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TRENDS OF HEALTH IN THE UNITED STATES[1]

By ROLLO H. BRITTEN, *Senior Statistician, United States Public Health Service*

I

In this cursory examination of trends of health in the United States there will be no endeavor to ascertain in what specific instances improvement is due to public health organizations. The progress in many fields is the result of definite scientific advances properly applied. In other cases changes are part and parcel of our altered social existence and, in some instances, of mutations in the natural virulence or invasiveness of the disease itself.

Except for the broadest purposes, little meaning can be derived from mortality rates for the whole population, without regard to age or other factors. The advance in health is customarily measured by such "crude" rates. However, unless some radical changes continue to give us a population abnormally high in the young adult ages (and with the reduction in immigration this seems unlikely), the death rate, unadjusted for age distribution, may be expected to rise, even if there should be improvement in the rate at each specific age.

Most of the material offered is necessarily limited to mortality. Sickness and medical examination data can not reveal the trend of health. For a few of the notifiable diseases, information for the last 15 years will be of value; but even in this group of diseases, inferences as to trend are subject to much error. Yet one of the impressive phenomena of the last 50 years has been the continuous fall in the case fatality of many diseases. Thus a rather untrue impression is obtained from death rates.

A great deal of difficulty will arise from increasing accuracy of diagnosis and incompleteness of birth registration. Especially in the case of cancer and the degenerative diseases, the steady improvement in diagnosis has been so great as to make it almost impossible to determine what the true trends are.

The graphical material must be taken as illustration rather than as evidence. Each individual phase deserves an exhaustive presenta-

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tion; but that is not possible in a broad survey; and, as a matter of fact, no such detailed history of the public health in this country, based on objective social phenomena, has ever been carried out.

The rapid growth of the population of the United States needs to be recalled. At the date of the first census, 1790, there were only 5 cities with populations of more than 8,000. Now, there are 8 with populations of more than 800,000. This phenomenal expansion has a momentous bearing on the sanitary history of this country, because of increasing urbanization, changes in racial make-up, development of industry, and other factors.

One of the fundamental changes in the characteristics of the population has been that of its age distribution, reflecting both new levels of health and the influence of immigration. Between 1850 and the present time the percentage of persons 50 years of age and over has nearly doubled.

II

Requisite documents are not available for a complete history of mortality in this country from pre-Revolutionary days, especially in the case of pioneer populations and the more remote districts. It is to be recalled that registration of deaths in this country has lagged behind that in other leading nations. No national statistics of mortality were collected before 1860, and then only at 10-year intervals by census enumerators, such records being palpably incomplete. In 1880 the registration area was established, but included only Massachusetts, New Jersey, and a few cities. The area became a reality only in 1900, and even then was confined to the eastern and central sections of the country. Mortality records back of this period must be procured from individual cities, and are therefore quite unrepresentative of a country which was then primarily agricultural. Even these are of no great help before the nineteenth century.

Such fragmentary information as is available suggests appallingly high mortality rates in the seventeenth century among colonists. Of 7,500 arriving in Virginia between 1618 and 1625, only about 1,000 were alive at the end of the period (1), giving an annual mortality rate far in excess of 500 per 1,000, a large part of which was due to warfare and starvation.

For most of the eighteenth century we have records by keepers of burying grounds in Boston. They are regarded as quite complete, and indicate an average annual mortality of about 70 per 1,000 population among colored slaves and of 33 among the white population (2). There were wide fluctuations in the rates. During the Revolution—in fact, until 1811—no further records are available for Boston; but in the early nineteenth century the rates had fallen to a

much lower level—21 per 1,000 for the total population during the period 1811–1820.

To illustrate the order of magnitude of mortality in this country in the nineteenth century, curves are given in Figure 1 for Chicago (3)

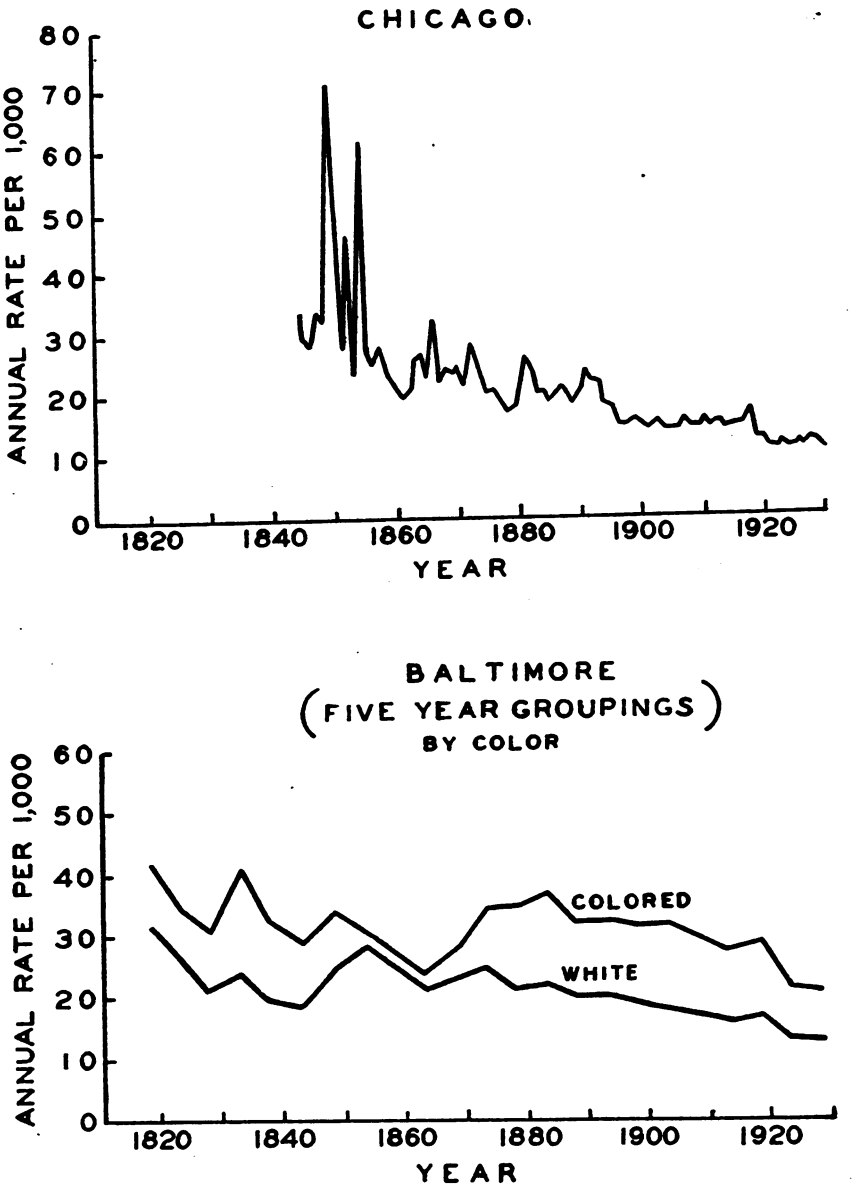


FIGURE 1.—Trend of mortality from all causes in two cities

and Baltimore (4), the latter in 5-year periods. From about 1875 or earlier the rates in the large cities of the United States begin to exhibit a definite downward trend.

One is curious to know whether this downward course was evinced at each age. Retaining Baltimore as an illustration, there is given in Figure 2 the specific mortality rates for some representative ages on semilogarithmic paper (4). Up to 1880 there was no great change in the level of mortality at any age. After that the rate among

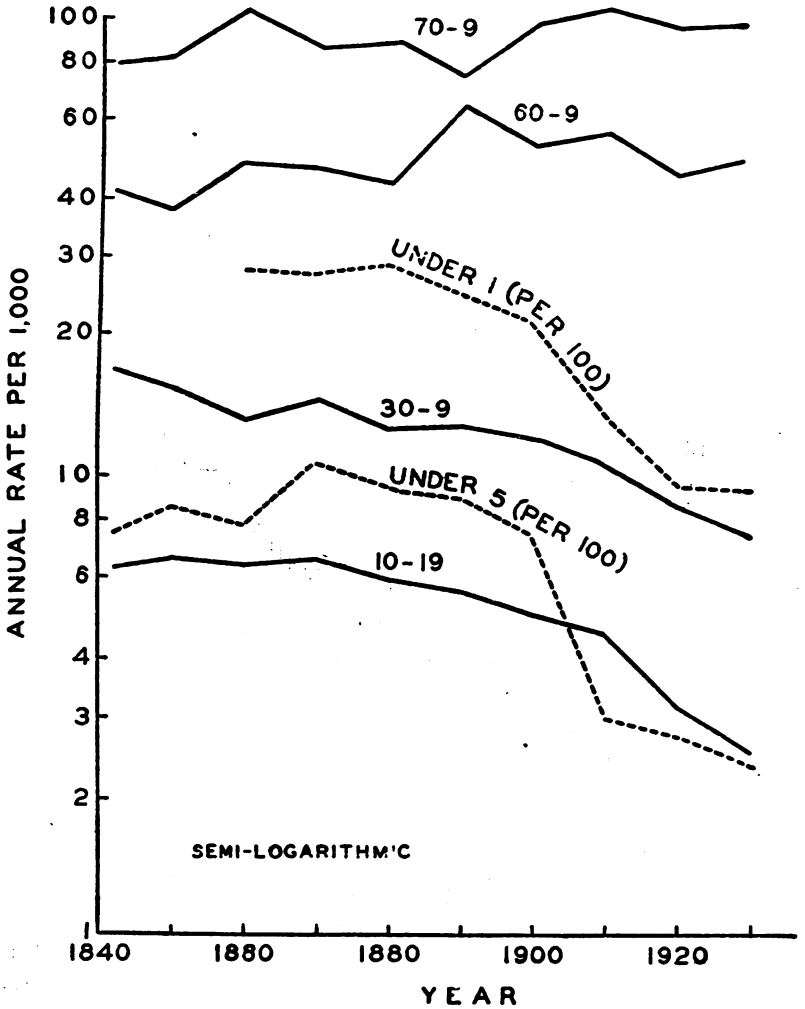


FIGURE 2.—Trend of mortality from all causes in Baltimore, in certain age groups

children and young adults has shown a continuous decrease, whereas that of older ages has remained about the same. This illustrates what will be found to have occurred in large cities generally.

Changes in States as a whole can be adequately represented only for recent years. In order to avoid the factors involved in an expanding registration area, the comparisons (Fig. 3) are confined to the 10 States which were in the area in 1900, these being the New England

States, together with New York, New Jersey, Michigan, Indiana, and the District of Columbia (5). As the purpose is one of illustration merely, only 1900 and 1929 are used; but these two years depict changes which have been in uninterrupted progress during the period. States added to the registration area at later dates also manifest similar tendencies.

