinguished endemic foci.

The lack of precise statistics concerning these phenomena is readily explained by the fact that malaria is essentially a disease of rural and poor areas, where physicians are scarce even in peacetime, and to whom only a small proportion of malaria patients ever resort. Notification of cases is consequently the exception rather than the rule.

[1] The latter cause was responsible for an epidemic of " tropical " malaria as far north as the Murmansk coast in 1919.

C. THE NUTRITIONAL SITUATION IN EUROPE

From information available regarding the dietary of European populations during war years, and its effect on public health and mortality, it is apparent that there is no uniformity in the food and nutritional situation of Europe. Conditions differ profoundly from one country to another, and even more from one area to another within a given country. This is particularly true of those countries where food scarcity prevails. This makes it difficult accurately to classify countries according to the extent of this scarcity and of its effects on public health. They can, however, be roughly classed in three groups :

(1) Those where the food situation has not affected public health ;

(2) Those where food shortages have affected public health ;

(3) Those where actual famine has occurred.

(1) Countries where the Food Situation has not affected Public Health

The food and nutritional situation can be considered as quite satisfactory in *Portugal*, *Sweden* and *Switzerland*, which have been able to supplement their own, unhampered, agricultural production by some foreign imports. The situation was also satisfactory in *Germany* and *Austria*, and to a lesser degree in *Italy* until the end of 1942, where local production was supplemented by requisitioning and other methods of obtaining goods by compulsion from occupied areas, vassal and satellite States.

In *Slovakia*, *Hungary*, *Roumania* and *Bulgaria*, which are agricultural countries, there was relative abundance of food in spite of increased exports, and only comparatively small groups suffered from want in the cities owing to racial discrimination or merely to economic disturbance.

Among occupied countries, *Denmark* enjoyed a privileged situation, owing to her rich agricultural meat and dairy production, and the continuation, relatively speaking, of her administrative independence.

Bohemia and *Moravia* occupy a position somewhat comparable, but on the whole not so favourable, as, in pre-war days, the standard of living of the Czech population was not so high as that of the Danes.

In none of the above-mentioned countries have malnutrition and deficiency disease been reported in the medical literature,[1] nor has there been any increase in tuberculosis or infant mortality ascribable to food shortages, nor any increase in general mortality which cannot be ascribed to the higher mean age of the population owing to mobilisation of the young.

The same remarks apply also to the *United Kingdom*, where social and economic improvements, the disappearance of unemployment, higher wages and efficient food production and distribution under Government control, have apparently more than compensated for the reduction in food imports due to war restrictions of sea traffic.

Although since 1939 vital statistics are lacking concerning the *Baltic States*, the available information regarding them suggests that the health of their inhabitants has not deteriorated as a result of lack of food, notwithstanding occasional shortages in the towns, thanks to the rural and agricultural character of these countries.

(2) Countries where Food Shortages have affected Public Health

In considering those countries of Europe where food restrictions have been severe enough to cause malnutrition and even influence mortality, it must be borne in mind that there are even greater variations between urban and rural areas, and between the various economic strata of urban populations, than between nations, as regards food scarcity and its consequences.

A breakdown of communications and of the administrative control of State authorities, such as has taken place in France in

[1] We allude here to the appearance of malnutrition in population groups which were free from it, or at least from severe manifestations of it, in peace-time. Except, of course, where the disappearance of unemployment and the extended intervention of the State in food distribution have improved the lot of the "under-privileged" classes, the war cannot be expected to suppress the malnutrition which existed in *all* countries before the war, in certain categories at least of the population.

1943 and 1944, only increases the contrast between the lot of the countryman and that of the city dweller.

These internal and fundamental differences make it difficult to classify countries according to the relative stringency of the food and nutritional situation prevailing in them. A further difficulty lies in the fact that, for most of them, we possess information regarding food shortage, but no adequate vital statistics showing its detrimental effects on public health. There are only too many proofs—statistical as well as clinical—of these effects in *France* [1] and *Belgium*, chiefly in the towns and particularly in the largest cities. The cutting-off from their area of supply of the largest French cities in the summer of 1944, and the exhaustion of local stocks, have merely brought to a climax an extremely serious food and nutritional situation. The gradual requisitioning and, later, the destruction of communication facilities which was more acutely felt in France, where distances were greater and the network of railways less dense than in Belgium, have resulted in an increasing disparity between the food supply in country districts and that in the cities.

In the *Netherlands*, it seems that the situation in the towns has been somewhat less serious than in either France or Belgium.

In *Norway*, which is not agriculturally self-supporting, requisitions, restriction of fishing, and the stoppage of imports have made the food situation definitely bad in towns. One may question the reliability of the published vital statistics for Oslo, which appear favourable, seeing that the censorship prevents the publication of those covering the whole country.

Finland is in a similar situation. There, again, local agriculture does not normally cover her needs and mobilisation has reduced production. Imports from Germany have not apparently made good the deficiency and appeals have been made to international relief organisations, chiefly for food for children. There, again, suppression of statistics by censorship prevents us from gauging the possible effects of the food shortage on public health.

[1] It might have been expected that the food situation would be more favourable in France than in Belgium, on account of the more agricultural character of the former country. It would appear that this advantage has been offset partially by the effects of the policy of " collaboration " practised by the Vichy Government, the requisitioning of French products having been rendered easier through its agency from the end of 1940 to 1942.

In *Poland* the food situation has varied enormously according to place and race, ranging from practical sufficiency in certain agricultural areas to acute want in cities and actual famine in ghettos. The effects on public health have accordingly been varied, ranging from mere deficiency diseases (such as rickets) to mass starvation. Relief efforts, concentrated chiefly on children, have been greatly reduced in their efficiency owing to opposition by the occupation authorities.

In *Yugoslavia* also considerable local differences exist in the stringency and effects of the food shortage. This shortage has been fairly general, particularly in the south-west, where guerilla warfare has combined its effects with those of the barrenness of the country.

In the absence of published vital statistics for the *U.S.S.R.*, we are ignorant of the effects on the public health of the food shortage that resulted from the German seizure of a large part of its most fertile agricultural land, including the whole of the famous black-earth area.

We know something of the misery endured by the besieged population of Leningrad, but we can only surmise as to the condition of the millions of refugees evacuated from the war zone. The hugeness of the food stores sent to the U.S.S.R. under lend-lease agreements is an indication of the extent of the need.

Among the countries affected by food shortage special mention must be made of *Spain*.

There food shortage was severe during the civil war in a large part of the country, indeed acute for two winters in Madrid and among the large number of prisoners. The economic and social consequences of the civil war were prolonged and aggravated by the European war, so that in 1941 the serious nature of the nutritional situation of the country appeared to be second only to that of Greece. Improvement has since taken place.

(3) Countries where Famine has occurred

While actual famine and resulting starvation have been experienced throughout Europe by many population groups (inmates of gaols, internment camps, prisoners-of-war camps, Jews in ghettos, inhabitants and garrisons of besieged cities); while severe hardships, considerable loss of weight, and deficiency diseases have been endured by even larger numbers of people in cities of occupied countries, lack

of food has been nowhere as acute and general on a nation-wide scale as it has been in *Greece*.

There, in peace-time, only a small proportion of the staple foods necessary for the population could be grown. Invasion, the cutting-off of outside supplies and large-scale requisitioning resulted in actual famine during the winter of 1941/42. It visited the towns first and extended even to the country. The limited relief supplies allowed to enter the country could but transform the famine condition into one of chronic and severe want. This was aggravated in 1943 and 1944 by the economic effects of inflation.

Chapitre IV.

SANITARY PROSPECTS

Differences between the Situation at the Close of the First World War and Now

Exaggerated fears regarding the expansion of epidemic diseases during and after the war that are commonly held by the general public and even the medical profession are based on the tradition of " pestilences " and epidemics during the wars of the past when etiology and prophylaxis of the infectious diseases were unknown, and on an imperfect knowledge and understanding of the epidemic situation which prevailed in the U.S.S.R. and neighbouring countries from 1919 to 1923.

In order to form a rational opinion concerning epidemic possibilities, one should therefore first consider that the diseases which caused the heaviest losses to armies in the past—typhus fever, smallpox, enteric fever and malaria—are now efficiently controlled with comparative ease.

One should, moreover, realise that the reason why infectious disease raged so disastrously in the U.S.S.R. from 1919 to 1923 was that the population of that country was essentially rural and scattered in character, and that for this reason it possessed a low degree of acquired immunity to the common infectious diseases.

Any large-scale migrations of this population, such as resulted from mobilisation, movements of White and Red troops during the civil war, and the famine due to the drought in southern Russia in 1922 and 1923, were therefore bound to cause widespread contagion and epidemics.

Destitution, moreover, favoured louse infestation, and consequently louse-borne typhus fever and relapsing fever.

Disorganisation of both military and civil health services, due to civil war and the dearth of physicians and trained sanitary personnel, and of drugs, aggravated the process.

It is quite understandable that, under these circumstances, the number of cases of typhus fever, relapsing fever, malaria, enteric fever should have been reckoned by millions, those of cholera, smallpox by hundreds of thousands.

It is also quite obvious that the return of millions of refugees from this hotbed of infection to Poland and the Baltic States, and of hundreds of thousands of prisoners-of-war, to Germany, Austria, Hungary, Czecho-Slovakia, should have involved a danger of the greatest magnitude for these countries and even for the rest of Europe.

It will be recalled that international action against this danger had to be improvised, first under the ægis of the League of Red Cross Societies, and finally under that of the League of Nations, [1] when it became apparent that the situation required not only the efforts of welfare organisations but also the effective co-ordination of the resources of all the Governments concerned.[2]

Now the epidemic situation differs considerably from what it was in 1918, as does the distribution in Europe of prisoners-of-war and refugees who will require repatriation during the armistice period. Most of them are at present in Germany, where none of the pestilential diseases prevails (at any rate in endemic form), where malaria and other sub-tropical endemic infections are practically non-existant, where the population is itself practically free from lice, and where the essential rules of sanitation are generally enforced.

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[1] The creation of the Epidemic Commission of the League of Nations recommended by an International Health Conference in London on April 14th, 1920, was ratified by the Council of the League of Nations on May 19th, 1920.

[2] *Report of the Epidemic Commission of the League of Nations*, doc. A.16.1921, page 4.

Crowded community life has caused many cases of the common epidemic diseases among them, but they have acquired immunity thereby so that they are unlikely to spread these diseases on their return home. They need to be considered as potential carriers only with regard to endemic, non-explosive infections such as venereal disease and tuberculosis.

The danger of dissemination of infection under these circumstances is infinitely smaller than it was in 1919-1921.

The United Nations Relief and Rehabilitation Administration, benefiting from the pioneer work of the Inter-Allied Relief Committees in London, allied military staffs and national health administrations have had time to prepare plans for the orderly repatriation of refugees and prisoners-of-war, medical relief and, if necessary, for anti-epidemic action. National Red Cross societies have prepared for emergencies.[1] One may therefore hope that international and national action will not be delayed by the necessity for preliminary discussions and organisation.[2]

We may be permitted to add that the continuance of the collaboration between the health and statistical authorities of most countries, irrespective of their political position, and the Epidemiological Intelligence Service of the League of Nations has made it possible for the latter to follow and study the movements of epidemic diseases and keep the interested health administrations and relief organisations informed of the situation and enable them to plan accordingly.

These are very weighty reasons, not for optimism, but for confidence in the future, and justify the hope that epidemics will be kept under control and that there will be a rapid improvement of health conditions in Europe after the close of hostilities.

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[1] Detailed suggestions for the preparation of post-war relief were given them in the Memorandum issued in 1944 by the League of Red Cross Societies, Geneva, under the title: "National Red Cross Societies and Post-war Relief".

[2] Progress in international co-operation in health matters is not limited to relief. The Health Organisation of the League of Nations, with its many technical committees, is a good example of collaboration between national health administrations, the League of Red Cross Societies, between national Red Cross bodies, etc.

Chapter V.

SUMMARY AND CONCLUSIONS

Increased contacts due to increased community life and migrations have resulted in a fairly general rise in diphtheria, scarlet fever and meningitis prevalence in Central, Northern and Western Europe. The same causes have had the same result with regard to typhus fever in Eastern and South-Eastern Europe.

Destitution—that is, lack of food, shelter, body-linen, soap, etc.—has contributed to the increase of typhus fever in endemic areas and among prisoners from these areas. These factors did *not* influence appreciably the incidence or severity of other epidemic diseases.

Food insufficiency in its more severe forms has caused actual starvation in the whole of Greece, and elsewhere among inmates of prisons and internment camps, and the poorer inhabitants of some cities. In its milder but chronic form, food insufficiency has caused a definite increase in the tuberculosis mortality and in the general mortality of several countries. It has been the main cause of many deaths, but also an effective contributory cause to a much greater number, particularly among elderly people.

As far as mortality is concerned, infants have, generally speaking, been spared, but this does not mean that they have not suffered from many forms of non-fatal malnutrition, as have adults, stunted growth in the young being the equivalent of loss of weight in older individuals.

Food shortage and malnutrition have prevailed far more severely in cities than in rural areas. This fact is of capital importance for the planning of food relief.

If this relief is prompt and adequate, and followed by a rapid economic improvement, one may expect, on the basis of experience gained during the aftermath of the first world war, a comparatively rapid physical rehabilitation of population groups now suffering from malnutrition, with a rapid fall of tuberculosis mortality.

One may reasonably expect that the return to their homes of the millions of prisoners-of-war, of the conscripted foreign workers now held in Germany, and of the demobilised soldiers of the victorious

and vanquished armies, will not result in large-scale epidemics, as most of the areas from which they will come can be regarded as clean from the epidemic standpoint.

Typhus fever prevalence in Eastern and South-Eastern Europe is the main exception to this general statement, and troops coming from these areas, as well as from North Africa, will require careful delousing to prevent the spread of that and other louse-borne diseases.

GIROUD'S INTRADERMIC TEST IN TYPHUS FEVER INFECTION[1]

Personal Observations, Techniques, and Possible Applications

by

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and

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The almost universal resistance of animal species receptive to typhus, and of man, to a new infection by the typhus virus has been, and is still, in accordance with the classical canons of immunity, ascribed to the presence of antibodies in the serum of subjects having survived a first infection.

From the beginning of his researches, NICOLLE has drawn attention in his many publications to the existence of certain substances which are capable of neutralising the virus in the serum of individuals suffering or convalescent from an attack of typhus fever. He did not, however, define the type of these neutralising antibodies, or find it possible to name them in accordance with the nomenclature in use in immunology.

There is no doubt that the discovery of the WEIL-FELIX (1) reaction, notwithstanding its lack of specificity, was the first demonstration of antibodies in infection through exanthematic typhus.

Later, the specific agglutination reaction given by the serum of typhus patients for *Rickettsia prowazeki* furnished proof of the existence of specific agglutinins. The test was made for the first time in 1918 by OTTO & DIETRICH (2) for classical typhus, and by ZINSSER & RUIZ CASTAÑEDA (3) for Mexican tabardillo. RUIZ CASTAÑEDA & ZIA (4) and ZINSSER & RUIZ CASTAÑEDA (5) have

[1] Received on March 26th, 1943, for publication, this paper appeared, in Spanish, with complete experimental protocols, in Vol. XVI of the *Revista de Sanidad e Higiene Publica*, Madrid.

used the agglutination of rickettsiae to measure the degree of immunisation against typhus obtained in the horse. FITZPATRICK (6) was the first to demonstrate the development of specific agglutinins against rickettsiae, in vaccinated monkeys and rabbits. A series of studies, begun in America by LIU, ZIA & WANG (7), showed the presence of these agglutinins in vaccinated persons. Indeed, this method is no longer in the experimental stage and the agglutination of rickettsiae by typhus sera is now a standard laboratory routine rightly called, in Europe, " Weigl's reaction " (8) as a tribute to its inventor, who first rendered it practicable, thanks to his technique of anal inoculation of lice for the cultivation of rickettsiae.

The study of precipitins, with the use of extracts of rickettsiae of Mexican tabardillo, was also carried out by RUIZ CASTAÑEDA (9), but his work on this particular point was not followed up.

As early as 1922, EPSTEIN (10) published a paper describing phagocytosis of *R. prowazeki*, obtained from lice infected by normal leucocytes in suspensions of immune human serum. Similarly, ZINSSER & RUIZ CASTAÑEDA (11, 12), showed that rickettsiae of the Mexican virus are often to be found in the polymorphs of guinea-pigs infected experimentally, particularly after the animals have been exposed to X-rays.

Indeed, the fundamental work on this question—as on so many others relating to immunity in typhus fever—is that of RUIZ CASTAÑEDA (13), who demonstrated the existence of *opsonins* for *R. mooseri* in the serum of persons suffering or convalescent from tabardillo, and for *R. prowazeki* in the serum of sufferers from Brill's disease. His experiments were made with the serum of the immunised horse and of infected guinea-pig.