It is apparent that in the past 30 years there has been a very great decline in the rate of mortality up to about 50 years of age, with no reduction in the highest ages. A comparison of 1920 with 1929

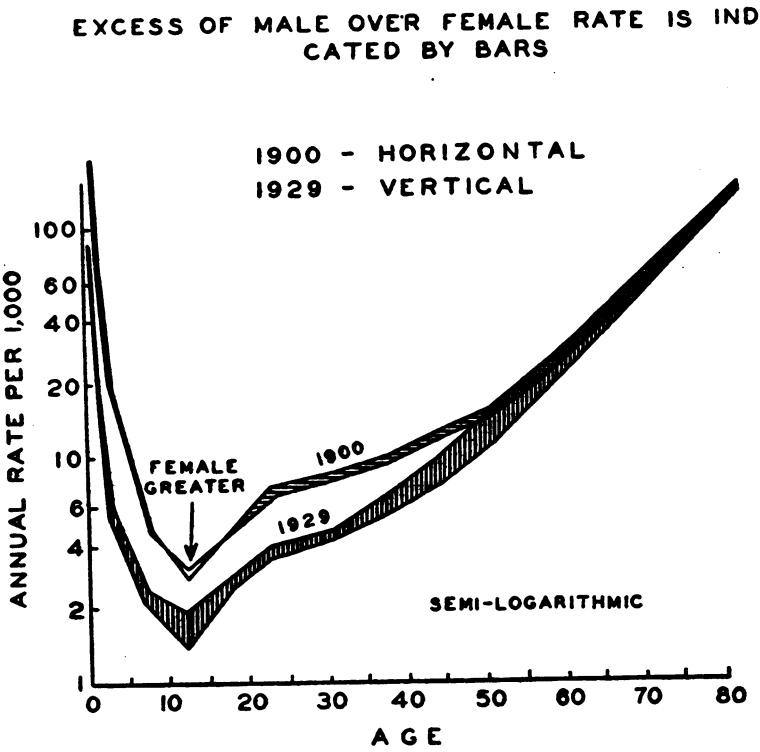


FIGURE 3.—Mortality from all causes by age and sex in original death registration States

indicates that the same tendencies have persisted in the last decade. One significent trend has been the greater improvement in recent years in the mortality among females. The mortality rates are definitely lower among females at each age at the present time, even during the period of childbearing. This was not true in 1900. It is evident that an astonishing change has taken place in the ages between 10 and 20 years—ages at which the male rates tended to be lower than the female in 1900.

Much thought has been given to the favorable mortality showing during the present period of depression. Owing to observed higher mortality in lower wage classes, a rise in mortality might be antici-

pated at the present time. However, previous history in this country will not bear out any such expectation. The major depression cycles in the past fifty years (1875–79, 1884–86, 1894–98, 1914–15) have tended to show general subnormal mortality rates (6). The fact that the present phenomenon is by no means unique, suggests that there may, at the end, be an upward turn in mortality requiring increased vigilance on the part of public health organizations.

No satisfactory comparison over a long period of years can be made for sickness or general physical condition, but it is necessary to remember at each point of discourse that mortality is not an adequate index of real health.

The rates for mortality under 1 year of age employed so far are based upon the enumerated population, which is notoriously uncertain at this period of life. A more satisfactory measure is the relation between the infant deaths and the births. In the United States this index can be employed only since the establishment of the birth registration area in 1915, because previous to that year births were not adequately reported. There has been a continuous decrease in infant mortality since that time, the level of the rates changing from about 100 per 1,000 live births to about 60.

III

The most conspicuous phenomenon in the history of public health in this country has been the reduction in the mortality from acute communicable disease. This tendency is not constant throughout the period of our study, nor in all parts of the country. Among the colonists in Virginia in the seventeenth century, mortality was extremely high from intestinal diseases, winter epidemics which may have been influenza, beri-beri, scurvy, and later, smallpox; but the group of common acute communicable diseases, such as measles, scarlet fever, and diphtheria, do not seem to have been mentioned in the historical records (1). In the eighteenth century, smallpox was extremely prevalent in this country. For instance, in 1721 in Boston alone there were 850 deaths recorded from this cause, giving an annual mortality rate of nearly 8,000 per 100,000 (2). At least five other severe epidemics of smallpox occurred during the century. However, records do not show inordinate mortality from scarlet fever, diphtheria, or measles. But in the nineteenth century mortality from these conditions increased to unprecedented altitudes. In Chicago diphtheria reached a height of 290 deaths per 100,000 in 1880 and scarlet fever of 270 in 1859 (3). In the Middle West and in the South there was a mortality rate from malaria far beyond anything which we can imagine to-day, accustomed as we are to the ravages of this disease. In fact, "sickness" and "malaria" were nearly synonymous. In Chicago in 1854 the mortality rate from "malaria"

was 105 per 100,000. Yellow fever and cholera are two diseases from which there were outbreaks in certain parts of the country, expecially along the coasts.

The order in which the most common of these diseases declined to constant low levels is of interest. No annual rates in Chicago have been more than 10 per 100,000 since 1891 for malaria, since 1895 for smallpox, since 1907 for whooping cough, since 1913 for typhoid fever and measles, since 1919 for scarlet fever, and since 1923 for diphtheria.

Without reproducing curves for the mortality from all these diseases, it is desired to call attention to the vast epidemic waves that characterized their course, the rapidity of the decline once it set in, and the relatively low levels at the present time. Curves for malaria and smallpox are given in Figure 4, as examples of two diseases where both incidence and mortality in an urban area have fallen in extraordinary degree (3).

The trend of mortality from different causes in recent years can be exhibited for large sections of the United States. Figure 5 gives the rates for the original registration States for 1900, 1910, 1920, and 1929 for two of these diseases (diphtheria and typhoid fever) (5). The recession in mortality is literally phenomenal in each age, even in this limited period of 30 years.

With respect to the incidence of these diseases our material must be confined to the recent past, but it will be sufficient to show that, in some, incidence has fallen with mortality, while in others, the disease itself seems to be nearly as prevalent as before. In the first group may be placed typhoid fever, diphtheria (in the last few years), and malaria (in many sections of the country). In the second group may be placed scarlet fever, measles, and smallpox.

The graphs for diphtheria and typhoid fever (Fig. 6), showing the trend of reported cases since 1912 in three eastern cities (7), serve as an illustration. Other cities with adequate reporting mechanism have similar tendencies. Although an increase in the incidence rate over a period of time might mean better reporting, it is manifest that a decrease is more likely to mean a real reduction in the prevalence of the disease.

I have not shown the course of case fatality rates themselves, but one of the important indications is the reduced fatality of a number of these diseases. This must be particularly true in the case of scarlet fever. If the present fatality rate of about 2 per cent had been true in Chicago in 1859, with a mortality rate of 270, one seventh of the population would have had the disease during the year—which is not possible, when the age incidence of scarlet fever and the possible number of susceptibles are considered. Reduced virulence of many

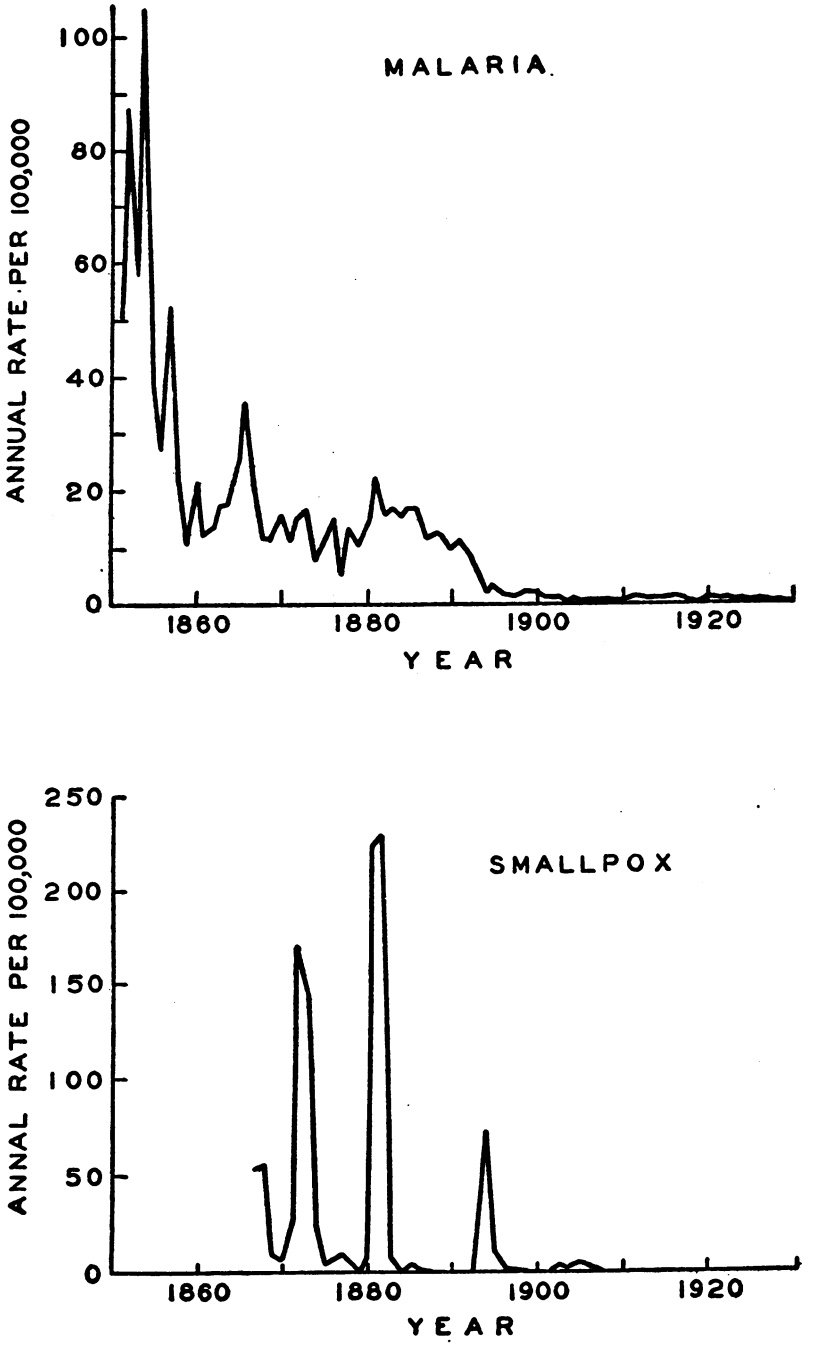
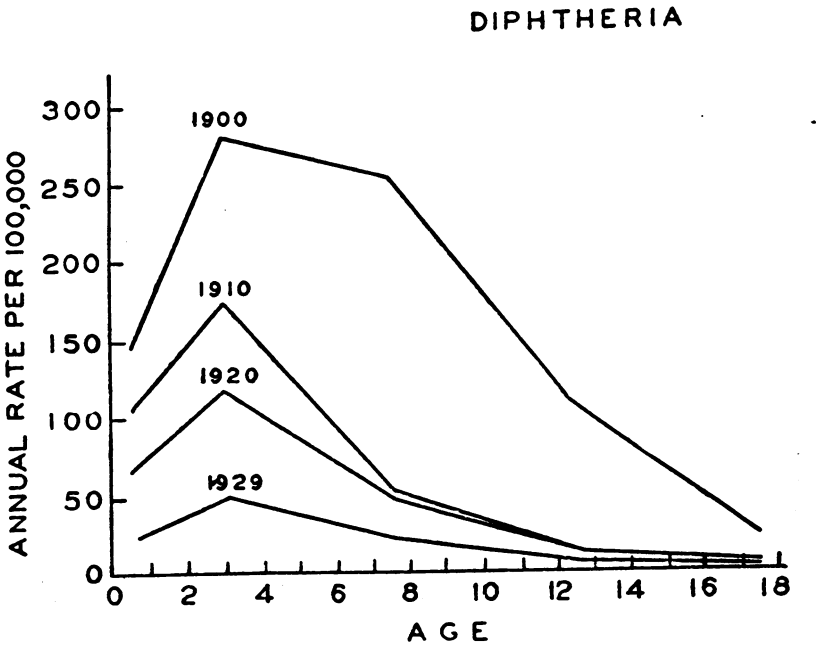


FIGURE 4.—Trend of mortality in Chicago from two specified causes



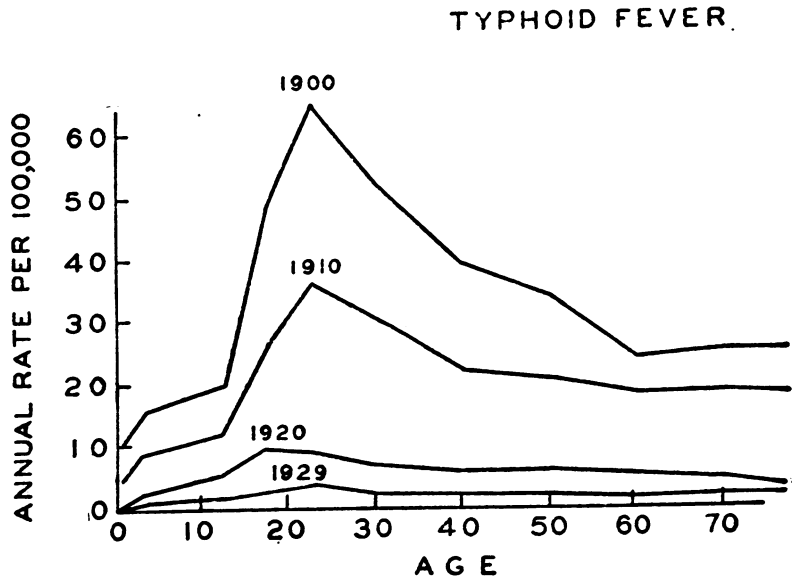
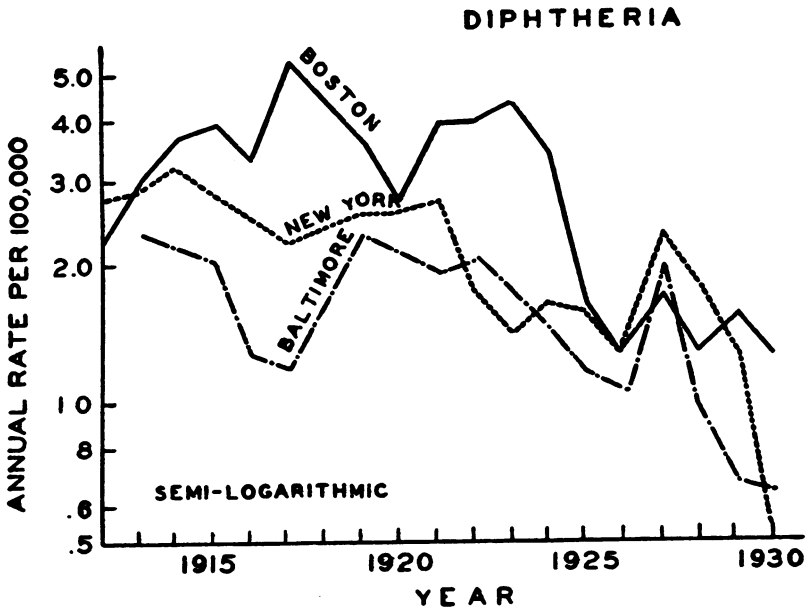


FIGURE 5.—Mortality by age (males) from two causes in original registration States, 1900 1910, 1920, and 1929



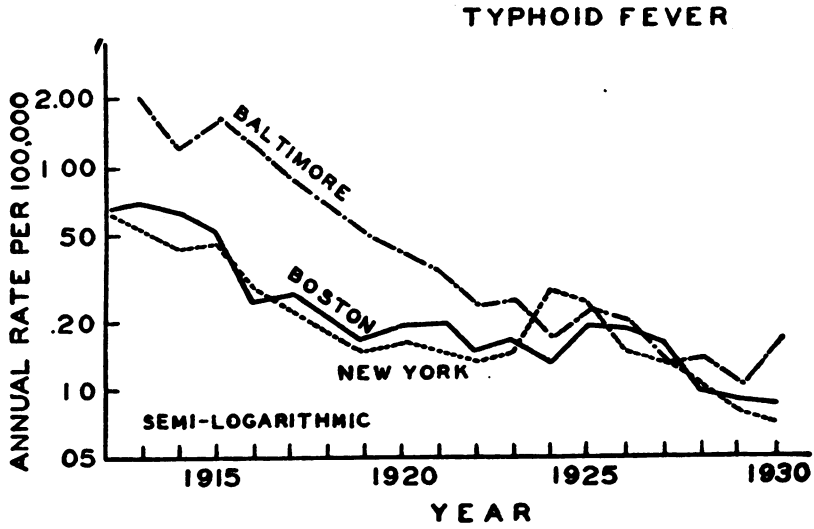


FIGURE 6.—Trend of reported incidence for two causes in three eastern cities

communicable diseases is one of the world-wide tendencies of the present epoch.

Influenza occurs in vast, uncontrolled waves, of which we have, in other countries, rather definite information back into the seventeenth century. During the period of detailed statistics in the United States

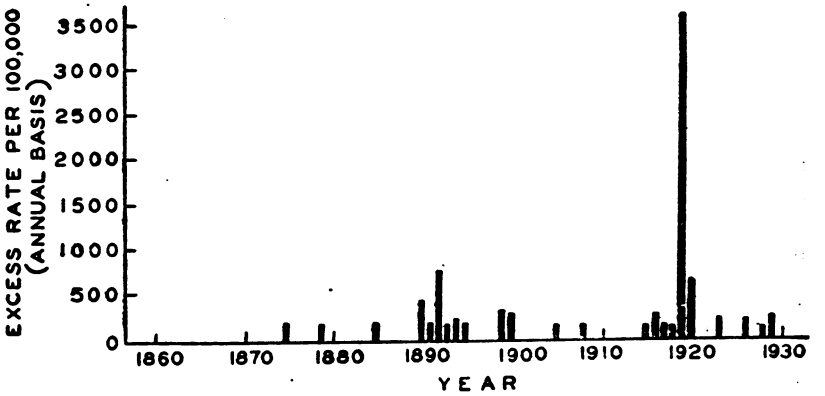
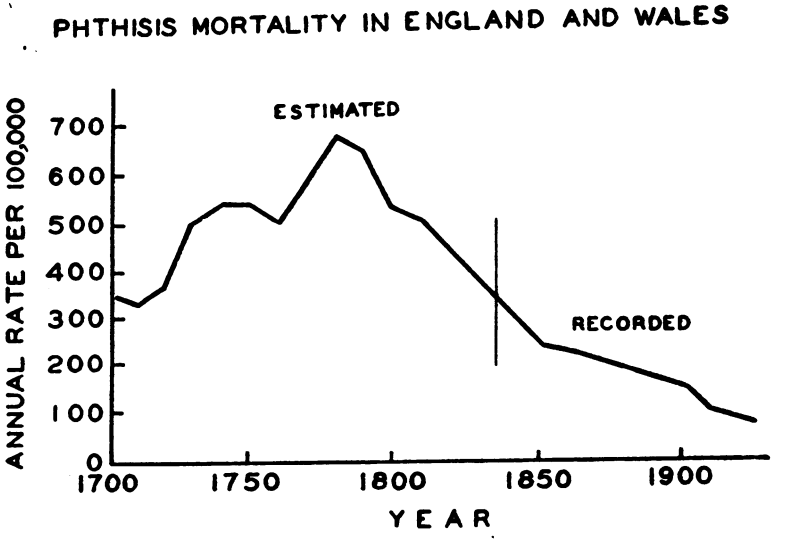


FIGURE 7.—Excess mortality from influenza-pneumonia in Massachusetts, 1857-1930—monthly maximum in each year (July-June) if 100 or more

there have been two major epidemics, one in 1889–1893 and another in 1918–1920. Figure 7 presents a picture of the course of mortality from influenza-pneumonia in Massachusetts since 1857 (8). From the annual rates for each month was subtracted the normal seasonal curve, and in this graph is given the maximum excess monthly rate in various years (July–June), leaving out cases in which this maximum excess did not reach 100 or more deaths per 100,000, such deviations being regarded as possibly a matter of chance.

Available data for the latter part of the nineteenth century do not indicate any great change in the risk of mortality from childbearing; but the material is quite unsatisfactory, because the true risk can be expressed only in terms of births and the births were not adequately registered. In the last 15 years there has apparently been little change in the level of mortality from puerperal septicemia and other puerperal causes, when related to the number of births. However, it is felt that the mortality from puerperal septicemia is much less than in the period prior to the discovery of its infectious nature.