RUIZ CASTAÑEDA (14) also showed the presence in these sera of substances capable of fixing the complement. He demonstrated, of course, what had already been pointed out by JACOBSTHAL (15), (16) in 1916 and 1917, and by EPSTEIN (10) in 1922. Jacobsthal used alcoholic extracts of infected lice as antigen, and Castañeda emulsions of tunica vaginalis and of peritoneal exsudate of rats. These emulsions contained about 1,000 rickettsiae per field. BENGTSON (17) is at present studying this question in full detail.

However, notwithstanding its practical importance, the study of the neutralising potency of typhus sera on the virus has been neglected and it is rather remarkable that Castañeda, in his publications on immunity in exanthematic typhus, does not concern himself

with this question. When measuring the degree of protection acquired against an infection or in the course of a hyperimmunisation, it is naturally more satisfactory to follow a procedure which measures directly the protection conferred rather than that, indirect, of assaying antibodies.

The method of measuring the protective potency of a serum by injecting suspensions of the serum and virus into an animal of a receptive species, necessitates the use of many animals. For these tests the customary method is to use guinea-pigs alone, although this means that many of these animals are necessary, in order to obviate the drawbacks of the irregularity of the reaction. These difficulties were recognised by GIROUD (18), even when working with anti-exanthematic serum as active as Zinsser's.

By analogy with the method for measuring the potency of toxins, particularly diphtheria toxins, Giroud hit on the possibility of using a skin " test ". In 1938, he published his first studies demonstrating the presence of typhus antibodies by a skin test (19).

At that time the demonstration of the antibodies had been facilitated by the knowledge then available that typhus virus was capable of producing macroscopic nodular lesions in the skin of the guinea-pig. According to Giroud, the antecedents in this question are to be found in the following studies : COMBIESCO, in 1931 (20), succeeded in obtaining local skin infections with the virus of " boutonneuse " or Mediterranean fever. DURAND, in the course of experiments not yet published, has succeeded since 1932 in obtaining skin reactions in monkeys and men through the inoculation of small doses of murine typhus virus and of boutonneuse fever virus. CAMINOPETROS & CONTOS (21) studied the local lesions produced by boutonneuse fever rickettsiae in the dermis of man and of animals, whether infected or not. BALTAZARD (22) studied the local reaction in the dermis of the guinea-pig in the course of murine and boutonneuse infections obtained by injection of virulent organs and of infected ticks.

In our opinion, however, the most fundamental antecedent in this question is the paper published by RUIZ CASTAÑEDA (23) in 1936, in which the lesions produced in the skin of the guinea-pig by the injection of rickettsiae of Mexican typhus are described in full histological detail. The work of Castañeda in this connection is really fundamental and it is surprising that, although it is included in the cycle of his studies on the mechanism of typhus immunity, its author,

who had made a thorough study of the intradermic typhus lesion, did not employ it for the detection and measurement of the protective substances in typhus sera. The merit of applying it thus falls entirely to Giroud.

Giroud followed up his first publication with several others. Directly or indirectly, they deal with the intradermic method, which he claimed could be used not only for the assay of protective antibodies, but also for the determination of the degree of virulence of different strains (24), (25), (26), (27), (28), (29).

Several of the studies we carried out at the National Institute of Hygiene at Madrid had as their object the determination of the presence of protective or neutralising substances in certain sera. We used the Giroud test, with which we were familiar through the publications of its inventor and also through personal indications he gave us. Although the number of techniques previously at our disposal had been large, we had not found them satisfactory for the strict application of the reaction. This prompted us to study the details of application and thus to obtain precise data from personal experiment, which we trust can usefully be published.

Our work includes the study of the skin lesions produced by the virus of typhus fever, and of the protection against these lesions conferred by certain human sera.

The Experimental Skin Lesion

Ruiz Castañeda's work on this question (23) was exclusively concerned with the study of the processes by which Fraenkel's nodules are formed, following experimentally the development of these lesions, and observing microscopically the reactions and the local immunity against the typhus virus. He used the guinea-pig as receptive animal and rickettsiae of Mexican typhus obtained from the tunica of guinea-pigs and rats and from the peritoneal exsudate of rats that had been exposed to X-rays.

Macroscopically, congestion of the skin was observed, followed by an induration which lasted about a week. Some cases showed a necrotic centre.

He succeeded in reproducing the perivascular lesions, in which he was able to demonstrate the presence of rickettsiae and Mooser's cells—*i.e.*, macrophagocytes, containing inclusions and rickettsiae. At first the leucocyte reaction was made up of polymorphs, which

were replaced from about the 3rd or 4th day by macrophagocytes. Some cases presented zones of necrosis in the infiltrated area.

Giroud's work had the definite object of obtaining a skin reaction which could be used for a protection test. He used the rabbit because its skin is thinner and more flexible. It is an animal, moreover, which is little receptive to the virus of typhus. Later, theoretical considerations confirmed him in his choice. The first studies on sero-protection which he published were made with a murine virus. Later, when his method of intranasal inoculation and production of pneumonia provided him with large quantities of rickettsiae, he used classical virus as well (24), (28).

The lesions produced in the rabbit have the same macroscopical characteristics as those described by Ruiz Castañeda in the guinea-pig, but are much more evident, sometimes attaining a diameter of 4 cm.

Microscopically, Giroud does not describe any lesions, but a dense infiltration of the dermis with, in places, necrotic foci, accompanied by the classical congestion and degeneration of the capillaries.

Technique and Results.

We have ourselves carried out a certain number of experiments on guinea-pigs. As the aim of the sero-protection test is, however, to determine the existence and the degree of neutralisation of the virus, we were chiefly concerned with the macroscopical aspect of the lesions. These are not easily recognised and studied in the guinea-pig, for the following reasons :

Intradermic inoculation is difficult, because the epidermis is thick ; erythematous or nodular reactions are slight ; the area of skin on the back is small, precluding inoculation of many dilutions in the same animal ; more rapid growth of the fur tends to hide the macroscopic lesion.

Abandoning the experiments on this animal for the reasons given, we next used the adult white-skinned rabbit. The back of this animal is much more suitable for the test, once the fur has been removed by electric clippers (these have the advantage of completely removing it without irritating the skin). Depilatories, shaving, or plucking are not advisable.

The intradermic injections were always made with virulent material diluted in ordinary culture broth. The quantity inoculated was constant : 0.25 cc.

Using varying dilutions of virus, we were able to obtain macroscopic reactions varying in size and in intensity. During the first 24 hours following the first inoculation, the skin became red. From the 2nd to the 3rd day, nodular reactions were observed. They were of varying sizes, and in some intense reactions there were necrotic foci.

We made a detailed histological study of the lesions produced in the dermis of the rabbit by the typhus virus, both classical and murine, both of which were obtained by cultivation on the vitelline membrane of the chick embryo. A summary of these results is given later.

From the first few days, a fairly intense infiltration of the dermis by polymorphs was observed. The conjunctive fibres showed hyaline degeneration. This, together with karyolysis and karyorrhexis of the polymorphs, reveals the necrotic nature of the lesion.

In the second stage, there were signs of a reparative reaction ; the presence of mononuclears and the multiplication of fibroblasts. Eosinophil polymorphs predominated in the leucocyte reaction we observed. The perifocal reaction was fairly intense. The endothelium of the vessels was thickened and many of them were blocked. We have to thank Professor Tello for the advice he gave us in the course of these histological studies. Figures 1, 2, 3 and 4 (see pages 709 and 710) show the lesions reported.

In a further study, we intend to continue our investigations of these lesions, both macro- and microscopically, and particularly their specificity. In the present paper, we are concerned only with the reading of the sero-protection test, and therefore exclusively with the visible macroscopic lesions.

Most of our experiments using Giroud's method were carried out with rickettsiae of a classical type (Madrid strains), cultivated on the vitelline membrane of the chick embryo, according to the technique of Cox. We have, however, made other observations using as material for inoculation organs of guinea-pigs infected with classical and murine viruses.

Infective Material from Guinea-pig Organs.

(a) *Murine virus.*

At the time these experiments were carried out, we had no murine virus other than the Casablanca III strain, kindly supplied by Dr. Georges BLANC.

Our experiments revealed the complete inactivity of the tunica exsudate and of the defibrinated blood. Injected into the skin of the rabbit, they provoked no visible lesion.

At the site of the inoculation of triturated tunica there appeared on the 2nd day a diffuse redness at all dilutions, which lasted up to the 6th day, but without the formation of any nodules.

The inoculation of brain substance, in the higher concentrations, always gave rise to a nodule, which lasted to the 6th day, though with a diameter always much reduced.

(b) *Classical virus.*

To observe the reactions obtained with classical virus, we naturally did not employ the tunica, since this virus does not induce the orchitis characteristic of the murine type of virus, but guinea-pig brain, infected with classical virus, and defibrinated blood taken from the same animals. The results obtained through the use of brain substance containing classical virus did not appreciably differ from those obtained from the use of murine virus. The diameter and nature of the lesions are practically identical with those obtained in the preceding experimental series. No dermal reaction whatever was obtained with the injection of defibrinated blood.

Infective Material from Culture of Chick Embryos.

The material used was obtained from embryos infected according to Cox's method : embryos incubated 5 to 7 days, inoculated with an emulsion of infected guinea-pig's brain. Details of this technique may be found in one of our previous publications (30).

(a) *Murine virus.*

For intradermic inoculation, our experience is limited to the use of the vitelline membrane of chick embryos infected with the murine strain Casablanca III. With this material reactions were comparatively intense, necrosis appearing with the more concentrated dilutions.

(b) *Historical virus.*

Activity of the different parts of the embryo.—A priori, the greater density of rickettsiae on the vitelline membrane led one to expect that the maximum reactions would be obtained by the injection of

that part of the embryo. Nevertheless, we took steps to ascertain its maximum infectiousness, and particularly its virulence in the skin of the rabbit.

The vitelline membrane, the allantois and the embryo were washed several times in broth, in order to eliminate every trace of yolk. The emulsion of vitelline membrane was made as usual by trituration and agitation in a flask with glass beads. The emulsions of allantois and embryo were prepared by trituration in a mortar with sterile sand. The dilutions of these materials, as well as those of the yolk and of the albumen were made in ordinary culture broth.

The tests are summarised in Table 1.

The results clearly showed the strong reaction of the vitelline membrane, the slight reaction of the yolk and the complete absence of reaction of the triturated allantois and embryo, and of emulsions of albumen.

Activity of the different strains of the virus.—We have studied the intracutaneous activity of the various strains isolated in the Madrid typhus epidemic of 1940-1941 (Tenorio, Torrallo, Autopsia and Melitón Puerto).

Detailed examination of the experimental protocols shows that some of the strains used for the passages from egg to egg are very active, whilst others provoke a very slight reaction. Three of the 4 strains used provoked the complete reaction—*i.e.*, with the three zones of erythema, nodule formation and focal necrosis—the acme of the reaction occurring between the 3rd and 5th days. All the lesions reached a diameter of from 15 to 20 mm., even with dilutions of 1 : 10,000. The necrotic foci did not exceed 5 mm. in diameter.

The retrogression of the lesions began from the 5th day, with a fading of the erythema, which had completely disappeared on the 7th day. When present, the induration and the necrosis, while continuing to regress, lasted up to the 10th or 15th day, at which date the skin regained a completely normal appearance, except for the persistence for many days of a local alopecia. Figures 5, 6 and 7 (see pages 711 and 712) show these reactions.

On the other hand, some strains, the Melitón Puerto for instance, gave only very slight reactions with the lower dilutions, and none at all with the medium and higher.

Equal receptivity of rabbits.—There seemed to be individual variations in the intensity of reactions where vitelline membranes

Table 1.

REACTION OF RABBITS TO THE INOCULATION OF VARIOUS PARTS OF CHICK EMBRYOS AND THEIR ANNEXES INFECTED WITH TWO STRAINS OF HISTORICAL VIRUS

Animal, strain and parts inoculated	Days of observation	Reaction in millimetres following inoculation of various parts of chick embr os, diluted at 1 : 100			1 : 1000			1 : 10.000		
		e.	nod.	nec.	e.	nod.	nec.	e.	nod.	nec.
Rabbit 76 c										
Tenorio strain	1	15	—	—	15	—	—	—	—	—
19th passage :	2	.	.	.	.	.	.	.	.	.
Vitelline	3	20	20	—	20	20	—	20	20	—
membrane	4	25	25	—	20	20	—	20	20	—
	5	—	25	1	—	20	1	—	10	—
	6	—	20	1	—	15	1	—	5	—
Allantoid	1–6	—	—	—	—	—	—	—	—	—
Embryo	1–6	—	—	—	—	—	—	—	—	—
	1	—	—	—	—	—	—	—	—	—
	2	.	.	.	.	.	.	.	.	.
	3	10	10	—	—	—	—	—	—	—
Yolk	4	15	15	—	10	10	—	—	—	—
	5	—	10	—	—	10	—	—	—	—
	6	—	5	—	—	5	—	—	—	—
Egg white	1–6	—	—	—	—	—	—	—	—	—
		1 : 10			1 : 100			1 : 1000		
Rabbit 296 c		e.	nod.	nec.	e.	nod.	nec.	e.	nod.	nec.
Torrallo strain										
15th passage :	2	30	30	—	25	25	—	10	10	—
	3	30	30	5	30	30	1	10	10	—
Vitelline	4	30	30	5	25	25	2	10	10	—
membrane	5	.	.	.	.	.	.	.	.	.
	6	—	25	4	—	20	2	—	10	—
	7	—	25	4	—	20	2	—	10	—
Allantoid	1–7	—	—	—	—	—	—	—	—	—
Embryo	1–7	—	—	—	—	—	—	—	—	—
Yolk	1–7	—	—	—	—	—	—	—	—	—

e = erythema. nod. = nodule. nec. = necrosis.

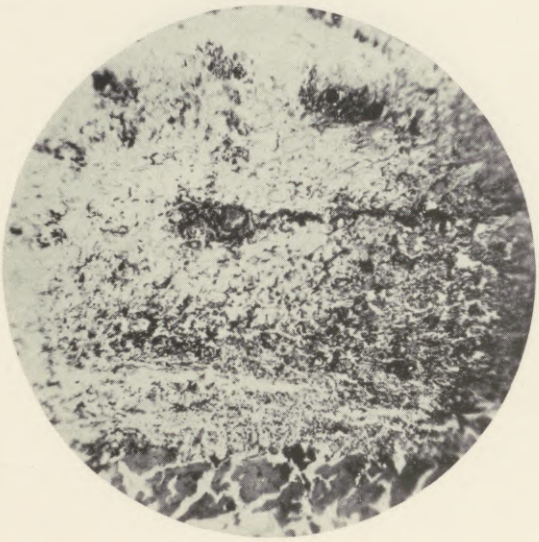

Figure 1. — Inflammatory reaction of the dermis. Nodules of perivascular infiltration *(from Fotos Stanek, Instituto Nacional de Sanidad).*

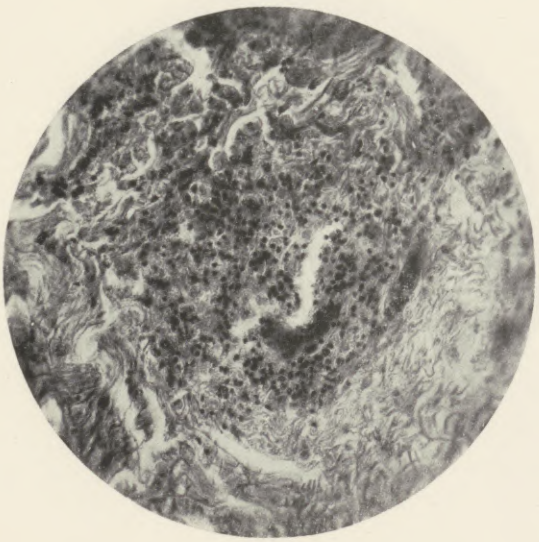

Figure 2. — Perivascular infiltration nodule.

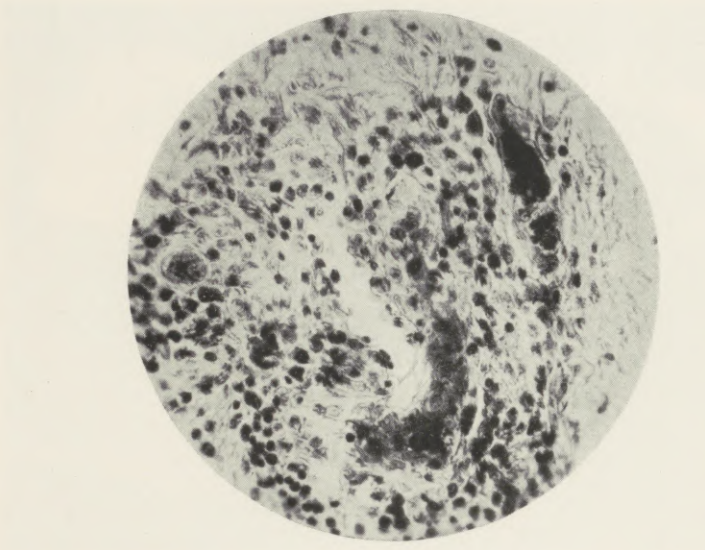

Figure 3. — Centre of the same nodule (enlargement of Figure 2).