One of the outstanding facts in our medical history is the decrease in mortality from tuberculosis. This is a world-wide phenomenon, like so many which we have discussed, and has been in progress, one suspects, for at least a century. The curve at the top of Figure 8 gives the trend of the mortality from this disease in England and Wales since 1700. Up to 1838 the proportion due to phthisis in London is applied to the whole country (9). By the end of the eighteenth cen-



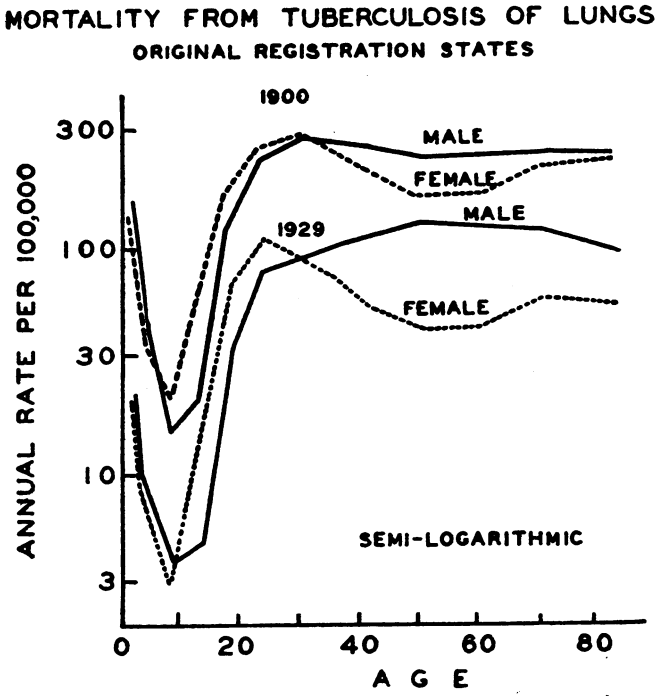


FIGURE 8.—Phthisis mortality in England and Wales and mortality from tuberculosis of lungs, origina registration States

tury the rate appears to have reached the unexampled level of about 700 per 100,000; since then it has shown a continuous decline.

The decrease in this country, as shown by available records, has been similar, and has occurred in all groups of the population, even among

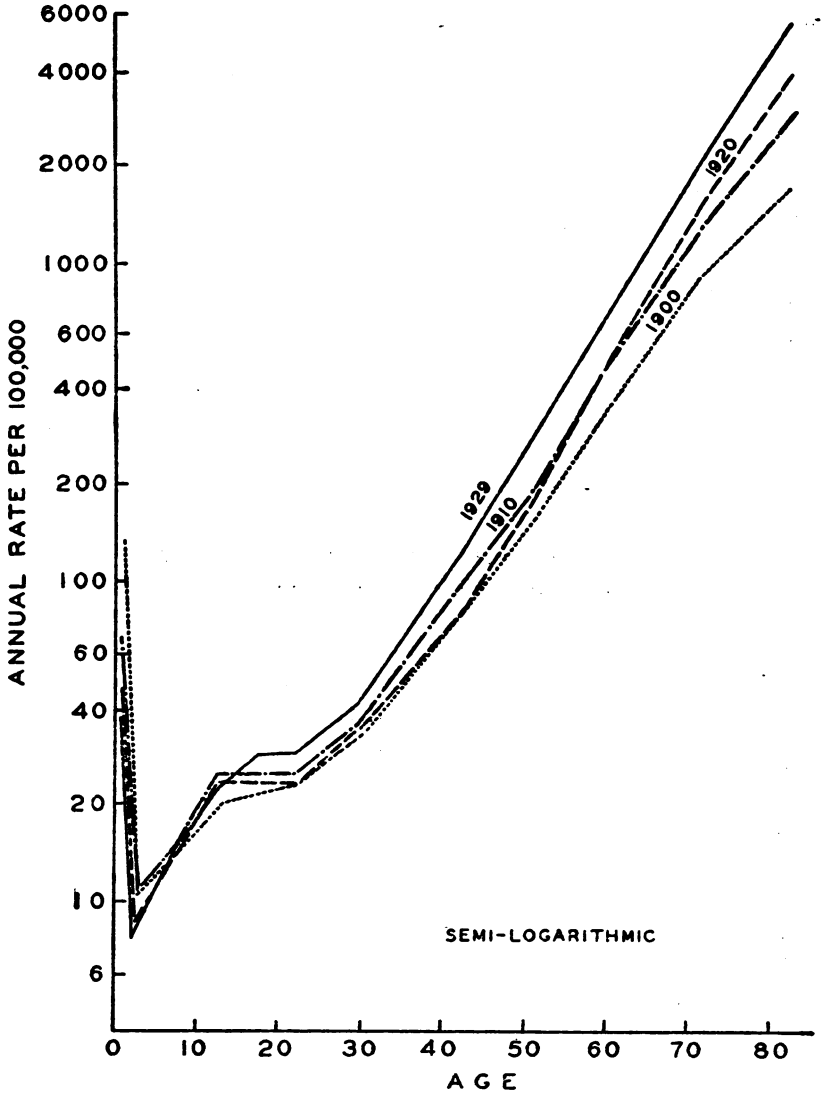


FIGURE 9.—Mortality from "organic" heart diseases in original registration States by age, 1900, 1910, 1920, and 1929

the colored, where the mortality has been so excessive. Perhaps of greatest interest at this moment are the tendencies to be noted at different ages in the two sexes. In Figure 8 (bottom) the age curve of mortality by sex in 1900 is given, as compared with 1929, for the original registration States (5). One is struck by the continuation of

the decrease in the mortality rate, the change at each age being about proportional, and by the accentuation of the differences between the two sexes.

With the lessening in the rate of mortality in the younger portions of our population, attention is more and more focused upon those diseases associated with the breakdown of the bodily organs. The general trend of these diseases is regarded as being upward, even when the age factor is taken into account. Over any long period of time it is difficult to judge this point for specific diseases. What we do know is the picture already presented: Little change in mortality among the older ages in the last 50 to 75 years, and possibly some increase. Actually, however, improvement in diagnosis in this class of diseases is a factor of undecipherable extent. In the thought that after 1900 the effect of this improvement in diagnosis may be less pronounced than previously, attention is centered on the last 30 years.

Organic diseases of the heart form a group of vital interest. Because of changes in classification, it seems preferable to consider this category as a whole. Figure 9 portrays the mortality by age for the original registration States for 1900, 1910, 1920, and 1929 (5). These years serve for illustrative purposes, but probably both 1920 and 1929 are a little too high because of influenza epidemics. The graph suggests that there has been a marked decrease among young persons. The constant rise in the adult population, becoming more and more marked as the older ages are reached, may or may not be real.

The mortality rates from cancer for specific ages continue to rise; the rates for the age group 45–54 in the four years we are considering were 139, 168, 174, and 186. Part of the increase is a matter of improved diagnosis. Is all of it? Whatever the trend in specific ages, it is evident that cancer is forming a greater and greater problem in public health because of the large proportion of people reaching ages at which it is prevalent.

Automobile accidents do not appropriately come under this discussion, but the unwonted increase in this form of death makes a reference to it desirable. Generally speaking, the rise in the rate appears to be roughly proportional to the increased use of automobiles. The rate is now more than 25 per 100,000.

One of the visible modifications associated with the mortality trends which have been discussed is a shift in the seasonal distribution of disease. Formerly mortality was greatest in the summer; now it is greatest in the winter. In Figure 10 two periods are contrasted to make this point clear (10).

There are many conditions, such as the common cold, that can form no part of this history because of the impossibility of determining the trend. Others have necessarily been omitted for lack of space. In most of the comparisons it has also been necessary, because of the broadness of the historical sketch, to consider the population as a

whole. It would be interesting, however, to trace the course of health more adequately in certain groups of the population; for instance, among industrial workers.

A review of this character should really be concerned with positive health, something which is being measured to-day in a degree through

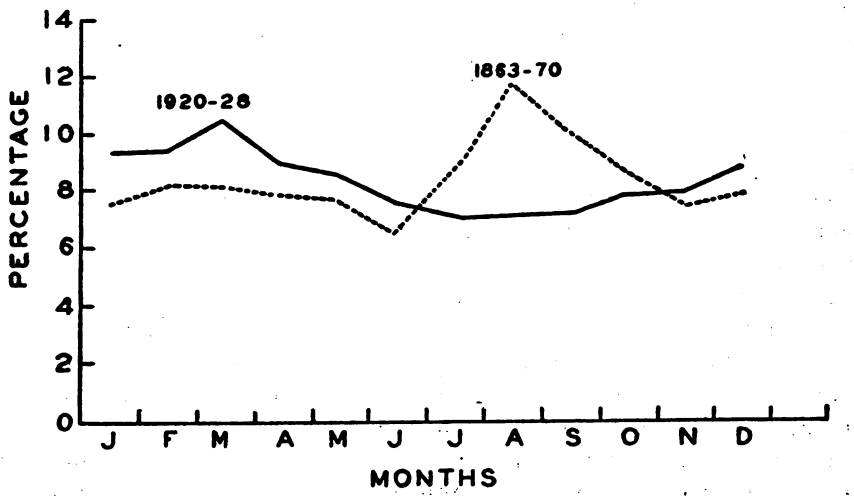


FIGURE 10.—Seasonal distribution of deaths from all causes in Massachusetts, 1863–1870, and 1920–1928

our sickness surveys and our medical examinations. But no comparison with the past in this regard has the slightest meaning, because no data exist for previous periods, and because, even if they did, there would probably be no basis of comparability.

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(10) Calculated from reports of State Registrar and from data furnished by Vital Statistics Division, Bureau of Census. Percentage distribution of the *median* number of deaths in any one calendar month for the two periods.

THE INFLUENCE OF OXYGEN TENSION ON THE RATE OF AUTOLYSIS OF CERTAIN MALIGNANT TUMORS AND NORMAL TISSUES

By MARY E. MAVER, *Biochemist*, J. M. JOHNSON, *Senior Chemist*, and CARL VOEGTLIN, *Chief of the Division of Pharmacology, National Institute of Health, United States Public Health Service*

In a recent paper Voegtlin and Maver (1932) have shown that the oxygen tension is a controlling factor in the autolysis of two typical transplantable neoplasms of the rat. Evidence was also presented indicating that molecular oxygen in the presence of naturally occurring catalysts (copper) has a tendency to oxidize the SH group of glutathione and tissue proteins. The work of Grassmann, v. Schoenebeck, and Eibeler (1931), and of Waldschmidt-Leitz, Purr, and Balls (1930) has demonstrated that reduced glutathione "activates" [1] certain plant and animal proteases, when digestion is carried out under ordinary conditions. Therefore, Voegtlin and Maver attribute the increased autolysis which they observed under conditions of greatly reduced oxygen tension to the slower rate of oxidation of the "activating" SH groups. This viewpoint received further support from experiments dealing with the proteolytic action of papain on substrates containing protein sulphydryl groups (PSH) or substrates free from SH but supplemented by the addition of reduced glutathione (GSH). All of this work dealt with the determination of the degree of proteolysis after a period of about 22 hours' digestion (pH-activity curves). It was stated in the first paper that experiments dealing with the *rate* of proteolysis would be reported at an early date. The purpose of the present communication is to describe this additional evidence. The experiments were carried out with the same two tumor strains and the same papain systems as used in the previous work. It seemed desirable to include also data on a few normal tissues for comparison with the cancer tissues.

EXPERIMENTAL PART

The technic of the preparation of the digestion mixtures and the pH buffers was the same as described by Voegtlin and Maver (1932). It was considered desirable to supplement the Sörensen amino nitrogen titrations by estimations of the remaining undigested protein. For this purpose the digests were treated with trichloracetic acid so as to obtain a final concentration of 16 per cent trichloracetic acid. The suspension was filtered until a clear filtrate was obtained. Aliquots of the filtrate were analyzed for total nitrogen according to Koch and McMeekin (1924). This method yields information as to

[1] The term "activation" merely means that the presence of certain SH compounds promotes proteolysis The mechanism responsible for this increased proteolysis by SH compounds is not satisfactorily explained. It appears to have some relation to the inhibiting action on proteolysis exerted by certain heavy metals.

the rate of disappearance of coagulable protein due to the action of proteinases upon the proteins of tissues.

The data in the first paper concerning the relation of SH compounds to proteolysis were based on qualitative tests by means of the delicate nitroprusside test for the presence or absence of SH. The only quantitative estimations of SH were made on the system papain-fibrin-reduced glutathione. It was obvious, therefore, that quantitative methods were needed in order to establish more forcibly this relationship between SH groups and proteolysis. Present knowledge indicates that the SH groups in tissues are attached to glutathione and certain tissue proteins. Cysteine apparently occurs only in traces. In a paper, which will soon appear, Johnson and Voegtlin [1] will report on experiments in which they have submitted the various methods for the quantitative estimation of GSH in tissues to a critical test and have arrived at the conclusion that the iodometric titration, using nitroprusside as an end-point indicator, yields reliable results. As regards the estimation of protein SH (PSH), no suitable methods which could be applied to the present problem have yet been devised. Mirsky and Anson (1930) have attempted to estimate the SH groups in denatured proteins. Their paper has appeared so far only in abstract form. In a paper from this laboratory, Rosenthal (1932) has shown by quantitative experiments that PSH reacts with arsenious oxides to form very firm combinations. However, this technic is hardly suited for the large number of estimations required in studies on proteolysis. Two of the present authors (Voegtlin and Johnson), therefore, have worked out a method which permits the estimation of the total SH concentration (GSH + PSH) in tissue extracts. This method, an account of which will be published in the near future, is based on the iodometric titration in an acid medium, using nitroprusside as end-point indicator. The results obtained with this method are not quite as reliable as those obtained in the GSH titration, but they are sufficiently reliable for comparative experiments. More detailed information could undoubtedly be secured by the application of the new method for total SH concentration and a separate estimation of GSH. However, in view of the fact, established by Voegtlin and Maver (1932), that both GSH and PSH appear to be "activators" of certain tissue proteases, the present studies were made by applying the estimation of total SH to the digests.

In order to reduce the O_2 tension during the digestion period, the digestion mixtures were placed in Thunberg tubes and the air was removed by an efficient vacuum pump (Cenco type). The actual O_2 tension of these digests before and after incubation was not measured, as the purpose of the work was merely to show whether or not the *rate* of proteolysis was markedly influenced by a great reduction in O_2

[1] This paper will appear elsewhere.

tension. For comparison, samples of the same digestion mixtures were exposed to atmospheric air in small Erlenmeyer flasks provided with cork stoppers. The tissues were obtained in the morning of the day of the experiment by decapitating and exsanguinating the animals. In the case of the liver it seemed advisable to remove the remaining blood as far as possible. This was done by short perfusion through the portal vein with a Ringer solution containing 9 g NaCl, 0.42 g KCl, and 0.24 g $CaCl_2$ per liter. The skeletal muscle and tumors contained only small amounts of residual blood. We are indebted to Dr. J. W. Thompson, of the National Institute of Health, for a supply of rats carrying the Jensen sarcoma and the Walker carcinoma No. 256. Tumors were selected which showed only a moderate degree of central necrosis. The necrotic portion and capsule were discarded. The preparation of the digestion mixtures, after the death of the animal, was done as expediently as possible, and required one to two hours before digestion at 37° C. was begun. Toluene was used to prevent bacterial growth. Samples of digests were removed after 2, 4, and sometimes 8 hours, and on the next morning (about 22 hours). Separate formol titrations, according to Sörensen, were made on duplicate samples; duplicates were also used for coagulable protein and for total SH concentration. Similar estimations were made, of course, on samples immediately before digestion was begun. In the charts illustrating some of the experiments which were performed the increase in amino nitrogen is always expressed as the difference between the undigested control samples and the digested samples, in terms of number of c c N/20 NaOH. The decrease in coagulable protein (whole protein) is given in terms of number of milligrams protein nitrogen, the first point on the curves representing the actual amount of protein nitrogen at the beginning of the experiments. The scale for the total SH concentration of the digests, in milligrams, is placed on the right side of the charts.

DISCUSSION OF RESULTS

Charts 1 and 2 illustrate some of the experiments done on the autolysis of the rat carcinoma and sarcoma. The results varied quantitatively from experiment to experiment, but the main features were the same in all experiments. During the first two hours of digestion the total SH concentration remains high under greatly reduced O_2 tension; in fact, there is a tendency for a marked increase of SH concentration. This increase is probably due to the high reduction potential of the tissue extract, which under reduced O_2 tension may reduce part of the sulphur which was oxidized during the preliminary aerobic preparation of the digests. On the other hand, the digests exposed to the atmospheric O_2 tension show a rapid decrease in the total SH concentration during the first two hours, and this

decrease progresses rapidly during the next few hours, reaching zero within eight hours in the experiment illustrated by Chart 2. At the end of the digestion period (22 hours) the digests exposed to the reduced O_2 tension show a much higher SH concentration than those exposed to O_2 at atmospheric pressure. As regards the digestion of protein, the charts show conclusively that the coagulable protein disappears from the digests at a greater rate when the O_2 tension is

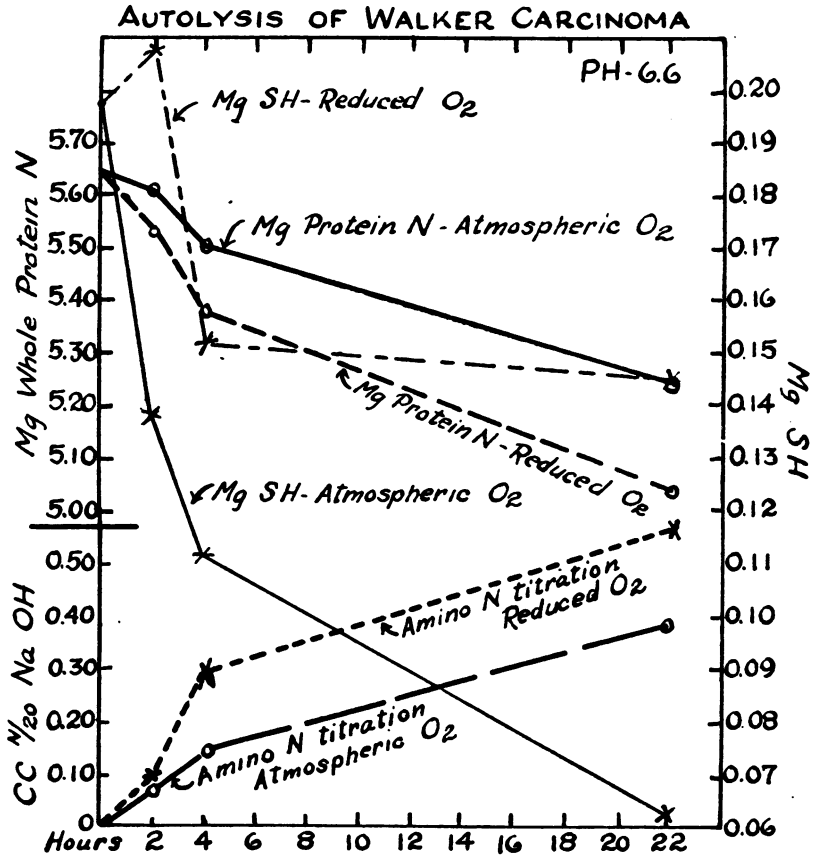

CHART 1.—Autolysis of Walker rat carcinoma 256. Fifty grams of fresh tissue from tumors 3 weeks old were ground with cleaned sand and extracted with 50 c c of glass-distilled water. The extract was filtered through cloth and the volume of the extract was 70 c c. Each autolyzing mixture contained 1 c c of this extract and 2 c c of McIlvaine buffer (citrate-phosphate) at pH 6.6, and 0.2 c c toluol. One-half of the digestion mixtures were placed in Thunberg tubes and evacuated to 7 mm. air pressure over mercury. The remainder were placed in 25 c c Erlenmeyer flasks and corked.

greatly reduced below that of atmospheric air. The lower two curves in the charts clearly show that the rate of increase of protein split products during digestion, on the whole, is greater under reduced O_2 tension, particularly during the first four hours. It is well to point out that these experiments were carried out within the pH range characteristic of these tumors in living animals. We may conclude that, under the conditions of these experiments, the rate of proteolysis

is favored by greatly reducing the O_2 tension, and it would seem that this result is consistent with the slower rate of oxidation of SH groups, which apparently promote the action of certain proteolytic enzymes.

Chart 3 illustrates the autolysis of the skeletal muscle of the albino rat when the pH of the digests is adjusted to 6.6. Preliminary estimations of the pH of this tissue in the living animal by means of the glass electrode indicate that the pH of the tissue is considerably on

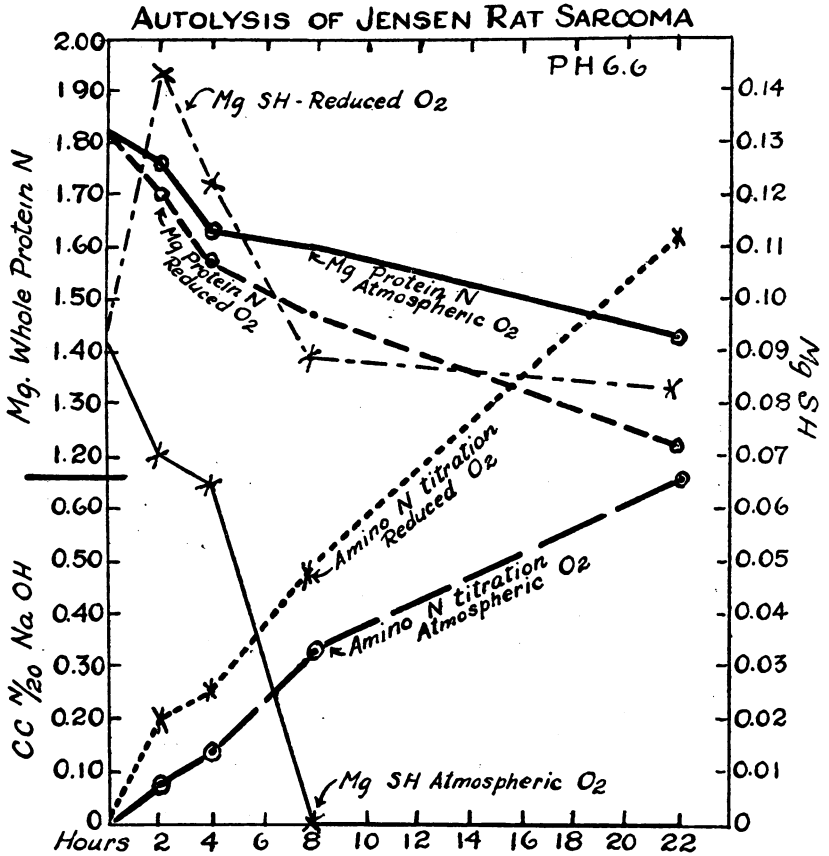

CHART 2.—Autolysis of Jensen rat sarcoma. Seventy-six grams of rat tumors 28 days old were ground with cleaned sand and extracted with 76 c c of glass-distilled water. The extract was pressed through cloth. The volume of the filtrate was 100 c c. Each autolyzing mixture contained 1 c c of this extract, 2 c c of McIlvaine buffer at pH 6.6, and 0.2 c c toluol.