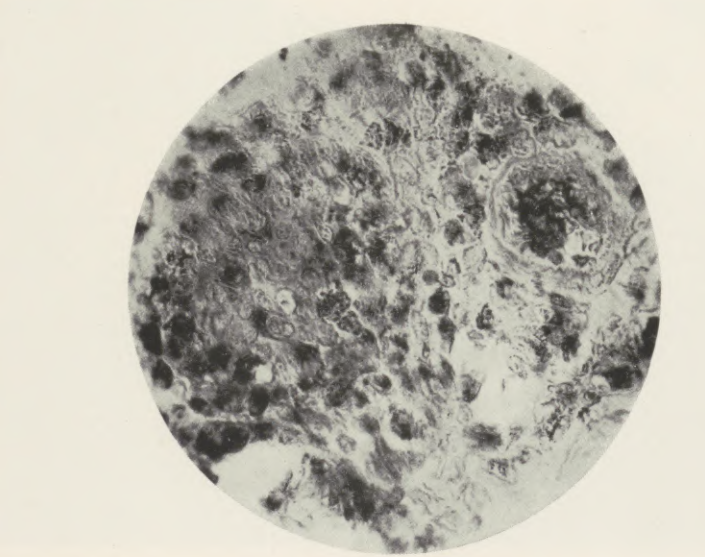

Figure 4. — Thrombosis and perivascular proliferation.

Figure 5. — Skin reactions following the intradermic injection of historical virus.

Figure 6. — Skin reactions following the intradermic injection of historical virus.

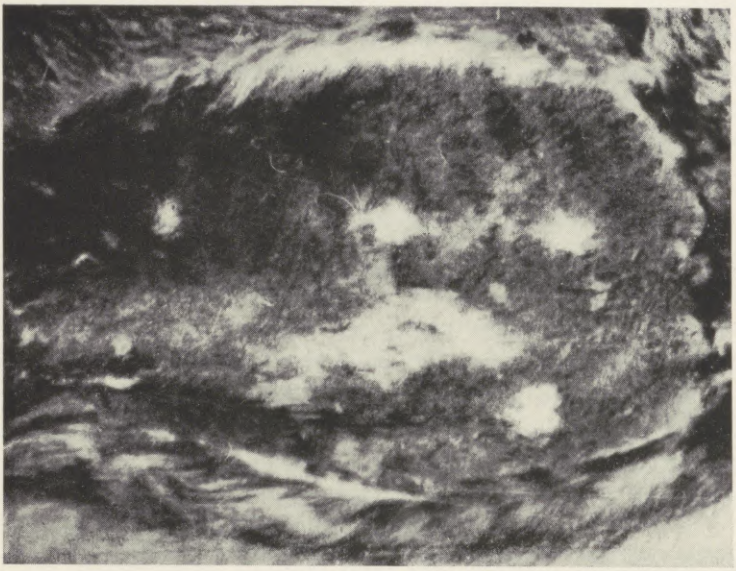

Figure 7. — Residual alopecia.

Figure 8. — Positive sero-protective reaction.
Only the control inoculations are visible.

of a single passage but from different eggs were used. Thinking that these variations might be due to differences in the receptivity of individual rabbits, we inoculated four of them with the same emulsion of vitelline membrane. The identical reaction in all four animals used proved that the differences recorded do not spring from individual differences in susceptibility of the animals experimented upon.

Variations in activity of the vitelline membranes of live and dead embryos.—Before concluding that the variations in the reactions were not due to differences in the receptivity of individual rabbits, we tried to find out whether they might not be due to differences in the infectiousness of different eggs of a same passage. We believed that we would find the true explanation once we were able to show that the embryos of the eggs inoculated on the same day and with the same strain do not all die at the same time. As material for inoculation, we therefore used embryos of which some had just died and others had been dead for some hours.

Our experiments showed that the vitelline membrane of live embryos was much more active than that of dead embryos, and should exclusively be used for sero-protection tests.

Finally, we must point out that our experiments showed that intradermic injections of normal vitelline membrane in the skin of the rabbit give practically no reaction.

Serum Protection Tests

Experiments with Techniques. Preliminary Experiments.

Once the effect produced by virulent material in the skin of the rabbit was known, we were able to proceed to the study of sero-protection—*i.e.*, the control of the suppression or attenuation of the skin reactions by adding serum to the virulent material.

Two methods were available for the preparation of suspensions of serum and virulent material: either fixed quantities of virulent material and variable dilutions of serum, or, inversely, fixed quantities of serum and variable dilutions of virus.

In our first experiments we chose the first method—*i.e.*, variable dilutions of serum added to emulsion of virulent vitelline membrane obtained from a strain possessing a high intracutaneous activity. In the sera-protection experiments, we injected 0.25 cc. of each control emulsion of vitelline membrane. On the other hand, when we

wanted to test a particular serum, we used 0.5 cc. of a mixture in equal parts of the serum studied and of the corresponding dilution of vitelline membrane. There thus remained for the rapid absorption of the serum the same quantity of virus as had been used in those cases where the serum had been lacking in protective power.

We tested, at dilutions of 1:25, 1:50 and 1:100, the protective power of the serum of healthy individuals, of a patient with pulmonary tuberculosis and of another suffering from malaria. The results were negative ; these sera were completely lacking in protective power.

The serum of a typhus convalescent gave complete protection, even at a dilution of 1:100. On the other hand, the serum of a convalescent from a mild attack of typhus gave only slight protection, even at a dilution of 1:25.

In another series of experiments, a different technique was employed. We used pure serum instead of dilutions, so that, in case the serum did have a slight protective power, this should not escape observation because of negative reactions resulting from a dilution of the serum. In the same way, we attempted to ascertain whether normal sera (*i.e.*, sera of healthy persons without history of or contact with typhus fever, and of those suffering or convalescent from other diseases) have a protective power when used pure or at very low dilutions.

We found that these sera gave only slight protection when pure and provoked no visible reaction when diluted.

Some examples of these reactions are given in Table 2.

Thus, our experiments clearly show that, even when undiluted, ordinary sera possess no protective power.

Observing that one serum, undiluted, did give a slight protection, we established its protective power *vis-à-vis* a fixed dose of virus, when pure and in progressive dilutions.

Conversely, we wished to ascertain what was the protective power of an undiluted serum against different dilutions of virus.

Finally, we made parallel experiments, using undiluted serum, for a test with three doses of virus which we considered sufficient to study the protective power of the serum with reference to the corresponding controls.

These experiments conclusively showed that the dilution of a serum produced a reduction in, and even a complete loss of, its protective power. They furnish experimental proof of the fact, which

Table 2.

SERO-PROTECTION TESTS CARRIED OUT WITH NORMAL HUMAN SERA

Serum used *Protection*	Day of observation	Serum	Reaction in millimetres following inoculation of virulent vitelline membranes diluted at 1 : 10			1 : 100			1 : 1000		
			e.	nod.	nec.	e.	nod.	nec.	e.	nod.	nec.
Normal serum	2	0	20	20	—	20	20	—	20	20	—
	»	+	20	20	—	20	20	—	20	20	—
No protection	4	0	—	20	—	—	15	—	—	15	—
	»	+	—	20	—	—	15	—	—	15	—
	7	0	—	15	—	—	10	—	—	10	—
	»	+	—	15	—	—	10	—	—	10	—
Serum of malaria patient	2	0	10	10	—	10	10	—	10	10	—
	»	+	10	10	—	10	10	—	10	10	—
	4	0	—	10	1	—	5	—	—	2	—
No protection	»	+	—	10	1	—	5	—	—	2	—
	7	0	—	10	—	—	5	—	—	2	—
	»	+	—	10	—	—	5	—	—	2	—
Serum of patient with pulmonary tuberculosis	2	0	20	20	—	20	20	—	20	20	—
	»	+	20	20	—	20	20	—	20	20	—
	4	0	—	20	—	—	20	—	—	20	—
	»	+	—	20	—	—	20	—	—	20	—
No protection	7	0	—	15	—	—	15	—	—	10	—
	»	+	—	15	—	—	15	—	—	10	—

e. = erythema. nod. = nodule. nec. = necrosis.

might logically be assumed, that a serum which neutralises a certain dilution of virus will also neutralise lower concentrations or quantities of the same virus. The use of three dilutions of virus at 1:10, 1:100 and 1:1000 alone as controls, on the one hand and, on the other, added to pure serum as reagent for the test, is, in our opinion, the most suitable technique.

Technique selected.

Finally, we decided to adopt as the technique for the intradermic test of sero-protection, the following formula : (1) use of strains having an evident reactive potency ; (2) use of controls with the three dilutions of 1:10, 1:100 and 1:1000 ; (3) mixture in equal parts of these dilutions of virus with pure serum ; (4) injection of 0.25 cc. of the dilutions of virus for the controls, and of 0.50 cc. of the suspension of serum to be studied and virus (0.25 cc. of serum, plus 0.25 cc.

Table 3.

SAMPLE SERO-PROTECTION TESTS CARRIED OUT WITH THE SERUM OF TYPHUS FEVER CONVALESCENTS

Initials and age of convalescent Number of months after the attack	Day of observation	Convalescent's serum	Reaction in millimetres following the inoculation of virulent vitelline membrane diluted at 1 : 10			1 : 100			1 : 1000		
Protection			e.	nod.	nec.	e.	nod.	nec.	e.	nod.	nec.
V.F. 43 years	2	0	30	30	—	25	25	—	20	20	—
2 months	»	+	—	—	—	—	—	—	—	—	—
	4	0	30	30	3	25	25	—	20	20	—
Complete protection	»	+	—	—	—	—	—	—	—	—	—
	7	0	—	25	3	—	20	—	—	15	—
	»	+	—	—	—	—	—	—	—	—	—
L.M. 40 years	2	0	10	10	—	10	10	—	5	5	—
3 months	»	+	—	—	—	—	—	—	—	—	—
	4	0	10	10	—	10	10	—	5	5	—
Complete protection	»	+	—	—	—	—	—	—	—	—	—
	7	0	—	10	—	—	10	—	—	5	—
	»	+	—	—	—	—	—	—	—	—	—
E.R. 45 years	2	0	20	20	—	10	10	—	10	10	—
1 month	»	+	—	3	—	—	3	—	—	2	—
	4	0	20	20	—	10	10	—	10	10	—
Almost complete protection	»	+	—	2	—	—	2	—	—	1	—
	7	0	—	15	—	—	10	—	—	10	—
	»	+	—	—	—	—	—	—	—	—	—
A.H. 32 years	2	0	30	30	—	25	25	—	20	20	—
3 months	»	+	3	3	—	—	—	—	—	—	—
	4	0	30	30	—	25	25	—	20	20	—
Almost complete protection	»	+	3	3	—	—	—	—	—	—	—
	7	0	—	25	—	—	20	—	—	15	—
	»	+	—	2	—	—	—	—	—	—	—
R.G. 60 years	2	0	20	20	—	15	15	—	15	15	—
1 month	»	+	5	5	—	5	5	—	—	—	—
	4	0	20	20	—	20	15	—	15	15	—
Fair protection	»	+	5	5	—	5	5	—	—	—	—
	7	0	—	15	—	—	10	—	—	10	—
	»	+	—	5	—	—	5	—	—	—	—
M.R. 31 years	2	0	15	15	2	5	5	—	5	5	—
3 months	»	+	5	5	—	—	—	—	—	—	—
	4	0	20	20	3	5	5	—	5	5	—
Fair protection	»	+	5	5	—	—	—	—	—	—	—
	7	0	—	15	3	—	5	—	—	5	—
	»	+	—	2	—	—	—	—	—	—	—
J.R. 30 years	2	0	10	10	—	10	10	—	5	5	—
2 months	»	+	10	10	—	5	5	—	—	—	—
	4	0	—	15	—	—	10	—	—	5	—
Very slight protection	»	+	—	10	—	—	5	—	—	—	—
	7	0	—	15	—	—	10	—	—	5	—
	»	+	—	10	—	—	5	—	—	—	—
R.L. 41 years	2	0	10	10	—	5	5	—	5	5	—
10 months	»	+	10	10	—	5	5	—	5	5	—
	4	0	10	10	—	5	5	—	5	5	—
No protection	»	+	10	10	—	5	5	—	5	5	—
	7	0	—	10	—	—	5	—	—	5	—
	»	+	—	10	—	—	5	—	—	5	—

e. = erythema. nod. = nodule. nec. = necrosis.

of virus) for the actual tests; (5) period of contact more than 20 minutes, but less than 90.

Once selected as the result of our previous tests, this technique was used for testing 46 sera—42 from convalescents from typhus fever and 4 from healthy persons or persons suffering from diseases other than typhus.

Again these tests gave negative results with the sera of healthy persons and of those who were suffering from diseases other than typhus.

A certain number of examples of these reactions is given in Table 3.

The results obtained with sera from typhus convalescents, grouped according to the time elapsed from beginning of convalescence and the taking of the sera, are summarised in Table 4.

Table 4.

SUMMARY OF THE SERO-PROTECTION TESTS CARRIED OUT WITH THE SERUM OF TYPHUS FEVER CONVALESCENTS

Time elapsed between the beginning of convalescence and the taking of serum	Number of sera tested	Sero-protection					
		Complete	Almost complete	Fair	Very slight	Total positive	No protection
1 month	(11)	1	4	6	—	11/11	0
2 months	(20)	3	5	10	2	20/20	0
3 months	(9)	3	2	4	—	9/9	0
1–3 months . . .	*(40)*	*7*	*11*	*20*	*2*	*40/40*	*0*
10 months	(1)	—	—	—	—	0/1	1
11 months	(1)	—	—	—	1	1/1	0

It will be seen that the positive character of the test showed up with great regularity, since in only one case did the serum of a convalescent give no protection. In this case the test had been carried out at the end of 10 months following recovery from a severe attack of typhus. On the other hand, the serum of another convalescent, of about the same age and having also recovered from a severe attack of typhus fever, was still quite protective 11 months from the beginning of convalescence. It cannot therefore be said that, beyond this time limit, there is a complete loss, or even diminution in, the protective power of a serum.

All these tests were made with fresh sera. To verify the preservation *in vitro* of their protective power, one serum was tested fresh, and later kept in the ice-chest at a temperature of 2° C. The experiment, although not repeated, is, in our opinion, quite conclusive. Taken from a convalescent from a severe attack of typhus two months after recovery, the serum was kept in an ice-chest at 2° C for a further period of 2 months. Another sample of blood was then donated and tested alongside the one kept in the ice-chest. Complete protection was given by the serum taken 4 months from the beginning of convalescence, but no protection was given by that donated 2 months earlier and *apparently* preserved in an ice-chest for a further 2 months.

Figure 8 (see page 712) gives a sample of completely positive sero-protection test.

Comments

Magnificent skin reactions are obtained by using for virulent injection material—as we have done—vitelline membranes of chick embryos infected with classical virus according to the technique of Cox. On these membranes great abundance of rickettsiae may be observed. These germs may be injected without rigorous exclusion of the vitelline membranes, since, like the yolk of the normal egg, they are quite inactive.

We believe that we are the first to have studied the test by using this material, which gives such excellent results.

Nearly all strains of classical typhus virus show similar local intracutaneous activity. Nevertheless, it should be kept in mind that this is the case for nearly all, but not for all. The lack of virulence of one of our strains (Melitón Puerto) in the skin of the rabbit shows the necessity for making a preliminary study of the material that is to be used for the tests.

Our technique differs from that of Giroud, essentially, in the use of three injections with three corresponding controls, using three variable doses of typhus virus. We believe that the advantages of this method are obvious. It furnishes a clear demonstration of the variety of results obtained with the different dilutions and particularly of the intensity of the protection. As normal serum does not possess the slightest protective power, we consider it is unnecessary to use it as a diluent for the control injections.

Naturally, the protective action of the sera requires the maintenance *in vitro* of the serum-virus mixture for some time. In our experiments, this time was never less than 20 minutes. The fact that a longer period of contact does not confer any greater protective power shows that the fixation of the active substances (the pathogenic substances of the virus and protective substances of the serum) is complete within 20 minutes.

However, sero-protection experiments do not constitute an infallible method for measuring the pathogenicity of typhus viruses. All our attempts to establish some relationship between the dose which will provoke an intradermic reaction in the rabbit—which we may call Lr (limit reaction)—and the one which will provoke a febrile reaction in the guinea-pig—Li (limit infection)—have so far yielded no result of value.

The sero-protection test is therefore highly specific and constant, as well as lasting in convalescents. As we have already shown, we obtained positive reactions in individuals who had had an attack of typhus as long as 11 months previously. A chronological study of the quantitative development of the protective power of the serum in typhus convalescents still remains to be made.