the alkaline side of 7. In order to make a comparison of the autolysis of skeletal muscle with that of the previously described experiments on malignant tumors, pH 6.6 was chosen. It will be noted from Chart 3 that the total SH concentration during the first two hours remains unchanged, whether the digestion is carried out at atmospheric or reduced O_2 tension. Digestion of protein, however, proceeds under reduced O_2 tension, but not under atmospheric O_2 tension. During the two to four hour period the SH concentration

under reduced O_2 tension increases, whereas it decreases under atmospheric O_2 tension. Simultaneously, the coagulable protein decreases further under reduced O_2 tension, and there is a slight indication of increase in coagulable protein under atmospheric O_2 tension. In the latter case, at the end of 22 hours the protein is present in almost the same amount as at the beginning of the experiment. The increase

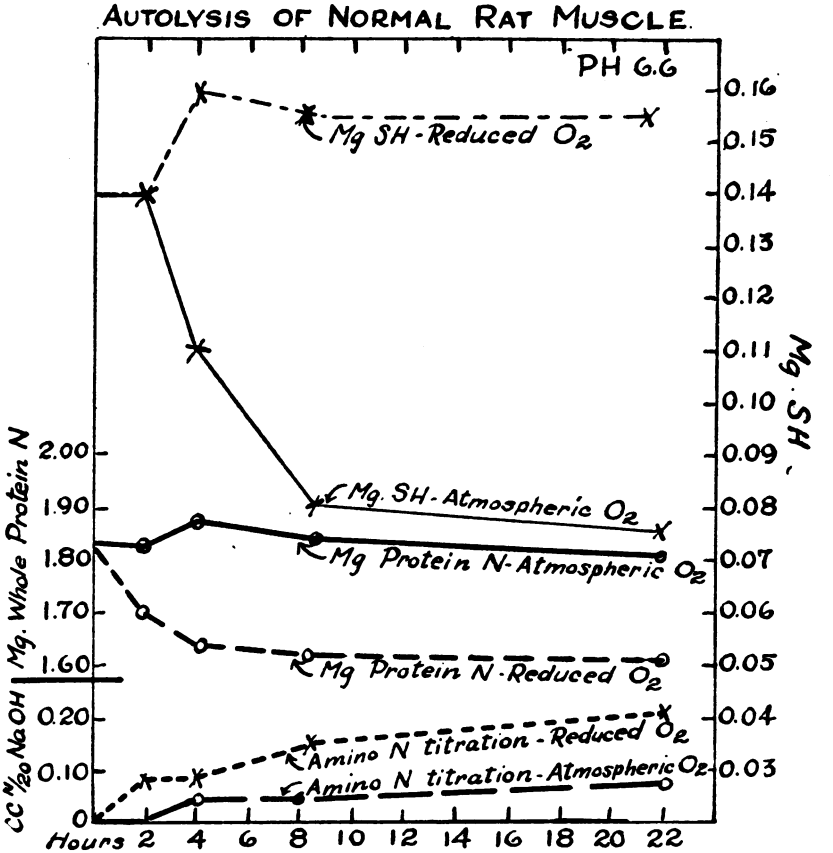

CHART 3.—Autolysis of rat skeletal muscle. One hundred and eighteen grams of fresh rat skeletal muscle were ground with sand and extracted with 100 c c glass-distilled water. The extract was filtered through cloth. The volume of the filtrate was 90 c c. Each autolyzing mixture contained 1 c c of the muscle extract and 2 c c of McIlvaine buffer at pH 6.6 and 0.2 c c toluol.

of protein split products (lower two curves) again shows a slightly greater rate of digestion under reduced O_2 tension.

The autolysis of rat liver at pH 6.6 and 7.6, respectively, is illustrated by Charts 4 and 5. Here again proteolysis takes place at a greater rate under reduced O_2 tension, and the rate of decrease of SH concentration is less than under atmospheric O_2 tension.

Chart 6 gives the results obtained in the study of a digestion system in which the SH groups are represented exclusively by PSH.

Here, too, the same general relationship holds between rate of proteolysis, O_2 tension, and SH concentration.

The last system studied is the action of papain on blood fibrin in the presence of added GSH. (Chart 7.) The results obtained need no further comment, as they are of the same general nature as those in the preceding systems.

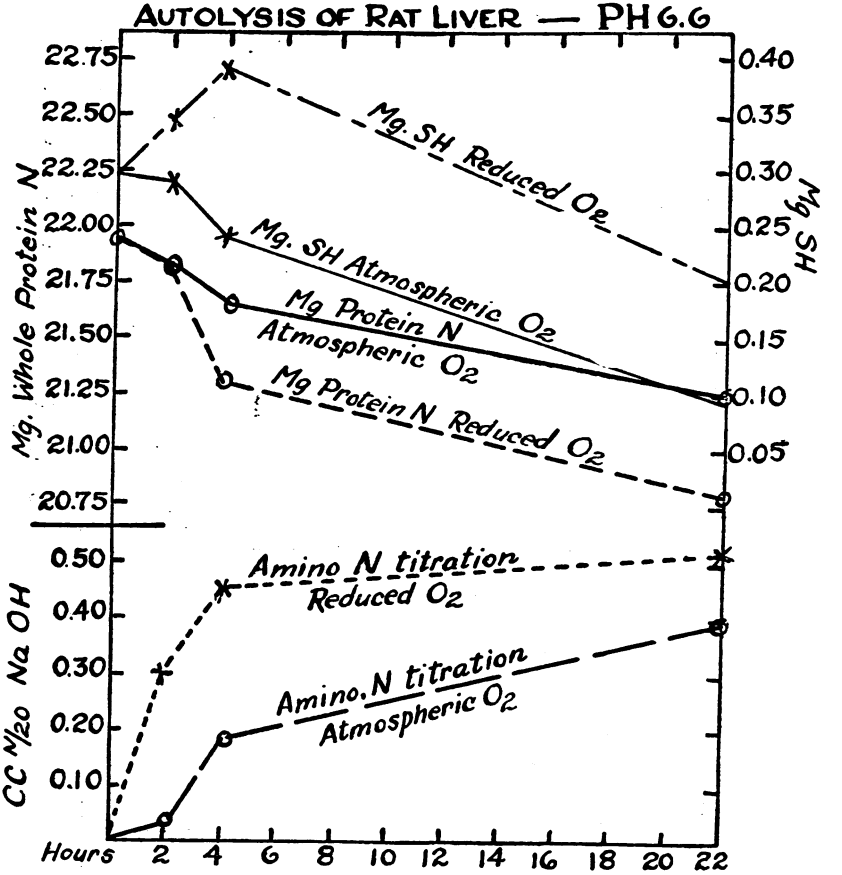

CHART 4.—Autolysis of normal rat liver at pH 6.6. The livers of normal rats were perfused with Ringer's solution containing no sodium bicarbonate or dextrose to wash out as much blood as possible. Sixty-three grams of these perfused rat livers were ground with sand and extracted with 90 c c glass-distilled water. The extract was filtered through cloth. The volume of the filtrate was 120 c c. Each autolysis mixture contained 1 c c of this liver extract and 2 c c of McIlvaine buffer at pH 6.6, and 0.2 c c toluol.

It will have been noticed that the SH concentration in some of these experiments declined considerably even under the greatly reduced O_2 tension produced by an efficient vacuum pump. It is difficult to decide whether there was a sufficient amount of residual molecular oxygen or possibly a slight leakage of the Thunberg tubes to account for this oxidation or whether the oxidation of SH was

brought about by some easily reducible components of the system. At any rate, it is certain that the method used for reduction of O_2 tension was sufficient to bring out marked differences both in rate of oxidation of SH and rate of proteolysis.

In these experiments only a few time intervals were used for estimating the rate of change. In work which is in progress a more

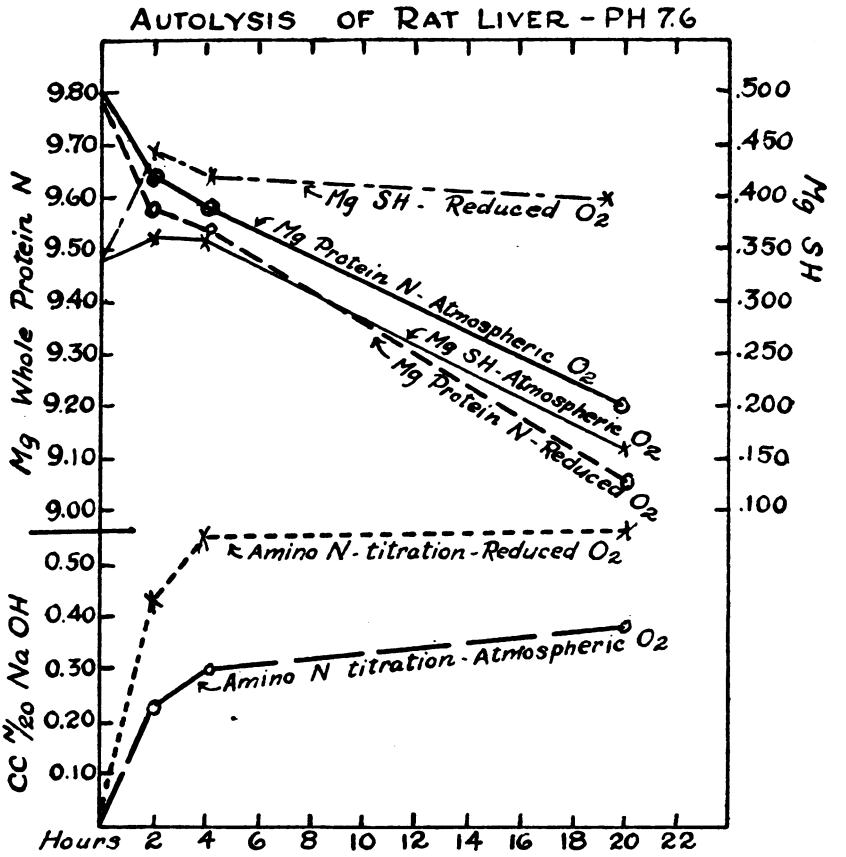

CHART 5.—Autolysis of rat liver at pH 7.6. Fifty-two grams of perfused rat livers were ground with sand and extracted with 75 c c glass-distilled water. The extract was filtered through cloth. The volume of the filtrate was 100 c c. Each autolyzing mixture contained 1 c c of this liver extract and 2 c c of McIlvaine buffer at pH 7.6, and 0.2 c c toluol.

detailed study is made of the changes taking place during the first few hours of digestion.

SUMMARY

The oxygen tension exerts a marked influence on the rate of autolysis of two malignant neoplasms, the skeletal muscle and the liver of the albino rat. Under greatly reduced oxygen tension the rate of

proteolysis is increased and the rate of oxidation of SH groups is lowered, as compared with digestion under atmospheric oxygen tension.

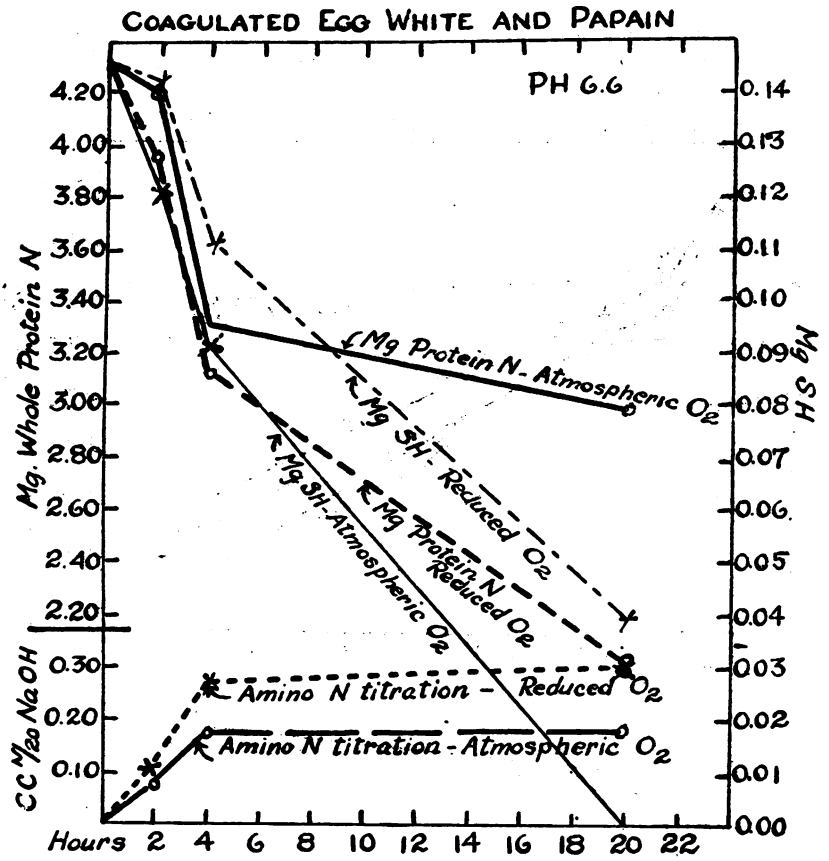

CHART 6.—Coagulated egg white and papain. One hundred and twenty c c of egg white was dissolved in 240 c c of 0.8 per cent NaCl. The solution was coagulated in a boiling water bath with mechanical stirring until a maximum nitroprusside test for sulphydryl was obtained (approximately 10 minutes). Each digestion mixture contained 1 c c of coagulated egg white, 4.8 mg of purified papain, 2 c c of McIlvaine buffer to maintain a pH of 6.6, and 0.2 c c toluol.

The same relationships are found in the digestion of coagulated egg white by papain and the digestion of blood fibrin by papain in the presence of reduced glutathione.

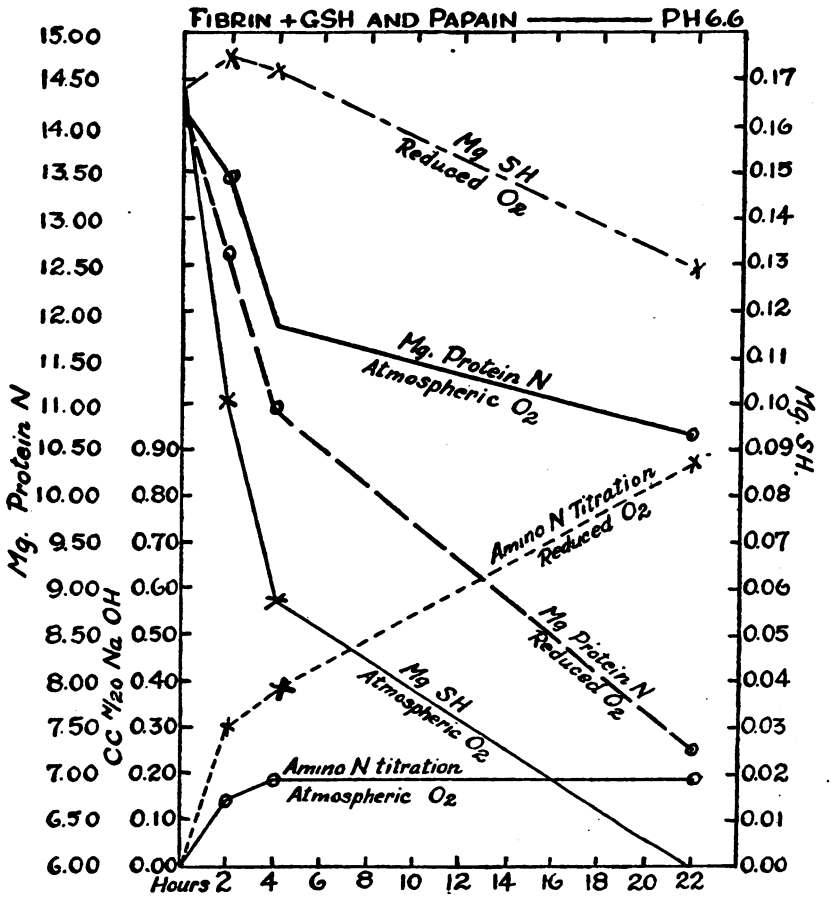

CHART 7.—The action of papain on fibrin in the presence of reduced glutathione. These digestion mixtures contained 1 c c of a 10 per cent ball mill emulsion of blood fibrin (Merck), 4.8 mg of papain and 1.5 mg of GSH, 2 c c of McIlvaine buffer at 7.3—which gave the mixture a pH of 6.6—and 0.2 c c toluol.

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COURT DECISION RELATING TO PUBLIC HEALTH

Statute requiring notice to be given regarding use of liquid, frozen, and dried eggs and other egg products imported from foreign countries held constitutional.—(California Supreme Court; Ex parte Bear, 15 P. (2d) 489; decided Oct. 21, 1932.) The title of chapter 280 of the 1931 California Statutes read as follows:

An act to provide for the inspection and certification of liquid eggs, frozen eggs, and dried eggs, and any other egg products produced in the State of California and within the United States and imported into the State of California from without the United States for the purpose of human consumption; to prescribe certain powers and duties of the State department of public health with respect thereto and to provide penalties for violations of the provisions of this act.

One provision in the law required restaurants, hotels, cafés, bakeries, and confectioneries using egg products imported from without the United States to display a sign to that effect, while another required manufacturers of food products using egg products so imported to label each package so as to show such use.

The petitioner, a food manufacturer, was charged with violating the statute and, in a habeas corpus proceeding, contended that the provisions set out above were unconstitutional. The reasons assigned against this part of the statute were: (1) It interfered with the power of Congress over interstate and foreign commerce; (2) it was an unreasonable and arbitrary exercise of police power; and (3) it was not embraced within the title of the act. The supreme court ruled adversely to the petitioner on all three points, taking the view that the portion of the statute assailed was constitutional and valid.

DEATHS DURING WEEK ENDED DECEMBER 24, 1932

[From the Weekly Health Index, issued by the Bureau of the Census, Department of Commerce]

	Week ended Dec. 24, 1932	Corresponding week 1931
Data from 85 large cities of the United States:		
Total deaths	9,614	7,383
Deaths per 1,000 population, annual basis	13.7	10.7
Deaths under 1 year of age	691	555
Deaths under 1 year of age per 1,000 estimated live births [1]	56	43
Deaths per 1,000 population, annual basis, first 51 weeks of year	11.2	11.7
Data from industrial insurance companies:		
Policies in force	69,276,593	74,282,027
Number of death claims	13,977	10,920
Death claims per 1,000 policies in force, annual rate	10.5	7.7
Death claims per 1,000 policies, first 51 weeks of year, annual rate	9.5	9.6

[1] 1932, 81 cities; 1931, 77 cities.

PREVALENCE OF DISEASE

No health department, State or local, can effectively prevent or control disease without knowledge of when, where, and under what conditions cases are occurring

UNITED STATES

CURRENT WEEKLY STATE REPORTS

These reports are preliminary, and the figures are subject to change when later returns are received by the State health officers

Reports for Weeks Ended December 31, 1932, and January 2, 1932

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended December 31, 1932, and January 2, 1932

Division and State	Diphtheria		Influenza		Measles		Meningococcus meningitis	
	Week ended Dec. 31, 1932	Week ended Jan. 2, 1932	Week ended Dec. 31, 1932	Week ended Jan. 2, 1932	Week ended Dec. 31, 1932	Week ended Jan. 2, 1932	Week ended Dec. 31, 1932	Week ended Jan. 2, 1932
New England States:								
Maine	----	6	72	2	----	375	0	0
New Hampshire	----	6	----	----	1	2	0	0
Vermont	1	----	----	----	1	162	0	0
Massachusetts	29	44	51	11	97	345	1	0
Rhode Island	1	5	48	7	1	666	0	0
Connecticut	6	5	96	2	27	61	0	0
Middle Atlantic States:								
New York	66	121	[1] 649	[1] 16	789	646	3	9
New Jersey	21	16	164	14	255	16	3	0
Pennsylvania	106	125	----	----	297	941	3	3
East North Central States:								
Ohio	72	159	1,178	40	449	153	1	3
Indiana	68	64	1,899	30	14	64	3	21
Illinois	68	122	363	19	43	36	21	3
Michigan	40	42	167	2	314	69	3	3
Wisconsin	8	15	1,906	15	215	79	1	2
West North Central States:								
Minnesota	3	19	55	3	52	48	1	3
Iowa	12	22	3,436	----	3	6	1	1
Missouri	36	55	257	3	23	10	4	1
North Dakota	2	6	4,618	----	26	24	1	0
South Dakota	3	6	199	----	3	35	0	0
Nebraska	11	6	365	2	6	5	2	0
Kansas	17	45	27,779	2	17	20	1	1
South Atlantic States:								
Delaware	4	8	9	1	2	1	0	0
Maryland [2] [3]	11	49	1,390	42	8	13	1	1
District of Columbia	10	6	74	----	4	2	1	1
Virginia	26	----	----	----	113	----	0	----
West Virginia	13	29	1,911	15	109	265	0	0
North Carolina [3]	29	73	804	34	85	67	1	3
South Carolina	5	24	2,179	387	35	21	0	0
Georgia [3]	8	11	1,467	58	3	----	7	0
Florida	14	9	70	3	----	1	0	0
East South Central States:								
Kentucky	21	53	3,064	----	----	----	0	6
Tennessee	19	52	4,098	49	14	10	3	4
Alabama [3]	24	45	4,424	52	----	6	0	1
Mississippi	7	23	----	----	----	----	0	1

See footnotes at end of table.