We believe that the test might be very useful for the study of inapparent typhus infection, a subject of which little is as yet known. The study of the protective power of the serum in vaccinated persons might perhaps help in finding a means of measuring immunity procured by vaccination.

Finally, in summarising here the possibilities of the test, we would draw attention to its suitability for measuring the activity of immune serum intended for prophylactic or therapeutic purposes.

Conclusions

1. The brain, tunica vaginalis, and blood of the guinea-pig, infected with classical or murine viruses, produce intradermic reactions that cannot be utilised for the study of sero-protection.

2. Comparative study of infection with classical virus of various parts of the chick embryo has shown that it is the vitelline membrane that provokes the strongest reactions.

3. Vitelline membranes used as reagents must be procured from embryos presenting an abundant development of rickettsiae—*i.e.*, live embryos.

4. In general, the different strains act all in the same way—*i.e.*, provoke reactions which are similar. There are, nevertheless, some strains which show little local activity when inoculated in the skin.

5. Pathological examination of the intradermic lesions provoked by the infected vitelline membrane always showed—apart from the histological lesions described by Ruiz Castañeda and by Giroud—a leucocytic reaction predominantly cosinophil in character, of an intensity out of all proportion to the slight local eosinophilia produced by injection of normal vitelline membranes.

6. The most satisfactory technique for carrying out sero-protection tests is that of using pure serum to varied dilutions of rickettsiae.

7. Sera of typhus convalescents always give protection, of greater or less intensity.

8. Sero-protection persisted in the individuals studied for a period which exceeds 11 months.

9. The sero-protection test is specific. Sera of healthy individuals and of individuals suffering or convalescent from diseases other than typhus do not reveal—at any rate in those cases we have studied—the slightest indication of protective power.

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FAMINE DISEASE AND ITS TREATMENT IN INTERNMENT CAMPS

EDITOR'S NOTE.

The following study[1] *is an abstract from reports summarising the observations made by a medical organisation, set up jointly by several national and international relief institutions, in a series of internment camps harbouring some 20,000 adolescent and adult internees.*

All facts that might incriminate the authorities of Governments responsible for the internment of these persons and for the conditions obtaining in the camps have, as far as possible, been eliminated, and the report is confined to the medical aspect of the observations recorded. These observations are in fact of considerable scientific interest, by reason of their number and continuity, which give them, in some ways, an experimental value; furthermore—and this is the main reason for their publication in these pages—they give therapeutic information which is capable of wide practical application in communities suffering from famine.

The reader will readily understand our reasons for suppressing the names of the doctors who have taken part in the medical relief accorded to the internees and in the preparation of this report, and also for suppressing all geographical particulars concerning the camps. These doctors prefer to remain temporarily anonymous, rather than allow the internees in their charge to suffer as a result of the interruption of the activities of the organisation to which they belong and of the relief which might well ensue from the premature publication of these particulars.

We intend, when hostilities are over, to lift the veil of anonymity and to give the names of the authors of the report and of the relief institutions which have made their activities possible and effective.

[1] Received for publication in August 1943.

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APPEARANCE OF AN " EPIDEMIC " OF FAMINE DISEASE IN THE CAMPS

After a prolonged period, during which the internees showed surprising physical and moral resistance to the very unfavourable health and nutritional conditions to which they were subjected, the situation changed completely from August 1942 onwards—that is, from 12 to 16 months after the original internment of some 20,000 of them [1]. *Famine disease* invaded the camps.

A first outbreak had already occurred in the spring of 1941, but it had disappeared during the fine season.

The first spell of cold weather led to a recrudescence of the disease, which spread rapidly throughout all the camps, creeping into every group, into all the huts, as though possessed of contagious characteristics. Though at first discreet, the symptoms became more and more evident, the trend more and more disquieting. In many ways the disease showed the classical signs observed during famines, but it also presented characteristics peculiar to the special environment of the camps throughout which it was spreading.

The death rate continually increased. During a first period, from September 1940 to January 1941, an average of two deaths a day were recorded in a camp containing 12,500 internees; in another containing 5,000 internees there were five deaths every other day. But this mortality increased by stages, revealing, after a period of resistance, the deep exhaustion and extreme physiological destitution of the organism. The men, who were the first to be affected, appeared

[1] Between 1940 and 1943, the internment camps received an influx of inmates the number and composition of which, by age, sex, race and nationality, varied considerably according to military and political conditions obtaining at the time, succeeding administrative enactments despatching to the camps or abroad different categories of persons.

This explains why famine disease broke out among internees at different dates, according to the date of the beginning of their internment, the time-lag of the disease varying with the season and the severity of the restrictions imposed.

The condensation and anonymity of this report make it impossible to give details concerning the variations in the interned population as a whole or in that of the camps where relief action was possible.

to be much less resistant than the women, the adolescents, and the children. Of a group of 40 patients transferred from an internment centre to a camp hospital, there was not a single survivor at the end of four weeks...

In view of this grave situation, urgent relief measures became imperative. The doctors delegated by private relief organisations to visit the camps drew up a plan of action which, once the initial administrative difficulties were overcome, it was possible to carry out, thanks to the help of the large relief organisations and to the good-will of the administrative authorities of the camps.

PLAN OF ACTION FOR THE MEDICAL RELIEF OF THE INTERNEES

The plan comprised the following essential points :

1. The discovery of sufferers from famine disease in the camps by doctors entrusted with the examination of all internees from the point of view of their state of nutrition. These doctors were also to organise and co-ordinate the programme of the relief organisations in co-operation with the official services in charge of the camps.
2. The hospitalisation of famine patients in huts specially fitted to facilitate their observation and treatment.
3. The organisation of special dietetic kitchens under the joint responsibility of the relief organisations.
4. Emergency medical treatment consisting in a large-scale administration of dietetic products, vitamins, minerals and tonics.
5. The segregation of persons " threatened " by the disease in a centre for prophylactic treatment separate from the camp.

Relief action was begun in February 1942.

Almost at once the medical investigators began work in most of the camps. Kitchens for cachectic persons were organised in the larger camps.

In the camp of G..., for instance, 9,000 persons underwent a thorough medical examination and subsequently were kept under regular observation.

The task of the doctors comprised the following duties :

1. The discovery, among the different groups in the camps, of persons revealing the deficiency-syndrome.

2. The clinical and therapeutic study of the sick hospitalised in the quarters for cachectic patients.

3. The study of the food situation.

4. The co-ordination of the work of the relief organisations in the camps, and the drawing up of medical instructions to be followed in the carrying out of this work.

5. The critical examination of the results obtained.

The doctors carrying out the investigations examined methodically 85% to 95% of the total number of internees.

The method of procedure was as follows :

Method of Discovering and Classifying Cases of Malnutrition

In the course of a preliminary examination, the internees were weighed and measured, their medical history noted, pulse-rate and blood-pressure recorded. Wherever possible, the weight of the internees before internment was ascertained.

In the course of a subsequent examination, patients were medically examined, and the results of this examination recorded on individual cards.

The investigation was necessarily limited to rapid and simple examinations, which alone are practicable when dealing with large numbers of persons.

The following data were used for a first classification of the persons examined :

A. Weight in relation to height.
B. Condition of the skin and subcutaneous tissue.
C. Muscular tone.
D. Station.
E. Cardiovascular system.
F. Œdema.
G. Blood counts.

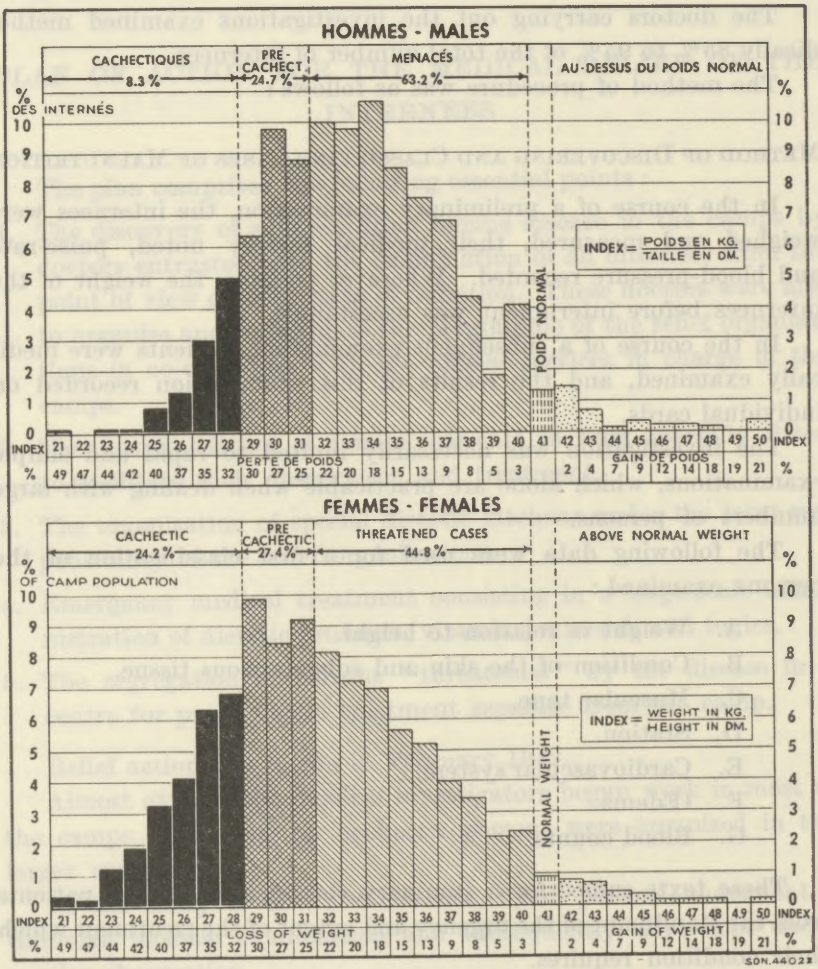
These texts suffice for a summary classification of the patients from the standpoint of the urgency and extent of the treatment which their condition requires.

A. *Weight in relation to Height.*

A $\frac{\text{Weight}}{\text{Height}}$ index was established for each person examined. This index, which summarises by means of a single figure the bodily condition—well-nourished or emaciated—of the individual, has no meaning by itself and must be read in conjunction with the normal, physiological index, such as is given, for instance, by the Dubois-Raymond

Distribution of Internees according to the Relationship between Their Body-weight and Their Height.

(Weights are expressed in kilogrammes; heights in decimetres.)



tables. The observed values were therefore expressed as a percentage of the normal $\frac{\text{Weight}}{\text{Height}}$ indices.

Care was needed, when making the calculation, to take into account, first, the real weight, by subtracting as far as possible the fallacious weight due to œdema, and second, the real height by allowing for the frequent spinal curvatures resulting from the bone lesions due to starvation (famine osteopathies). Wherever possible, weight *before* internment was recorded and, in the case of men, the height inscribed on identity cards and military papers was taken as the true measure.

The representation by graph of the relation between the observed and the normal $\frac{\text{Weight}}{\text{Height}}$ index of all inmates of a camp shows at a glance the collective state of emaciation (*cf.* graph, page 726).

Although an important decrease in weight is of real prognostic value in an individual case, experience has shown that such loss of weight does not, of itself, justify the conclusion that the condition of the patient is incurable. A variation in weight recorded in cachectic persons in a camp has more significance if a variation occurs at the same time among non-cachectic persons in the same camp.

Hernia is extremely frequent in greatly emaciated patients, and is rarely absent in men—even young men—in a pre-cachectic condition.

B. *Condition of the Skin and Subcutaneous Tissue.*

Every degree of atrophy of the cellular tissue can be observed among the patients, from those who have simply lost weight to those who are actually cachectic : in the latter a condition of veritable " anatomical dissection " can occur. The degree of atrophy is best observed when examining the patient from behind. At the extreme stage of atrophy, particularly in a formerly obese person, the skin is draped on the body like an article of clothing that has become too big.

C. *Muscular Tone.*

Early muscular hypotonia is observed in many patients. Up to a point, this explains the difficulty encountered by many patients in remaining standing and keeping their heads still. Patients do

not hold themselves properly, spinal curvatures are frequent. At a certain stage, the patients drop things held in the hands. They are incapable of the slightest effort. Dynamometer measurements taken of a fair number of persons gave figures corresponding to those obtainable from boys 10 or 12 years of age only.

D. *Station.*

At an advanced stage patients cannot stand. Some were observed trying to crawl back to their pallets on hands and knees. It was often desirable to measure the length of time a patient could stand. After some seconds or minutes, they felt pins-and-needles in the limbs and began to shiver, with or without profuse sweating.

Fainting is common, and is sometimes the danger signal pointing to a deficiency condition ; when it occurs two or three times a day the prognosis should be reserved.

E. *Cardiovascular System.*

Systematic recording of pulse-rates (volume, rhythm) is important, because it is easy to carry out and supplies useful indications.

Sometimes bradycardia, sometimes an acccleration of the pulse-rate, are observed. At a certain stage the rhythm may be irregular. This was true also of blood-pressure, which was recorded in all patients examined.

F. *Œdema.*

The more fixed and voluminous œdema becomes, and if it extends to the visceral organs, the more significant it is. The characteristic facies of the malnourished person, due partly to swelling and partly to anæmia, betrays a condition of advanced physiological destitution.

G. *Blood Counts.*

Blood examinations of under-nourished persons, picked out at random, revealed in every case an anæmic condition.

As in a widespread epidemic, it was essential at once to segregate obvious cases of deficiency diseases and, on the other hand, to trace incipient cases with a view to preventive action and the limitation of the havoc played by the disease.

It appeared, however, useful also to use this experimental field in order to obtain dietetic and therapeutic information that might ultimately be applicable to free communities.

The fact that patients showing the syndrome of deficient nutrition were segregated in one place, subjected to the same rules and easily supervised, permitted a thorough scientific analysis of the symptoms recorded and of the individual developments of this disease. Over and above its theoretical interest, this research furnished the practical possibility of studying a nutritional régime suitable outside the camps for the population as a whole, among whom the deficiency syndrome had already begun to show itself.

In this respect it may be recalled that publications appearing at the end of the first world war concerning malnutrition in countries then suffering from food shortage were not only of scientific interest, but of practical value, inasmuch as relief organisations and medical missions benefit at present from the information supplied by these works. That is why a professor of biological chemistry, an acknowledged authority in this branch, was associated with the direction of scientific research. Unforeseen events have abruptly interrupted this research, and with it all medical activity.

Classification

Faced with the task of emergency food relief, ordinary classifications [1] have no practical value. It is necessary to divide the patients according to their condition into three main categories, indicative of the degree of priority to be accorded in their treatment. For this reason the internees were grouped, after examination, under one of the following heads :

(a) Cachectic cases.

(b) Pre-cachectic cases.

(c) Threatened cases.

(a) *Cachectic Cases*, the diagnosis once made, were admitted to the special infirmaries called " quarters for cachectic cases ".

Even from a distance, one is struck by the gaunt appearance of these patients, which is due to extreme emaciation and the typical facies. The skeleton-like emaciation is remarkable. Cutaneous and muscular atrophy is general. The subcutaneous adipose layer has long since disappeared. The skin is dry and scaly. Many adults of medium height weigh only about 40 kg (88 lb.).

[1] E. J. Bigwood : " Guiding Principles for Studies on the Nutrition of Populations ". Document L.o.N. C.H.1401, Geneva, 1939.

The complexion of cachectic patients is ashen or of a subicteric pallor, according to whether a beginning of cyanosis or a hemolytic process predominates. But anæmia and paleness of the mucous membranes are common to almost all cases. The emaciated features give these patients a cadaverous appearance. A detailed clinical description of this class of patient will be found later.

(b) *Pre-Cachectic Cases* revealed the same symptoms, but to a lesser degree, muscular atrophy being less pronounced. Anæmia appears also to be less advanced. Loss of weight is not so great, nor is the general condition so serious, while the results of the different tests showed that the deterioration was less grave.

(c) *Threatened Cases* were in a better general condition than those in the two previous categories. The tendency to hunger-œdema begins to show itself here, as at this stage it is fugitive, migratory and recurrent. Emaciation is, however, somewhat frequent. Among these cases were also included convalescents from typhoid fever, from acute attacks of gastric and duodenal ulcer, from flare-ups of tuberculosis, cases of Parkinson's disease, as well as chronic uncompensated cardiac cases.

Results of the Examinations undertaken.

In G... camp, in a population of about 11,000 internees, of whom 9,000 were examined, *331 cachectic, 839 pre-cachectic, and about 4,000 threatened cases were found.*

These proportions, however, were not static. *Famine disease*, which developed like a virulent epidemic, constantly progressed in the camps. Every week systematic investigation discovered new pre-cachectic and threatened cases, as though the virulence of the pathogenic agent were far from spent. Examination of the causes of this development will be the subject of a special chapter.

CLINICAL AND THERAPEUTIC STUDY OF THE PATIENTS HOSPITALISED IN THE QUARTERS FOR CACHECTIC PERSONS

From the first results of the work of investigation, it appeared that more than half the inmates were threatened by the grim symptoms of famine disease.

I. Clinical Forms

We shall begin by describing the form most frequently encountered, to which we gave the name of " humid form ".

A. *The Humid Form.*

Œdema is the principal symptom in these patients.

Its frequency. — Taking the examinations carried out as a whole, an average of 50% of the internees in the camps showed famine-œdema. [1] But œdema is far from constituting the unique or pathognomic sign of a state of physiological destitution.

Contrary to the opinions usually expressed and to what was at first observed in the camps, a greater number of famine œdemas were met with in women than in men. Among the men, however, there were a greater number of cases of bradycardia and of hypotension—a proof that they were the more seriously affected. Thus it was in men that ascites was most frequently observed.

Œdema in young girls of from 17 to 22 years of age, with fairly stable menstruation, was also observed, contrary to what has often been affirmed.

Sites of the œdema. — Œdema usually affected the back of the foot, the malleoles, the inside of the thighs, the regions under the chin, the cheeks and the eyelids.

Its characteristics. — Soft, white œdema, at first transitory and migratory, gradually becoming permanent following several recurrences. This was observed particularly in cases of peripheral œdema.

[1] In camp G...., in June 1942, the proportion of famine œdemas was 48.2% among men (572 out of 1,187), including 1.7% with ascites only, and 46.5% with generalised œdema. The proportion among women was 52.4% (758 out of 1,447).

A certain number of patients were found to have ascites, without their having noticed it or being incommoded by it. Ascitic cases (about 10% of the œdema cases) are more resistant than other cases to the action of diuretics and improved nutrition.

Œdema was sometimes observed, unaccompanied by loss of weight or other symptoms of famine disease, in a certain number of men who appeared to be in a moderately good general condition, with well-developed musculature and a practically normal subcutaneous adipose layer. Subjects showing this syndrome were nearly all members of the staff of the canteens and kitchens. It might be wondered whether these œdemas resulted from dietary imbalance or were of alimentary toxic origin.

Associated symptoms (common to all the clinical forms). — Apart from œdema, these patients often suffered from pollakiuria, more particularly nocturnal. This symptom was apparently mainly due to general demineralisation, but to a certain extent also to the cold, as in many camps the nights were cool in huts that were not weatherproof.

Analysis of urines, moreover, did not reveal the presence of pathological elements.

Pleural effusion, another form of visceral œdema, is rare. In no way does it differ, as regards development or prognosis, from inflammatory œdemas.

It should be noted that all the œdematous subjects had a good appetite until a very advanced stage of the disease. The extreme fatigability experienced by all, at a certain point in its development, is felt as a constant, painful sensation. Sometimes this sensation occurs after only slight exertion.

Skin infections are frequent, sensitiveness to cold extreme. The temperature of the patients is often low (sometimes under 95° F (35° C)).

Laboratory tests. — Blood tests made at random among cachectic and pre-cachectic patients showed that they were all anæmic. None of the patients examined showed a count of 4,000,000 red corpuscles per cc. of blood. In the great majority a count in the neighbourhood of 3,000,000 was obtained. In barely 5% of the patients was the hemoglobin index higher than 70%. In all other cases it varied around 60%.

Thus, as regards blood, just as in the case of the weight, the *dangerous reduction of 30 % in physiological value* was commonly recorded.

Other clinical forms of famine disease, depending on the predominance of some particular symptom, the origin of the internees and the victualling conditions, were observed, and are described below.

B. *The Dry Form.*

Characterised by the absence of œdema, it was met with more commonly among Spaniards and Italians. It should be noted that these men were suffering from chronic under-nourishment extending over a period of several years. The skin of these persons is dry and often scaly. In their case the forms taken by atrophy are particularly striking.

C. *The Anæmic Form.*

Blood examinations showed a certain number of hyperchromic anæmias (43% of the cases studied). Taken in conjunction with the results of the neurological examinations, this fact is of considerable pathogenic and therapeutic interest. It would indeed be well to ascertain whether the absence of a factor to be found chiefly in meat does not result in anæmias of the pernicious type, with the neuro-anæmic syndrome recorded. It should be noticed that a subject suffering from the most persistent neurological disturbances, while at the same time having put on weight, had a $\frac{\text{Weight}}{\text{Height}}$ index of 1.23, a hemoglobin index of 57% and only 3,330,000 red corpuscles and 2,700 leucocytes per cc. of blood.

As a rule, the blood sedimentation rate of patients of this group had not increased.

Petechiæ were observed on the back of the hand and on the forearm in some cases.

D. *The Circulatory Form.*

At the beginning of the disease—particularly among less-seriously affected patients—blood pressure appeared to be unstable, varying in both directions, sometimes from morning to evening.

Later, hypotension became permanent and was often found together with bradycardia, at first fugitive, then also becoming permanent. Hypotension then constituted a danger signal pointing to the gravity of the deficiency condition. Hypertension, which was more

frequently observed than is generally supposed, persisted, notwithstanding the general asthenia, until a fatal issue supervened in serious cases.

The heart appeared to suffer, like the other muscles, from hydremia. It is curious that a doubling of the first sound with no other signs of gallop rhythm was frequently detected.

Cardiac rhythm disorders (partial and complete arrhythmia) were not observed to be more common among cachectic persons than in other milieux.

E. *The Neurological Form.*

Ataxia and Romberg's sign were the most striking symptoms. As a rule reflexes were quick. Paresthesia and polyneuritic pains were found. Vestibular tests were most often negative. General sensibility was normal.

In some of these patients, a remarkable mixture of Parkinsonian and ataxic syndromes were observed, sometimes a well-marked " steppage " gait. Muscular tests (Barré's test, for instance) were negative. As a rule in these cases the sedimentation rate was *greatly increased.*

F. *The Mental Form.*

Patients suffering from this form had a fixed, lifeless and apathetic gaze, they were completely confused, replied to questions only in monosyllables and after a certain lapse of time. Prostrated on their pallets, their debility was extreme.

Confusional and amnesic states were very impressive. Even where convalescence was advanced, one was struck by the retardation of ideation. Spasmodic laughing-and-crying was commonly observed. Bulbar disorders and signs of meningeal reaction were seen in some fatal cases.

Lumbar puncture carried out on comatose patients revealed no abnormality, apart from a slight hypertension of the cerebrospinal fluid. Lumbar puncture is contra-indicated in patients suffering from shock and vasomotor disturbances.

A more prolonged observation would certainly have permitted better classification of the various forms mentioned and the addition of some others. Nevertheless, the experience acquired during the

investigations it was possible to make has clearly demonstrated the existence of these clinical forms in all the camps, one form or another preponderating according to the geographical situation of the centre and the origin of the internees.

The determination of these clinical categories has been very useful, not only from a scientific point of view but also for prognosis and treatment of cases.

II. Development and Termination of the Disease

We were able to follow the development of the famine syndrome in newly-arrived internees, in a normal state of nourishment and abruptly subjected to the camp fare.

The deterioration of the general food supply and the growing destitution of the internees led to the appearance, at the end of 1942, of the first symptoms of malnutrition after a certain " time-lag ".

The revealing signs generally succeeded each other in the following order :

(1) During the first three months, heavy loss of weight, of from 17 to 33 lb (8 to 15 kg.) per month ; then a gradually decreasing loss from month to month. Gradual disappearance of the fatty tissue. Appearance of inguinal hernia.

(2) General fatigue ; irritability : complexion very typical, becoming straw-coloured, lemon-yellow, or white ; dry skin, fugitive matinal œdema, sometimes lasting for 10 minutes.

(3) Headaches ; mental disorders, depression or excitement ; rapid reflexes, static disturbances, amenorrhea.

(4) Giddiness, ankle-cramps, fixed œdema with a tendency towards generalisation. Cardiac arrhythmia.

If active intervention is not attempted, the outcome of famine disease is inevitably death.

Restrictions on freedom of movement, cold, unfavourable hygienic conditions and moral depression can only hasten this fatal development.

Death. — Careful and detailed observation of the conditions under which death occurs was very instructive from the pathogenic point of view. One was struck by the abrupt transformation from

an apparently normal state—during which the patient goes about his own affairs—to a state of coma, followed at a more or less short interval by death. Sometimes the patients are suddenly struck down and collapse while walking about, sometimes they die in their sleep.

At other times, the patients pass slowly away, showing signs of progressive asthenia. (Causes of death from pre-existing or intercurrent diseases are not alluded to here.) Several *post-mortems* revealed nothing of note macroscopically, apart from the occasional presence of cerebral œdema.

It is interesting to note that, even with experience, it is impossible from observation and from the seriousness of the condition to forecast death or even approximately to fix the date at which it will occur.

Mortality. — We give below the mortality for two camps during January and February 1942. With a few exceptions, death was due to famine disease.

	January	February
Camp I (harbouring about 400 persons)	45	49
Camp II (harbouring about 2,800 persons) . . .	59	38

III. Complications

A. *Tuberculosis.*

Two thousand fluoroscopic examinations were carried out in a camp well equipped for the purpose. Thirty cases of pulmonary tuberculosis were found, of which the sufferers were ignorant. Of these, 20 were cases of fibroid degeneration, non-progressive in appearance. On the other hand, progressive lesions were discovered in 10 cases, among which were 3 non-febrile forms of miliary tuberculosis.

It was regrettable to discover this form present in a nurse. These findings show how indispensable it is to carry out a careful and systematic medical examination upon arrival of each person destined to live in the camps, either as a member of the staff or as an internee.

As far as conditions permitted, X-ray screen examinations were extended to all the inmates of the other camps.

B. *Cardiovascular Disturbances.*

Persons in the fifties (20% of the inmates) frequently showed cardiac enlargement, and the question arose whether these were all cases of myocardial hypertrophy or whether, in some of them, a pericardial exsudate was not present. The aorta was, as a rule, transparent throughout the whole of its length. Incidentally, the marked accentuation of the bronchovascular outline should also be mentioned.

C. *Alteration of the Bones.*

Generally speaking, there was a striking decalcification of the bones, of the ribs as well as of the vertebral column ; sometimes the osseous trabeculæ were quite visible. Taken as a whole, these observations are typical of a deficieney eondition. They reveal an abnormally advanced degree of decalcification and, more broadly speaking, of demineralisation.

D. *Vascular Disturbances.*

Angiospasms of the toes and finger-tips were often observed. Some cases were so pronounced that a diagnosis of Raynaud's disease was made, but the condition cleared up with the administration of vitamins, and particularly of nicotinic acid. Numerous cases of frost-bitten extremities were observed during winter, some of them very severe, necessitating, for instance, the amputation of fingers in two children. Many eases of " senile " opacity of the crystalline lens and frequent cases of precocious senility were also observed.

E. *Endocrine Disturbances.*

Clinical and X-ray observations lead us to add some remarks on endocrine disorders. The field of investigation in this domain was considerably restricted for want of a suitable apparatus. Hyperthyroid cases were comparatively rare, but were nevertheless to be found. Goitres were frequent. The large proportion of diving goitres was surprising. Perhaps they may have some connection with the amenorrhea which was frequently recorded (30% of the women).

Puberty in girls appears to have been retarded. Below is a table concerning inmates of a large camp :

Number of women with amenorrhea: 171 (over 15 and under 45 years of age)

Amongst whom, cases of —

Bradycardia	21 = 12.3%	
Hypotension	22 = 12.8%	
Hypertension	3 = 1.7%	
Œdema	87 = 50.8%	(47 of a mild form)

The question of adrenal insufficiency will be treated in a later study. Advanced forms were not met with. But the co-existence of hypotension, hypoglycemia and asthenia should stimulate research with regard to adrenal alterations.

The discovery of alterations of spermatogenesis, not surprising in malnourished subjects, is of only theoretical interest in an internment camp.

Dysfunction of genital endocrine glands suggests pituitary disturbances.

On the other hand, there is no doubt that diabetics, who were fairly numerous, benefited from the deficient diet. No case of diabetic coma has been recorded.

The same remark applies, moreover, to rheumatic, metabolic diseases.

IV. Prognosis

The evolutionary curve of an epidemic shows, in a first phase, a progressive increase in morbidity, consequent upon contamination, till it reaches a high level or a peak, and then falls again when a sufficiently large proportion of the population has acquired immunity, or when active measures effectively prevent the spread of the disease. At the time when our observations were brought to an end, famine disease, which behaved very much like an epidemic disease, had not reached its peak in the camps. Every week, systematic investigation revealed new pre-cachectic and threatened cases. Thus the causes of the disease were far from having disappeared. The reasons for this atypical development will be discussed later. For the moment, we shall review the prognostic factors.

A. *Sex.* — Observations made among women were particularly interesting. They were affected after a time-lag of ten months, as compared with the men, and the increasing number of women who,

in July 1942, showed the deficiency syndrome clearly illustrates—alas !—the general deterioration in the situation. The fact that the calorie requirements of women are 20% lower than those of men, and the slowing-up of metabolism due to amenorrhea, explain in part why they have been more resistant to famine than men. It appears that in the end their resistance broke down in its turn, and that the prognosis in the case of women became less favourable than it had previously been.

B. *Seasonal influences.* — There is more chance of saving a patient during mild seasons than during winter. Cold, in fact, makes for a less favourable prognosis because it is a weakening influence, a cause of calorie losses, and aggravates existing vascular and nervous disorders. It has often been possible to observe œdema disappear simply as a result of warmth. It can be said with certainty that patients who died during the winter died as much from the cold as from famine. The effect of cold on under-nourished patients was frequently seen and, among other things, showed itself by an abnormally low body-temperature. The appearance of numerous and sometimes serious chilblains, even at the end of winter, is connected with the harmful action of cold on the circulation.

What we have said of the circulatory form of famine disease explains, moreover, that such patients are equally sensitive to the action of heat, quite apart from the danger of the dysentric syndrome which then also threatens. Thus it is that the two extreme seasons are most to be feared for the under-nourished person.

C. *Hypotension.* — Among the other elements of the prognosis, we single out hypotension which, once it becomes permanent, is a more serious condition than bradycardia.

D. *Clinical types.* — Those persons suffering from neurological and mental forms have the most unfavourable prognosis.

E. *Intestinal complications.* — The appearance of abundant diarrhœa is of very grave prognosis owing to the dehydration and demineralisation it provokes, as well as to the faulty assimilation of nutrients and vitamins which follows. The incidence of diarrhœa varies greatly from one camp to another.

F. *Purpura.* — The appearance of purpura generally constitutes a rather unfavourable prognostic sign.

G. *Asthenia.* — Inability of a patient to keep on his feet for some minutes calls for extreme reserve in forecasting the probable evolution of the case. It is indispensable to prescribe strict and complete rest for these patients if they are to be protected from sudden death, which occurs only too often when patients get up too soon or without proper supervision.

H. *Atrophy.* — Pronounced atrophy and extreme emaciation are inseparable companions of cachexia and adversely affect the prognosis.

I. *Previous disease.* — In this study, we have not taken into consideration patients attacked by famine disease who were already suffering from other diseases. Their condition becomes worse owing to the vicious circle thus created. Naturally, such patients need special treatment and supervision.

J. *Treatment.* — *The possibilities of cure depend* on the age of the patient, on the season, on a certain number of factors enumerated above, on the early institution of treatment, but *particularly on the extent of the therapeutic action undertaken.* Contrary to what was at first thought, *an incurable condition* (apart from terminal coma) *does not exist.* Notwithstanding a remarkable loss of weight, profound asthenia and a very serious general condition, it was found possible, through *persevering and energetic treatment*, to save individuals considered to be irrevocably lost. This finding is of considerable social importance and fundamentally alters our pathogenic and therapeutic ideas.

Thanks to unremitting treatment based on a substantial diet rich in fats and proteins, and on hypertonic injections of glucose, patients were saved who had been considered hopeless cases, suffering as they did from generalised œdema with ascites and pleural effusion, myocarditis, hemorrhagic purpura of the skin and mucous membranes, and losing as much as 55% of their physiological weight. Mental cases reacted more slowly.

K. *Segregation of serious cases.* — The segregation of severe cases is an indispensable therapeutic factor. Administrative action which prevented this segregation hindered many cures.

Segregation is particularly indicated in cases of mental disorder.

V. Vitamin Insufficiency

The exact rôle played by vitamin insufficiency in famine disease cannot yet be exactly determined.

Although it was easy to discover signs of an insufficient calorie intake (emaciation, loss of weight, etc.), clinical examination did not enable us to diagnose with certainty a deficiency condition resulting from the lack of any definite vitamin.

Moreover, the vitamin requirements of the human body can vary and be subject at the same time to individual factors and to endogenous influences, which govern the absorption and utilisation of the vitamins consumed.

Vitamin C Deficiency.

Mention has already been made of several cases of petechiæ which appeared on the forearm and on the back of the hand.

Dentists also reported blood suffusions in gums and hemorrhages more severe than usual after operations.

It was found that the vegetables served in the camp were particularly poor in vitamins C and D. Therapeutic results obtained by administration of vitamin C have been satisfactory in these cases.

Under the conditions of examination existing in the camps, it was not possible to measure the amount of vitamin C in the urine and to make saturation tests.

Vitamin D Deficiency and Decalcification.

The number of children suffering from rickets was large. Although, on the whole, the number of serious forms was not alarming, some very distressing forms were often seen.

There was a large proportion of cases of spinal curvature. Fortunately a certain number were still curable, provided special supervision and early methodical treatment were available.

Dental caries were numerous. But doctors were more impressed by the cases of gingivitis and dental periostitis.

A certain number of cases of falling finger- and toe-nails were cured by a calcium and anti-rickets vitamin treatment.

Vitamin A Deficiency.

A certain number of cases of rebellious eczema and trophic ulcers in young persons were cured by the administration of vitamin A.

Two cases of xerophthalmia and 2 of blindness in adolescents who presented the complete syndrome of famine disease were recorded.

Night-blindness was detected in a small number of cases.

A certain number of cases of precocious presbyopia were observed.

One was sometimes struck by the pallor of the entire pupil, quite apart from any disease of the optic nerve or refractive disorder (10% of the cases).

All the doctors who examined the patients were of the opinion that the lack of vitamin A was the most obvious.

B Complex Deficiency.

The satisfactory therapeutic results obtained in mental and neurological cases, as well as in neuro-anæmic cases, with vitamin B_1 and brewers' yeast, proved to a certain extent the lack of vitamin B_1.

One was also struck by the very marked tympanitis observed among almost all the internees. The lack of vitamin B_1, hindering digestion, may perhaps play an important part in the genesis of this symptom. Here again, the administration of aneurin was sometimes attended by satisfactory results.

Nicotinamide Deficiency.

It might be asked whether some of the numerous cases of diarrhœa were not due to a nicotinamid deficiency. This possibility should be borne in mind, particularly in cases where severe enteritis is accompanied by glossitis or aphthous stomatitis. Cases are more common than is generally supposed. The rapid disappearance of all symptoms two days from the beginning of treatment with nicotinamid is sound proof of lack of the vitamin being the origin of these disorders. This syndrome was met with in about ten cases, while during the course of prolonged observation, several thousand internees suffered from intestinal disorders.

We must, however, emphasise how rarely were clear-cut cases of deficiency of one or more vitamins discovered among the thousands of internees suffering from famine disease. We consider this fact to be of fundamental importance. Notwithstanding systematic search—inspired, we must admit, more by medical literature than by our own experience—it was rarely possible to find positive proof of avitaminosis either by clinical examination or by therapeutic tests.

Although we did not have a perfect apparatus at our disposal, much care was devoted to the search for night-blindness in children, adolescents and adults. The results of these investigations were, on the whole, negative.

It was the same with digestive disorders and neuritic symptoms, which might be attributable to the pellagra syndrome. It is possible that a monotonous diet is more likely to result in the development of a vitamin deficiency syndrome than even a serious degree of under-feeding which extends to every nutritive element.

STUDY OF THE VICTUALLING OF INTERNEES

It is obvious that the miserable physical condition of the internees was due to the inadequacy of their food, from the point of view of both quantity and quality.

To realise this, and before setting out on the study and interpretation of the nutritive value of the food supplied, we should like to recall here some elementary notions concerning calorie requirements and food values.

It will suffice to mention briefly the indispensable elements of a normal diet and to compare it with that served to the inmates of the camps.

General Considerations concerning the Normal Diet, from the Point of View of Quantity and Quality.

A. *Quantitative Requirements (in Calories).*

Let us recall the standard figures laid down in 1936 by the Technical Commission on Nutrition of the Health Committee of the League of Nations.[1]

An adult of medium weight satisfies his daily requirements, when at rest, with a diet yielding a minimum of 2,000 calories.

2,400 calories a day is considered enough for the same person leading an ordinary life and not engaged in manual work.

The supplement for light muscular activity is 75 calories an hour, making about 600 calories a day. The total would thus be 2,400 plus 600, equalling 3,000 calories a day. When the energy-value of the diet,—*i.e.*, the calorie intake—is less than the energy expended, the body has to draw upon its reserves to make good the deficit, which it does at the expense of its own tissues.

[1] " Physiological Bases of Nutrition ". *Bull. Health Org.*, 1936, **5**, 391-415.

Insufficient food first leads to *emaciation*, more or less severe according to the degree of the food insufficiency. To compensate the indispensable minimum physiological expenditure, the body first draws upon those of its substances which are the most calorigenic, which give the highest calorie yield with the maximum economy—*i.e.*, fats. Laying the adipose tissue under contribution is not fraught with immediate danger providing that a certain limit is not exceeded and that the balance of the diet is maintained.

The menace of famine disease draws closer as the fat reserves begin to be exhausted, and as the proteins—muscles and other organs—are drawn upon to compensate the inevitable combustion. Emaciation progresses faster from that moment because proteins yield only about 4 calories per gramme—that is, only half those yielded by a gramme of fat. Moreover, the drain on the protein reserves has most harmful consequences, because the requirements in calories are satisfied at the expense of more highly differentiated tissues: muscles and other organs.

Thus, at this stage, cases of disastrous emaciation are observed.

It is true that, as a result of a chronic condition of under-nourishment, a certain state of adaptation ensues wherein expenditure of energy is below the normal. After a certain time, chronic under nourishment results in a reduction of the corporal volume to a point at which the impoverished diet will again suffice to nourish the body, diminished through loss of body-proteins and the fat of the adipose layers. Nevertheless, it cannot be said that there is any physiological adaptation to the situation created by the diminution of the calorie intake, or any physiological equilibrium, so long as loss of somatic weight persists.

It is impossible to reduce the ration of an average adult to a point *lower than 1,800 calories* without causing progressive emaciation and physiological disorders, which vary in gravity according to the general condition of the individual, the duration and degree of under-feeding, and the amount of physical effort he has to exert.

B. *Qualitative Requirements.*

Apart from their calorie content, different nutrients possess *specific properties* which can in no way be replaced by those of other nutrients.

For the human body to lead a normal existence, a *well-balanced* diet, comprising carbohydrates, fats and proteins, and the necessary

amounts of minerals and vitamins, is required. Nothing can replace these basic elements, which are indispensable for the sound functioning of the living body.

The following are the usual proportions advocated in Western civilised countries for the maintenance of the energy-producing equilibrium of a diet :

50% carbohydrates (glucids),
30% fats (lipids),
15% proteins (protids), proteins of animal origin composing 60% of the total.

An unbalanced diet results in profound physico-chemical disturbances in the tissues and fluids of the body ; the organism becomes incapable of fixing calcium or even retaining that already present in the body ; mineral equilibrium is upset. This explains the famine osteopathies and famine œdemas that prevailed in an almost " epidemic " form among the internees.

It seems certain that, in the prevention and treatment of famine disease, it is essential to pay more attention than is usually done to the question of mineral equilibrium. Already in normal times, but more particularly since the war, European dietaries have been deficient in mineral salts—especially calcium, phosphorus and iron—as a result of the predominance of starchy foods with low protein content, and an insufficient consumption of vegetables and fruit.

With regard to proteins, it is interesting to note differences in biological values (Thomas) of the different protein substances—that is to say, the proportion of tissue proteins replaceable by nutritional proteins. All proteins are not equally capable of fulfilling body requirements with the same efficacy : in particular, proteins of vegetal origin, in no matter what quantity, cannot satisfy all the bodily needs. *Certain protein requirements* must be met by the proteins of meat, milk, or milk products (amino-acids of animal origin).

Study of the Food Situation in the Camps

Famine disease is the consequence of a chronic quantitative and qualitative deficiency in the diet. It is necessary to stress this nevertheless obvious truth, having regard to certain views—not, it is true, authoritative—which have been expressed on this subject.

A. *Amounts of the Daily Ration expressed in Calories.*

Below we give an example of the fare provided in a camp during one month, from October 1st to 30th, 1942 (per person):

	Grammes	Calories
Bread	7,500	17,000
Fats	240	2,160
Sugar	480	1,960
Pumpkin (served morning and evening for three consecutive weeks)	30,000	6,100
Jam	2,225	2,250
Sardines	1,800	1,674
Cheese	200	600
Meat	913	911
Tripe	770	465
Carrots	3,750	1,406
Cabbage (carrots and cabbage served morning and evening for a week)	3,750	1,125
		35,651

i.e., 1,188 calories per person per day (35,651 : 30) resulting from an average daily intake of:

	Grammes
Proteins of animal origin	15
Proteins of vegetal origin	37
Fats of animal origin	12
Fats of vegetal origin	2
Carbohydrates	210

In computing the calorie value, no account has been taken of losses through different causes which, according to the competent authorities, can amount to 15-20% of the theoretical energy value of the nutrients, nor of the coefficient of resorption, nor of the fact, for instance, that the sardines contained 30% of their weight of salt (NaCl).

Allowing for these factors, the value of the daily ration per person was not more than *950 calories a day.*

In an annex at the end of this report, we give other examples of actual diets, taken at random from other camps.

We shall indicate here only their calorie value.

In one camp, the quantitative value of the daily ration amounted to 1,070 calories per person on February 26th, and to 1,092 on March 4th, 1942. In the same camp, the theoretical daily ration of calories was 845 calories per person, on April 4th, 1943.

This calculation includes the supplement supplied to cachectic persons by the camp authorities.

For other internees, the calorie value of the daily ration was lower by another 100 calories.

In a third camp, the average daily calorie value of the ration was 958 in the month of June 1942 (calculated by weight).

In yet another camp, during the week of May 17th-23rd, 1942, deliveries of victuals for the camp showed, on paper, a calorie ration of an average value of 1,310 calories per person per day.

A calculation of the calorie value *by weighing the ration actually served out in the internee's mess-tin*, on the other hand, gave much lower figures, viz.: 1,070, 1,109, and 1,077 calories per person per day.

It will be seen from these facts that the *quantitative value*, expressed in calories, never exceeded 1,100 *per person per day* in any camp, and from October 1941 to June 1942 varied between 950 and 1,100 calories.

Many documents are available concerning the food situation in Germany and the invaded countries from 1914 to 1918. The situation of the towns was considered to be critical when the daily protein intake fell to 40-50 grammes, the daily intake of fats to 20-30 grammes, and the daily calorie value to 1,400-1,800. The average ration for an adult was 2,232 calories (LOEWY's 1916 enquiry). During the first three years of the war, German workers received 2,200 calories (LUSK enquiry, CODVELLE). Practically speaking, the loss of weight recorded was the consequence of the reduction in the amount of fats consumed.

The lowest figure—yet which is well above those recorded in our internment camps—was found in occupied northern France in 1917-1918. It was 1,467 calories (LAMBLING enquiry).

B. *Qualitative Value of the Daily Ration in the Camps.*

The daily protein ration in the camps was not more than 30-40 g. of almost exclusively vegetal origin; the actual intake of fats was

8-10 g. a day. The intake of proteins and the number of calories contained in the rations distributed in the camps in no case allowed the maintenance of protein balance.

When the protein ration and the total daily number of calories simultaneously fall below the physiological minimum, not only is nitrogen balance upset, but elimination of proteins is abnormally increased. Failing a sufficient quantity of protein, food should contain at least an important proportion of carbohydrates and even, in certain circumstances, of fats. Yet it would still not be possible to combat cachexia, even by administering large quantities of carbohydrates or of fats, and this addition to the diet would not increase the patient's weight if the indispensable physiological minimum of proteins required were not supplied.

The situation in fact was as follows.

Instead of a normal daily ration of 1 gramme of protein per kilo body-weight—*i.e.*, 60 grammes for a person weighing 60 kilos—we found in the diet provided 48 grammes of vegetal protein. Moreover, instead of 24 grammes of animal protein—the strict minimum that should be provided—15 grammes at the most were recorded. A continual nitrogen deficit resulted which, even if it amounted to only 9 grammes a day, represented for these internees 3,300 grammes of protein in the course of a year—*i.e.*, 16 kg. 500 g. (36 lbs) of their muscles and other organs.

Over and above these considerations with regard to qualitative deficieney of the ration, from a purely biological standpoint, other qualitative factors relating to the freshness, preservation and preparation of the articles of food further aggravated the food situation in the camps.

Mass victualling, which does not take individual requirements, nor those of any particular state of ill-health, into account, is attended by unavoidable disadvantages.

The extreme monotony of the fare should also be borne in mind.

As is well known, the best way to induce a deficiency condition is to maintain an unvaried nutritional régime over a long period of experiment.

As a result of large-scale purchases of a single seasonal article of food—*e.g.*, cabbage, pumpkin, turnip, Jerusalem artichoke or carrot—the diet in the camps remained unvaried for long periods of time. Other factors, which had no connection with seasonal, administrative or economic requirements, favoured this dangerous

uniformity. The same vegetable appeared on the table alone for many weeks, having been improperly stored and *for too long a time*, badly prepared, swimming in an excessive amount of liquid. Such a vegetable, poor in vitamins and mineral salts, engendered various disorders of associated vitamin deficiency. Thus, for a whole month, in one camp, day after day, pumpkin was served at midday and in the evening, with the result that everyone's face turned yellow.

Moreover, a considerable number of internees suffered for a long time from acute or sub-acute *intestinal disorders*. As is now known, defective intestinal absorption can be at the root of a vitamin deficiency even though the vitamin intake be adequate. This sufficiently explains why all internees sooner or later showed pre-deficiency conditions if the food situation was not improved.

Food Situation outside the Internment Camps

Through the kindness of the Nutrition Section of the Regional Hygiene Institute situated in a large industrial town, near which the camps were situated, we obtained a study of the nutritional conditions prevailing among its population (more than a million inhabitants). This study relates to October-December 1941.

In the preceding chapter, we have therefore chosen figures covering the same period in the case of the camps. It should be remarked, however, that subsequently the situation in the camps deteriorated to an infinitely greater extent than in neighbouring regions.

First, let us consider the *quantitative value*.

From *February to September 1941*, the general average of the different social groups studied amongst the town population was :

1,737 calories for adults of both sexes ;
1,565 calories for young persons 12-19 years of age.

The lowest figures for some *persons* having a particularly low ration were :

1,600 calories, male adults (as against 2,217—the highest figure) ;
1,400 calories, female adults (as against 1,741—the highest figure) ;
1,400 calories, young persons 12-19 years of age (as against 2,097—the highest figure).

The average for the least-favoured *group* was not lower than :

1,876 calories, male adults ;
1,517 calories, female adults ;
1,477 calories, for young persons 12-19 years of age.

For the quarter *October-December 1941*, the figures were as follows :

Average of all the groups studied, taken together :
1,700 calories, adults of both sexes ;
1,620 calories, young persons 12-19 years of age.

The lowest figures for some partieularly unprivileged persons are not given, but those for the least-favoured group did not fall below :

1,764 calories, male adults ;
1,509 calories, female adults ;
1,610 calories, young persons 12-19 years of age.

The perusal of these different doeuments gives the impression that the food situation in this town population was eomparable to that of the German towns in 1917 and was even in some respeets less favourable.

As to the food situation in the eamps, the figures were not unlike those of the ration of the average inhabitant of Madrid towards the end of the siege, during the eivil war. That ration was 852 ealories, after having been 1,514 ealories at the beginning of the siege. (These figures are taken from the remarkable studies made by F. GRANDE, during the civil war in Spain, from 1937 to 1939.)

If the rations of the internees are compared, from the *qualitative point of view*, with those of the town population quoted above, it will be seen that, for the period Oetober-Deeember 1941, the latter ineluded an average of 65 g. of protein a day for men, and 55 g. of protein a day for women, of whieh an average of 21 g. was protein of animal origin.

It should be noted that these rations followed those of the period February-September 1941, whieh had definitely provided the minimum protein intake. This, however, was not the ease as regards the internees, who had already been suffering from a condition of nitrogen defieieney for at least one to two years.

Vitamin and Mineral Salts

The daily ration in vitamins and mineral salts for the internees has not been calculated in detail. Nevertheless, certain considerations enable us to weigh up the situation.

In the first place, the report on the food situation among the town population reached the conclusion that there was an insufficient intake of A, B and C vitamins. In the second place, since the internees received food rations which were much poorer in vitamins, it follows that they must have suffered from a definite insufficiency of these biocatalysers, an insufficiency which was intensified by the high incidence of intestinal disorders among the inmates of the camps. This question is dealt with in the chapter on vitamin deficiencies. We shall mention here only that the discovery of a serious lack of calcium and phosphorus applies even more to the inmates of the camps than to the population studied in the large town.

Conclusions

From this comparative study, it followed that:

(1) The ration supplied to the internees in the camps was 40-50% lower than that of the civilian population of a nearby large industrial town, which was certainly very unfavourably situated from the point of view of food supply;

(2) The epidemic of famine disease was bound to persist in the camps failing an increase in the actual net ration;

(3) The efforts of the organisations to combat famine disease by supplying additional nourishment and by applying intensive drug therapy would, under the existing conditions, remain ineffective.

The raising of the ration in all the camps to the level of that of the civilian population, which was also that laid down for the centres under supervision, was thus urgently necessary.

If the concentrated efforts of the organisations were not to be in vain, all losses during the handling of the victuals, from the moment of their entry into the camp to the time they were served up in the internee's mess-tin, should have been prevented by efficient supervision.

If the efforts made by the organisations on behalf of the cachectic patients in the special quarters provided for them had been supported by a similar effort on behalf of the inmates of the entire camps, it might really have been possible to save them all. For this, it would have sufficed to make sure that the internees were receiving the strict ration to which they were entitled.

THE CO-ORDINATION OF THE WORK OF THE VARIOUS RELIEF ORGANISATIONS IN THE CAMPS AND THE ESTABLISHMENT OF A MEDICAL POLICY GOVERNING THAT WORK

The considerable effort made by the relief organisations was illustrated by the fact that the supplementary foodstuffs sent to the centres represented the contents of several dozen goods trains of 50 wagons each.

It must, however, be recognised that this remarkable effort did not succeed in warding off famine disease.

Without any doubt this partial failure was due to the abnormal insufficiency of the basic food rations accorded to the internees.

Nutritional Therapy

From the moment of discovering that famine disease was spreading in the civilian internment camps, the organisations, concentrating and intensifying their efforts, installed several kitchens specially designed for the feeding of cachectic patients. The administrative authority of the camps continued to supply the foodstuffs of the ordinary basic ration in kind.

Thus cachectic patients were able to receive a daily ration of from 2,200 to 2,500 calories, and the quality of this ration was appreciably improved.

The relief organisations took action in two directions, every organisation assuming the task corresponding to its capacities.

In the first place, they sought rapidly to procure a nourishing diet, judiciously composed and adapted to the capacities of patients in a state of advanced cachexia (see Annex I).

But to master the famine " epidemic " it was necessary, in the second place, simultaneously to undertake energetic prophylactic measures. With this object in view, the organisations extended

their field of action to include those persons who were then only "threatened", by supplying an ever-growing number of internees with food supplements, in the form of pea soup, soups made of rice, noodles or macaronis, and pearl-barley, or dried or fresh fruit, representing 250 to 400 calories according to the particular camp supplied (see Annex I).

It is impossible in the present study to give an adequate idea of the immense effort exerted by the organisations engaged in relief work, the flexibility of their action and the spirit of co-operation animating them. Some inkling of it may be given by the fact that in less than a week the kitchens were installed ; that a detailed card-index was prepared and kept up-to-date containing the follow-up observations concerning patients cared for by each organisation ; that most of the necessary foodstuffs were purchased in the Balkans, Turkey, Portugal and Spain, and that others were despatched from the two Americas ; that storehouses were built ; and a great deal of work done in connection with the handling and storing of the supplies. When we add that, in addition to these material arrangements, many necessary negotiations were undertaken with the authorities on behalf of the internees either in general or in individual cases, a fair idea is given of the impressive work carried out jointly on behalf of the internees by a dozen international and national relief organisations.

The work of the organisations covered from 65 to 70% of the internees, according to the camp. During June 1942, in one large camp alone, 1,958 rations a day (of which 98 were double rations) were distributed among 2,750 inmates.

In the maternity section, with double rations, the proportion of rations to inmates rose to 102.1%. The fact that an increase in the weight of newly-born infants was obtained, from an average of from 4 lb. 10 oz. to 5 lb. 8 oz. (2 kg. 100 g. to 2 kg. 500 g.) in 1941 to 6 lb. 6 oz. to 6 lb. 13 oz. (2 kg. 900 g. to 3 kg. 100 g.) in 1942/43, is undoubtedly due to the particularly intensive action in favour of mothers.

The question of feeding elderly people calls for some special remarks.

Old people in the camps ate as much as, and often even more than, adults or adolescents. They ate all day and part of the night...

This was because, more than their younger companions in want, they suffered from an obsession which sharpened their hunger. The

fear of going hungry, of having less to-morrow than to-day, made them eat ; they finished up their food parcels at once, taking every precaution not to be seen or asked for a share.

Perhaps also their hunger was due to an insufficiency of the gastric and intestinal secretions preventing a satisfactory assimilation of food insufficiently masticated and often inappropriate to senile digestive organs.

It would be interesting to administer extracts of gastric, digestive and intestinal organs, such as pepsin, trypsin, pancreatin, etc., to such persons.

Results of tentative experiments made in this direction appeared to be sufficiently conclusive—a lessening of the sensation of hunger, better assimilation, easier digestion—to justify a systematic examination of this problem.

Drug Therapy

Differentiation of the various clinical forms of famine disease allowed of variation in the medical treatment.

Thus for the *dry form*, general tonics, drugs containing amino-acids, combinations of stimulants and of vitamins and phosphorus-containing products were administered under permanent medical supervision.

For patients suffering from the *humid form*, the usual diuretics were added to the above, mercury treatment excepted, owing to the susceptibility of the kidneys in cachectic patients.

Brewers' yeast, glucose, aneurin, were tried in the case of the *neurological* and *mental forms*, and in cases of diarrhœa.

In cases of *anœmia*, hepatogastric iron extracts and vitamins were administered.

* * *

We shall now examine in succession the use made of the products principally employed, and any indications that appeared to result as regards their future employment.

Brewers' yeast. — The large quantity of this product available permitted us to study its action on a large scale. Its effect on paresthesias of the neurological form was less striking than its favourable influence on the general condition, as shown by the increase in

weight and feeling of well-being. Its effect on diarrhœa was particularly noteworthy. In some rare cases, diuresis was favourably influenced. The doses prescribed were two soupspoonfuls a day. Frequently, however, intolerance to this drug was shown, and this considerably restricted its employment.

Calcium by intramuscular injection. — With this product, increase in weight, particularly when the injections were combined with vitamin B_1, remineralisation in patients suffering from falling finger- and toe-nails, from dental decalcification and osteoporosis, were often observed.

Calcium per os. — Among patients suffering from œdema, calcium has shown itself to be a very efficient diuretic. We shall not dwell upon the classical indications concerning its use in tuberculosis, etc.

Glucose. — Chiefly indicated for circulatory and hepatic insufficiency. As high doses were available, it was possible to administer it *per os* at the rate of 60 g. per day, and very interesting results were observed as regards the action of this drug as a diuretic, heart and liver tonic. In psychasthenia, the stimulating effect, and in mental disturbances, the tonic action, were found to be truly remarkable. But it was in cases of advanced cachexia most of all that intravenous injections of glucose, at the rate of two to three times 30 cc. of a solution at 10%, incontestably led to a " turning-point " in the malady, thanks to which it was possible to save cases which had appeared to be hopeless.

Iron. — Iron gave excellent results, particularly with young anæmic subjects, provided it was used in very high doses and that, when necessary, hydrochloric acid and vitamin C were added to it.

Insulin. — This rare drug enabled us to treat a large number of serious diabetic and hepatic cases.

Coramin. — Patients suffering from circulatory asthenia or instability, or from cardiac insufficiency, who formed an important element in the camps, were greatly benefited by this drug. In many cases where coramin would have been the drug most indicated, it was necessary to abstain from prescribing it, owing to shortage of supplies.

Vitamin B_1. — Aneurin was administered in 5-mg. tablets for the purpose of giving the high doses necessary to obtain the desired

effect. Two to three tablets a day, administered to patients showing the neurological and mental syndrome, rapidly improved the prognosis in cases which had formerly been severe.

Its favourable effects on the troublesome tympanitis resulting from a diet in which the proportion of carbohydrates was excessive should be mentioned.

In obstinate cases of diarrhœa, the effects of aneurin is similar to that of brewers' yeast. Owing to the continued disturbance in intestinal absorption in these cases, it is nevertheless more advantageous to administer this vitamin in the form of injections.

Vitamin A (cod-liver oil). — The lack of this vitamin made itself sorely felt.

All camp doctors were continually pressing for it. The insufficient supply of nutrients containing vitamin A explains certain disorders resulting from vitamin A deficiency.

The cod-liver oil placed at our disposal was gratefully received. Unfortunately, its vitamin A content was very low. To have obtained good results, we should have been able to dispose of a product richer in vitamin A.

Vitamin D. — Vitamin D was given in combination with vitamin A, in the form of an oil solution. Apart from its usual effects, this combination showed itself to be an excellent general tonic, particularly for children.

Vitamin E. — A useful stimulant in cases of asthenia and nervous depression. Indicated for mental and neurological types of famine disease.

Nicotinamide. — Excellent effect on angiospastic disorders, also in circulatory disturbances associated with chilblains (in which cases we were able to combine it successfully with lactoflavin injections). This vitamin is also indicated in cases of diarrhœa.

It has already been explained why it is not astonishing if, in these complex cases of a deficiency-condition, recoveries of the kind produced by a particular vitamin in a clearly defined case of avitaminosis are not recorded.

Vitamins are biocatalysers which make certain fundamental reactions in the body possible ; but first of all it is essential that catalysible matter should be present in the body.

The administration of vitamins to subjects suffering from deficiency disease creates the conditions favourable to an optimum assimilation of nutrients, but such nutrients must be supplied.

Hepatogastric extract in draughts. — A valuable drug owing to its anti-anæmic principles and indispensable amino-acids. It has been prescribed in large quantities with success. It produces a decided and rapid increase in the number of red corpuscles. This result is more pronounced if the patients take iron at the same time. In greatly debilitated patients it results in a very rapid improvement in the general condition.

Medicinal combinations of wheat-germ, mineral salts, and vitamins. — Such products, greatly in demand, not only because they provided an agreeable corrective to the monotonous taste of the soup, but also because of their reparative action, clearly stimulated increase in weight and diminished nervous disorders.

Ortedrin, Pervitin. — Supervised treatment with small doses of these drugs gave very good results in a certain number of cases of simple stupor and cyclic insanity.

Sulphonamides. — In a community of the size of the camps, extensive use was naturally made of these anti-infectious drugs, which, owing to their merited reputation, tend to be employed as a universal panacea.

In this connection a general observation on this subject should be made, and three suggestive indications mentioned.

1. A certain number of serious "sulphonamide-resistant" infections were observed. Their careful examination led to the conclusion that, in general medical practice and contrary to what would appear to be the case in venereology, fractional doses often resulted in an apparent resistance.

All cases—with the exception of a generalised streptococcal septicæmia—reacted favourably to strong doses, a third part being administered in the course of the first two hours of the morning, another third in the evening, and the remaining third during the course of the day.

2. During an outbreak of whooping-cough in the camps, very interesting results were obtained by sulphonamide therapy. In all cases the number and intensity of the paroxysms of coughing markedly decreased in the first 24-48 hours. Of the 22 children

attacked, 14 ceased to have paroxysmal coughing as from the third day. Applied at the onset of the disease, sulphonamide therapy manifestly cut it short in 3 other cases. In the 5 remaining cases, the frequency and intensity of the paroxysms were unquestionably and noticeably reduced, although the general duration of the disease did not appear to have been shortened.

In nurslings the drug was given by enema, in infants by intramuscular injections and by mouth.

3. A series of cachectic patients were observed who suddenly showed ataxia, rapidly complicated by dysphagia, aphasic and mental disorders, and meningeal symptoms, death occurring in from 5 to 10 days. Examination of these patients brought to light an abnormally heavy infestation by lice. Lumbar puncture revealed some leucocytes and a slight increase in albumen in the C.S.F.

An autopsy which was carried out revealed macroscopically cerebral œdema and congestion of the kidneys and the liver.

Intensive sulphonamide treatment saved 4 of these cases, which were following a similar course to those which had died.

These cases were therefore considered to be toxic infections in debilitated persons, in conjunction with a parasitic infestation of such intensity as to be remarkable even in this uniformly infested milieu.

General Measures of Hygiene

Rest. — Until they are out of the danger zone, complete rest is indispensable for cachectic patients. For pre-cachectic, and more particularly for threatened cases, a modified régime of rest with a regulated amount of activity must be prescribed. It is indispensable that these patients should be under constant medical supervision, in which muscular tests with the dynamometer play a far from negligible part.

Segregation. — To ensure this rest, and also for psychological reasons, it is absolutely essential that these patients should be segregated. The constant care and systematic supervision they require cannot be assured under any other conditions.

CRITICAL EXAMINATION OF THE RESULTS OBTAINED

On the whole, it can be claimed that the dietary and drug treatment applied from the beginning of February 1942 completely changed the appearance and condition of the patients.

At the end of three months treatment in the section reserved for cases of the neurological and mental forms, veritable resurrections were observed.

> A professor of law, 68 years of age, hospitalised at the beginning of March suffering from ataxia, apraxia and in an advanced state of stupor, on June 16th gave us a remarkable exhibition of jugglery with a bottle and three balls, as well as with a basin ; his repartees were delightfully subtle and to the point.
>
> An actor and stage-director who had been suffering from total amnesia and a crepuscular condition as a result of famine disease, six weeks from the beginning of his treatment recited to us an entire scene from " William Tell " by heart. It should be mentioned that, to be able to recite it, the patient had to mimic the scene. Suddenly, he lost the thread, he felt " his brain was getting hot ", he was congested. After a quarter-of-an-hour's rest, he was able to go on with his recitation.

Altogether, the atmosphere of confidence and optimism in the cachectic patients' quarters, which hitherto had been so distressing, was profoundly moving.

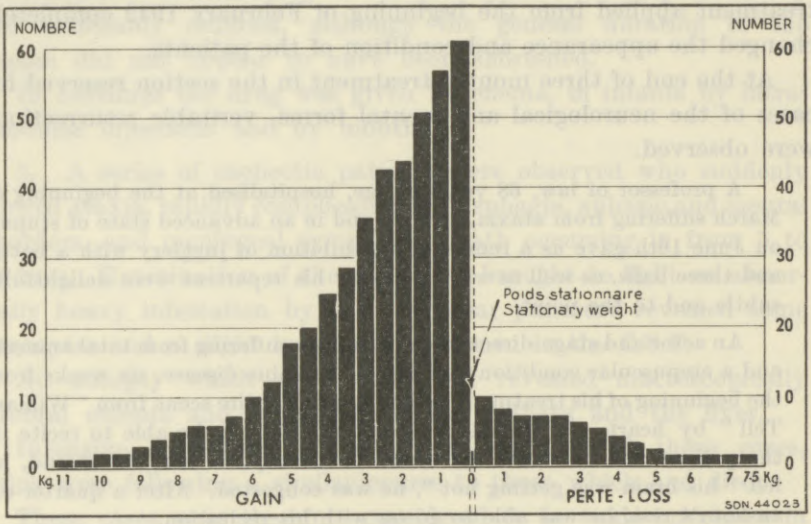
In all the camps from March onwards deaths practically ceased. This decline in mortality was abrupt, once relief was effectively organised.

Strength returned, the patients put on weight. One such patient, weighing only 70 lbs (32 kg.) on his entry into the treatment section increased in the course of a few weeks to 86 lbs (39 kg.), notwithstanding the disappearance of his œdema.

From April 16th to 30th, among 100 cachectic patients in a camp, an increase of weight was recorded in 32%, a stationary condition in 20%, loss of weight owing to decrease of fluid in 40%, and a real loss of weight in 8% of the cases. From May 1st to 15th, the number of cachectic patients who increased in weight reached 60.4%, decreasing slightly (by 17%) in the following fortnight. During the period from June 1st to 15th, there was an increase in 51% of cases, the weight remaining stationary in 19%, and decreasing in 30% ; this decrease was almost completely due to disappearance of œdemas.

Results obtained by Dietetic and Drug Treatment on the Body-weight of " Cachectic " Internees.

(Gains and losses of weight are expressed in kilogrammes.)



Below are some figures of weights recorded in another camp :

	Decrease	Stationary condition	Increase
	%	%	%
March. . . .	26.5	13.5	60
April	18.2	17.5	64.5
May.	48.7	17.7	33.6

Results for May were much less satisfactory than those for preceding months, because 79 new cachectic patients (24.7 %), either coming from prisons or from camps, had to be admitted to the hospital or to the quarters for cachectic patients. What is more, the inadequacy of the basic food rations supplied by the administrative authorities of the camps during this month was absolutely disastrous, consisting as it did mainly of salads and turnips. There were no dried legumes, no potatoes and but small quantities of noodles or macaronis. Further, the inclusion in the basic rations of too large amounts of salt fish (salted sardines) resulted in prolonged disorders due to aqueous retention, from which patients recovered only with great difficulty.

Nevertheless, 37 patients (5%) were able to leave the cachectic patients' quarters and the hospital after two months treatment, and were considered as being provisionally out of danger.

This example of the harmful effect of insufficient and completely incorrect feeding is not unique of its kind. The inadequacy of the ordinary camp fare necessitated a very reserved long-term prognosis.

While, thanks to the efforts of the organisations and to the collaboration of the administrative authorities of the camps in so far as concerned the cachectic patients only, it was possible to banish death from the quarters for cachectic cases, the alarming condition of nutritional deficiency due to the inadequacy of the basic rations provided for internees jeopardised the whole of the work that had been accomplished.

This inadequacy, in fact, was shown by the progressive increase in the number of cachectic cases and in the decrease of the number of convalescents.

The cachectic and pre-cachectic cases able to return to the ordinary life of the camp had relapses because they were exposed to the same pathogenic factor : famine.

Non-cachectic persons, in their turn, began to swell the ranks of the threatened cases, because when their bodily reserves were exhausted they were left more and more susceptible to the same pathogenic factor : famine.

The inadequacy and the irregularities of the rations effectively supplied to the inmates by the administrative authorities of the camps made it impossible definitely to save them. Those who were saved from the consequences of famine at the cost of great effort on the part of the relief organisations in supplying supplements of food would thus be still under suspended sentence of death, unless the camp authorities supplied them with a more or less adequate basic ration, this basic ration remaining the prime factor in the problem of saving famine-disease patients. When all is said and done, the obligation to solve this problem rested with the authorities responsible for the internment of the inmates of the camps.

CONCLUSIONS

Famine disease extended considerably in July 1942, at a time when many internees had spent from 10 to 14 months in the camps. In addition to the camp fare, they had been able to draw on their personal resources, thanks to the receipt of parcels and remittances from abroad. Apart from some serious cases, deficiency had on the whole been mild. The aggravation and the generalisation of the destitution which resulted some months afterwards from the total cessation of despatches from abroad, the more serious situation of the country as a whole, the elapse of five more months of the period of latency preceding the appearance of the symptoms of the disease, and, lastly, the cold, contributed to the almost " epidemic " outbreak of famine disease which swept through all the camps, extending to every class, both sexes and all ages.

* * *

Observation enabled a certain number of typical clinical forms, depending partly on the origin of the internees, to be distinguished, each type having its own characteristics and calling for a different medical treatment.

Energetic measures applied at the outset to a certain extent checked the ravages of the disease.

Prolonged experience showed :

(1) That, with supplementary relief rations and appropriate treatment, it was possible to cure many apparently desperate cases ;

(2) That a lasting result could be attained only if the basic rations supplied by the camps did not fall below a minimum of about 1,200 calories a day, the limit of safety. With a smaller official contribution, private initiative was incapable of making good deficiency of calories, owing both to the inadequacy of its financial resources and to the impossibility of obtaining the indispensable foodstuffs either in the country or abroad.

Thus, notwithstanding the considerable relief work extending over a whole year which, though handicapped by new administrative measures and by the changes in the European situation, was also carried on in 1943, famine disease still flourished, though its " virulence " sharply declined, thanks to the vigilance of the private relief organisations.

One definite result could be recorded—deaths from famine disease practically ceased. There were no more " acute " cases. Famine disease became " a chronic malady ", as though preventive and therapeutic measures had been sufficiently effective to prevent its spreading, but insufficient to cure declared cases.

Observation also showed that, as soon as the action of the relief organisations was restricted, for reasons outside their control, the " epidemic " character of the disease immediately reappeared.

In the camps under observation, the basic rations provided 1,200 calories daily at the end of 1940, and fell to 1,000 calories in 1941, 900 in 1942 and to 845 calories daily in 1943.

The quantity of proteins of animal origin supplied did not exceed 8 g. a day, and did not reach that figure every day ; that of vegetal proteins was 12 g. The ration of fats was calculated on the generous side as 15 g. and carbohydrates at 171 g.

A diet which contained no more than 1,200 calories daily led inevitably to famine disease under the conditions prevailing in the camps of life in general and of cooking in particular.

The deficiency of calories, the inadequate quantities of fats, protcins and minerals, as well as the disproportionate quantity of carbohydrates, resulted in famine disease rather than in forms of vitamin deficiency. It is possible that such forms of vitamin deficiency make their appearance only if the food ration reaches a minimum compatible with life.

If this supposition is correct, the various forms of vitamin deficiency would be observed with the usual symptoms in unconfined groups of the population rather than in the camps. This appears, in fact, to be the case.

Unhygienic mode of living, insanitary quarters, dirt, infestation by parasites, immobility, favour the appearance of famine disease.

Famine disease breaks out in cold weather ; it also appears, with less intensity and in another form (circulatory), during very hot weather ; it is thus more frequent during extreme seasons of the year.

The specific treatment of famine disease consists of substantial food, rich in proteins and fats. Because of the condition of the alimentary tract and the teeth of patients, nutrients should be easily digestible.

Glucose and mineral salts—particularly calcium, phosphorus and iron—are very useful drugs for " starting up " the therapeutic action of an improved diet.

Brewers' yeast, vitamins and liver extracts are necessary for the treatment of certain clinical forms.

The segregation of patients who are seriously ill is indispensable.

The prognosis is directly dependent on the amount of foodstuffs available and their content in proteins and fats, as well as on the promptitude of the relief action.

Untreated, and if the quantitative and qualitative inadequacy of the feeding which is its cause persists, famine disease inevitably ends in death.

Interned populations cannot long be kept alive in camps if the basic food ration allowed to them does not reach the living minimum allowed to the free civil population and if the elementary rules of collective and individual hygiene are not applied to them.

Such are the conditions of their survival.

Annex 1.

VICTUALLING OF THE INTERNMENT CAMP OF R....

Dietetic kitchen for cachectic persons. Fare provided from February 26th to March 4th, 1942.

February 26th: Received from the victualling service by the camp kitchens for 84 persons:

		Calories
12 kg.	turnips	3,000
2.1 „	macaronis	7,287
21 „	bread	56,700
1.4 „	sugar	5,600
21 litres	wine	4,788
1.2 kg.	fat	4,800
2.5 „	jam	1,680
30 „	salads	6,000
		Calories
	Total	89,855 : 87 = 1,070

Food supplement supplied by the American "Friends" and the Swiss Red Cross, per person:

100 g.	flour	340	
5 „	oil	43	
20 „	Gruyère (full-fat) cheese	81	
10 „	of a malted product	40	
¾ litre	fresh milk	525	1,029
	Total per person		2,099

February 27th: Received from the victualling service by the camp kitchens for 84 persons:

		Calories
21 kg.	bread	56,700
2.5 „	jam	1,680
1.35 „	sugar	5,400
1.2 „	fat	4,800
20 „	salads	4,000
22 „	turnips	5,500
2.1 „	macaronis	7,287
21 litres	wine	4,788
		Calories
	Total	90,155 : 84 = 1,073

Food supplement supplied by the American "Friends" and the Swiss Red Cross, per person:

100 g.	rice	340	
5 „	oil	43	
1	tangerine	15	
40 g.	Halva	200	
10 „	special flour	40	
¾ litre	fresh milk	525	1,163
	Total per person		2,236

February 28th: Received from the victualling service by the camp kitchens for 84 persons :

		Calories
21 kg.	bread	= 56,700
1.35 „	sugar	= 5,400
1.25 „	fat	= 5,000
4.2 „	meat	= 4,200
2.5 „	jam	= 1,680
22 „	turnips	= 5,500
20 „	salads	= 4,000
2.1 „	macaronis	= 7,287
21 litres	wine	= 4,788
		Calories
	Total	94,555 : 84 = 1,126

Food supplement given by the American " Friends " and the Swiss Red Cross, per person :

5 g.	oil	= 43
20 „	Gruyère cheese	= 81
10 „	of a malted product	= 40
76 „	macaronis	= 260
40 „	jam	= 28
¾ litre	fresh milk	= 525 977
	Total per person	2,103

March 1st: Received from the victualling service by the camp kitchens for 84 persons :

		Calories
21 kg.	bread	= 56,700
1.35 „	sugar	= 5,400
1.25 „	fat	= 5,000
2.5 „	jam	= 1,680
22 „	turnips	= 5,500
20 „	salads	= 4,000
2.1 „	macaronis	= 7,287
21 litres	wine	= 4,788
		Calories
	Total	90,355 : 84 = 1,076

Food supplement supplied by the American " Friends " and the Swiss Red Cross, per person :

100 g.	rice	= 340
40 „	stewed apricots	= 10
40 „	Halva	= 200
10 „	special flour	= 40
¼ litre	dried milk	= 200
¾ „	fresh milk	= 525 1,315
	Total per person	2,391

March 2nd: Received from the victualling service by the camp kitchens for 84 persons :

		Calories	
21 kg. bread	=	56,700	
2.5 ,, jam	=	1,680	
1.4 ,, sugar	=	5,600	
1.2 ,, fat	=	4,800	
12 ,, turnips	=	3,000	
15 ,, salads	=	3,000	
15 ,, cauliflowers	=	4,800	
4.2 ,, meat	=	4,200	
2.1 ,, macaronis	=	7,287	
21 litres wine	=	4,788	
			Calories
Total		95,855 : 84 =	1,141

Food supplement supplied by the American " Friends " and the Swiss Red Cross, per person :

100 g. flour	=	340	
5 ,, oil	=	43	
100 ,, dates	=	250	
10 ,, of a malted product	=	40	
¾ litre fresh milk	=	525	1,198
Total per person			2,339

March 3rd: Received from the victualling service by the camp kitchens for 84 persons :

		Calories	
21 kg. bread	=	56,700	
1.3 ,, sugar	=	5,200	
1.2 ,, fat	=	4,800	
2.5 ,, jam	=	1,680	
20 ,, turnips	=	5,000	
22 ,, salads	=	4,400	
2.1 ,, macaronis	=	7,287	
4.2 ,, meat	=	4,200	
			Calories
Total		89,267 : 84 =	1,063

Food supplement supplied by the American " Friends " and the Swiss Red Cross, per person :

100 g. rice	=	340	
5 ,, oil	=	43	
40 ,, olives	=	80	
20 ,, Gruyère cheese	=	81	
20 ,, pea flour	=	68	
10 ,, special flour	=	40	
¾ litre fresh milk	=	525	1,177
Total per person			2,240

March 4th: Received from the victualling service by the camp kitchens for 84 persons :

		Calories
21 kg.	bread	56,700
1.3 ,,	sugar	5,200
1.2 ,,	fat	4,800
2.5 ,,	jam	1,680
22 ,,	turnips	5,500
20 ,,	cauliflowers	6,400
2.1 ,,	macaronis	7,287
4.2 ,,	meat	4,200
	Total	91,767 : 84 = 1,092

Food supplement supplied by the American " Friends " and the Swiss Red Cross, per person :

50 g.	pea flour	170	
5 ,,	oil	43	
40 ,,	Halva	200	
10 ,,	of a malted product	40	
100 ,,	dates	250	
¾ litre	fresh milk	525	1,228
	Total per person		2,320

Daily average per internee :

	Calories
Official basic ration	1,091
Supplement of relief organisations	1,155
Total	2,246

Annex II.

RATIONS IN A CAMP FROM JUNE 8TH TO 15TH, 1942

Daily Average.

		Calories
Bread	250 g.	= 625
Fats	4.5 g.	= 41
Sugar	6 g.	= 24
Vegetables, cabbage (in soups)	500 g.	= 125
Canned tomatoes	40 g.	= 14
Dried potatoes (50% usable)	250 g.	= 125
Onion-garlic		4
		958

Note. — The amount of the nutrients served in this camp was calculated by weighing the ration actually distributed.

RATIONS IN A CAMP FROM APRIL 9TH TO 11TH, 1943

	1st day	2nd day	3rd day
Midday.	g.	g.	g.
Soup : water	300	255	275
and solids	145	170	90
(solids composed of cabbage, turnip, beans, kidney beans)			
Vegetable (cabbage, salad)	35	40	65
Evening.			
Soup : water	345	325	250
and solids	145	160	60
(cabbage, salad and 20 g. macaronis)			
			Vegetable :
Meat : gravy	35	40	water 185
and solids	35	45	solids 145

Daily average : 845 calories.

To this list must be added 275 grammes of bread a day, a teaspoonful of sugar a day, and three times a week half a skim-milk cheese.

Annex III.

CAMP X FROM MAY 17TH TO 23RD, 1942

THEORETICAL TOTAL VALUE of the official rations listed :

9,170 calories = *1,310 calories per day.*

EFFECTIVE VALUE according to the weight of the contents of the internee's mess-tin :

	May 16th		May 17th		May 18th	
Midday.	g.	Cal.	g.	Cal.	g.	Cal.
Soup :						
water	323		263		253	
solids (Jerusalem artichokes, turnips)	142	95	170	119	87	60
Vegetable (leeks)	35	7	37	7	63	25
Evening.						
Soup :						
water	323		345		254	
solids (Jerusalem artichokes, turnips)	142	95	144	96	60	30
Vegetable (Jerusalem artichokes) :						
water					18	
solids					145	145
Meat : gravy	38		38			
solids	35	56	44	70		
Bread	270	675	270	675	270	675
Sugar	16	62	16	62	16	62
Fat	15	80	15	80	15	80
Cheese	—	—	—	—	—	—
		1,070		1,108		1,077

Daily average from May 16th to 18th = 1,085 calories.

Annex IV.

MEALS OF CACHECTIC PERSONS IN A CAMP

Official Basic Ration supplemented by the Relief Organisations

May 16th, 1942.

	Grammes	Cal.
Coffee	10	10
Dried milk	14	66
Sugar	10	40
White cheese	220	422
Jerus. artichokes	50	40
Turnips	250	70
Leeks	200	63
Onions	50	22
Kidney beans	70	234
Tapioca	15	52
Oil	19	161
Bread	265	663
Sardines	90	84
Turnips	250	70
Leeks	200	63
Onions	50	22
Jerus. artichokes	30	24
Rolled oats	40	152
		2,258

May 17th, 1942.

	Grammes	Cal.
Cocoa	5	24
Dried milk	25	119
Sugar	13	52
White cheese	220	422
Turnips	250	70
Leeks	100	31
Onions	50	22
Beans	60	36
Barley	50	184
Sago	5	18
Snout	150	237
Oil	10	85
Bread	250	627
Roquefort cheese	80	242
Rice	80	277
Sugar	15	60
Dried milk	10	47
		2,553

POSTSCRIPT.

The changes in the military and political situation in Europe which have taken place since this study was written and set up enable us now to lift the veil of anonymity from its authors, from the organisations to which they belong, from the scene of their labours and from the bodies which supplied them with the wherewithal to carry out their work.

The study is, to a great extent, based on a report dated July 1942 of the "Health Commission" of the "Co-ordination Committee for Relief in Camps", consisting of Dr. René ZIMMER, *representing the Unitarian Service Committee of the United States, Dr. Maurice* DUBOIS, *of the Swiss Red Cross, Children's Relief (Secours aux Enfants) and Dr. Joseph* WEILL, *of the O.S.E. Union (Jewish Health Organisation).*

Dr. WEILL *was obliged very considerably to modify the text of the original report in order to enable it to be published and to incorporate later information in it.*

The camps in which the observations were recorded are situated in the south of France and, more particularly, in the Eastern Pyrences. They were organised in 1939 and 1940, in the first place to accommodate Spanish refugees, whence their name of "Centres d'hébergement", and then, during the first part of the world war, to receive "enemy aliens".

From July 1940, these camps were mainly filled with Jews of various nationalities. During the second half of 1942, tens of thousands of them were deported from the camps to Poland.

The camps designated by the letters "G...." and "R...." are those at Gurs and Rivesaltes respectively. The town the food rations of which have been indicated for purposes of comparison with those of the camps is Marseilles.

The charitable organisations which took part in the provision of relief for the internees were the following:

"Aide aux Emigrés", Swiss Section of the International Migration Service, Geneva;
The Confederation of Swiss Jewish Communities;
The Joint Relief Committee of the International Red Cross;
The Œcumenical Council, Geneva;
O.S.E. Union (Jewish Health Organisation), Geneva;
"Schweizerischer Aerzte-Vercin" (Swiss Medical Union), Zurich;
"Secours suisse aux Enfants", Geneva;
The Society of Friends, United States;
The Swiss Red Cross;
The Unitarian Service Committee, United States.

Furthermore, consignments of medicaments were supplied free of cost by the following manufacturers of pharmaceutical products in Basle: C. BOEHRINGER *& Co., C.I.B.A.,* GEIGY, *and* SANDOZ, *to whom the authors desire to express their gratitude on behalf of the internees who benefited by their generosity.* ED.

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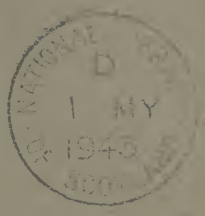

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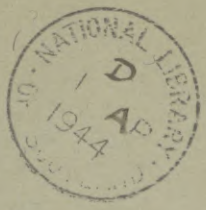

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* To ensure the sequence of the pagination of the English edition of Vol. X, the first page of this bilingual No. 3 is regarded as being numbered 155-201 (154 being the last page of the English edition of Vol. X, No. 2).

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