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended December 31, 1932, and January 2, 1932—Continued

Division and State	Diphtheria		Influenza		Measles		Meningococcus meningitis	
	Week ended Dec. 31, 1932	Week ended Jan. 2, 1932	Week ended Dec. 31, 1932	Week ended Jan. 2, 1932	Week ended Dec. 31, 1932	Week ended Jan. 2, 1932	Week ended Dec. 31, 1932	Week ended Jan. 2, 1932
West South Central States:								
Arkansas	12	19	10,054	6	----	1	4	0
Louisiana [3]	17	34	910	4	11	12	2	1
Oklahoma [4]	36	61	2,369	71	3	1	0	0
Texas [3]	70	94	2,794	14	450	----	0	1
Mountain States:								
Montana	1	1	7,073	----	256	98	0	0
Idaho	3	1	12	----	1	----	1	0
Wyoming	----	----	181	----	11	9	0	0
Colorado	5	4	109	----	7	1	0	1
New Mexico	24	38	1	----	2	1	1	1
Arizona	1	6	32	6	1	----	1	0
Utah [3]	2	----	44	4	1	----	0	0
Pacific States:								
Washington	3	5	154	----	2	187	1	1
Oregon	1	1	2,358	65	15	6	0	1
California	44	63	1,219	161	83	177	5	6
Total	980	1,598	90,102	1,140	3,849	4,642	77	79

Division and State	Poliomyelitis		Scarlet fever		Smallpox		Typhoid fever	
	Week ended Dec. 31, 1932	Week ended Jan. 2, 1932	Week ended Dec. 31, 1932	Week ended Jan. 2, 1932	Week ended Dec. 31, 1932	Week ended Jan. 2, 1932	Week ended Dec. 31, 1932	Week ended Jan. 2, 1932
New England States:								
Maine	0	2	21	35	0	0	2	3
New Hampshire	0	1	20	10	0	1	0	0
Vermont	0	0	11	12	0	10	0	0
Massachusetts	0	1	353	372	0	0	3	20
Rhode Island	0	0	36	50	0	0	0	0
Connecticut	0	0	110	65	6	2	0	2
Middle Atlantic States:								
New York	6	17	554	582	0	3	2	19
New Jersey	4	0	241	144	0	0	1	2
Pennsylvania	2	2	621	495	0	0	9	16
East North Central States:								
Ohio	1	2	615	595	8	22	6	20
Indiana	0	4	111	81	4	10	0	9
Illinois	2	1	374	287	0	38	9	13
Michigan	1	2	463	251	0	4	16	4
Wisconsin	0	1	65	65	5	8	0	3
West North Central States:								
Minnesota	2	1	83	46	0	9	0	1
Iowa	2	3	42	32	34	47	0	0
Missouri	0	0	74	56	0	19	1	0
North Dakota	0	0	6	18	1	12	0	3
South Dakota	0	1	15	14	0	12	3	2
Nebraska	0	1	36	39	1	5	1	1
Kansas	0	0	87	60	0	1	0	3
South Atlantic States:								
Delaware	0	0	6	17	0	0	1	0
Maryland [3] [5]	0	0	94	86	0	0	4	10
District of Columbia	0	0	9	23	0	0	0	1
Virginia	1	----	66	----	4	----	7	----
West Virginia	0	1	37	22	1	6	1	24
North Carolina [3]	0	0	60	73	1	0	3	4
South Carolina	1	0	12	14	1	2	3	12
Georgia [3]	1	0	12	26	0	1	5	7
Florida	1	0	8	12	0	0	1	1
East South Central States:								
Kentucky	1	0	49	81	1	0	2	3
Tennessee	2	1	72	43	5	12	2	13
Alabama [3]	0	0	27	44	0	1	0	17
Mississippi	0	1	17	17	0	22	3	4

See footnotes at end of table.

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended December 31, 1932, and January 2, 1932—Continued

Division and State	Poliomyelitis		Scarlet fever		Smallpox		Typhoid fever	
	Week ended Dec. 31, 1932	Week ended Jan. 2, 1932	Week ended Dec. 31, 1932	Week ended Jan. 2, 1932	Week ended Dec. 31, 1932	Week ended Jan. 2, 1932	Week ended Dec. 31, 1932	Week ended Jan. 2, 1932
West South Central States:								
Arkansas	0	0	9	17	0	26	1	2
Louisiana [3]	1	1	9	28	9	2	8	6
Oklahoma [4]	0	0	39	44	10	4	2	5
Texas [3]	0	0	69	49	15	22	0	12
Mountain States:								
Montana	0	3	12	21	0	2	3	1
Idaho	1	0	3	8	5	2	1	0
Wyoming	0	0	4	8	0	1	0	0
Colorado	0	0	42	21	0	5	2	1
New Mexico	0	0	19	29	0	1	4	3
Arizona	0	0	8	6	0	2	0	1
Utah [2]	0	0	9	5	0	0	0	0
Pacific States:								
Washington	1	0	21	56	6	10	0	3
Oregon	0	0	22	31	2	6	1	1
California	0	5	108	115	7	9	5	3
Total	30	51	4,781	4,205	126	339	112	255

[1] New York City only.
[2] Week ended Friday.
[3] Typhus fever, week ended Dec. 31, 1932, 15 cases: 1 case in Maryland, 2 cases in North Carolina, 4 cases in Georgia, 1 case in Alabama, 1 case in Louisiana, and 6 cases in Texas.
[4] Figures for the week ended Dec. 31, 1932, are exclusive of Oklahoma City and Tulsa, and for the week ended Jan. 2, 1932, are exclusive of Tulsa only.

SUMMARY OF MONTHLY REPORTS FROM STATES

The following summary of cases reported monthly by States is published weekly and covers only those States from which reports are received during the current week.

State	Meningococcus meningitis	Diphtheria	Influenza	Malaria	Measles	Pellagra	Poliomyelitis	Scarlet fever	Smallpox	Typhoid fever
October, 1932										
Hawaii Territory	1	22	141	----	----	----	----	----	----	6
Nevada	----	5	54	----	2	----	0	8	0	3
November, 1932										
California	13	439	5,806	2	217	4	17	811	9	43
Idaho	2	21	[1] 157	----	14	----	----	24	----	40
Kansas	10	128	25	----	33	----	2	382	7	14
Montana	----	3	65	----	723	----	0	51	----	13
Nevada	----	11	256	----	----	----	0	13	0	3
Oklahoma [2]	2	278	157	81	10	----	----	156	10	52
Oregon	----	12	291	----	163	1	3	100	3	8
Puerto Rico	----	36	352	4,620	148	2	0	----	0	15
Washington	2	38	73	----	9	----	15	139	19	16
Wisconsin	6	50	171	----	782	----	2	403	19	22

[1] Incomplete.
[2] Exclusive of Oklahoma City and Tulsa.

October, 1932

Chicken pox:	
Hawaii Territory	2
Nevada	2
Conjunctivitis:	
Hawaii Territory	34
Hookworm disease:	
Hawaii Territory	10
Leprosy:	
Hawaii Territory	4
Mumps:	
Hawaii Territory	1
Ophthalmia neonatorum:	
Hawaii Territory	1
Tetanus:	
Hawaii Territory	3
Trachoma:	
Hawaii Territory	27
Whooping cough:	
Hawaii Territory	8
Nevada	6

November, 1932

Chicken pox:	
California	1,532
Idaho	78
Kansas	613
Montana	245
Nevada	4
Oklahoma [1]	26
Oregon	229
Puerto Rico	22
Washington	650
Wisconsin	262
Conjunctivitis:	
Oklahoma [1]	1
Dysentery:	
California (amebic)	9
California (bacillary)	48
Oklahoma [1]	7
Puerto Rico	2,508
Washington	7
Filariasis:	
Puerto Rico	4
Food poisoning:	
California	16
German measles:	
California	33
Montana	2
Washington	9
Wisconsin	19
Granuloma, coccidioidal:	
California	2
Hookworm disease:	
California	1
Impetigo contagiosa:	
Kansas	20
Montana	35
Oklahoma [1]	1
Oregon	83
Puerto Rico	5
Washington	1
Jaundice, epidemic:	
California	3
Leprosy:	
California	3
Lethargic encephalitis:	
California	6
Oregon	1
Washington	4
Wisconsin	2
Mumps:	
California	537
Idaho	61
Kansas	254
Montana	16
Oklahoma [1]	9
Oregon	20
Puerto Rico	27
Washington	29
Wisconsin	364
Ophthalmia neonatorum:	
California	2
Puerto Rico	6
Wisconsin	1
Paratyphoid fever:	
California	1
Puerto Rico	3
Ptomaine poisoning:	
Kansas	2
Puerperal septicemia:	
Puerto Rico	1
Washington	2
Rabies in animals:	
California	52
Washington	7
Rabies in man:	
Kansas	1
Relapsing fever:	
California	1
Scabies:	
Kansas	10
Montana	6
Oklahoma [1]	1
Oregon	110
Septic sore throat:	
California	10
Idaho	2
Kansas	12
Montana	2
Oklahoma [1]	33
Oregon	1
Tetanus:	
California	8
Kansas	2
Montana	1
Oklahoma [1]	3
Puerto Rico	11
Tetanus, infantile:	
Puerto Rico	28
Trachoma:	
California	11
Puerto Rico	17
Washington	1
Wisconsin	5
Trichinosis:	
California	10
Tularaemia:	
Kansas	12
Montana	2
Oklahoma [1]	2
Wisconsin	2
Undulant fever:	
California	7
Kansas	1
Montana	1
Washington	2
Wisconsin	1
Vincent's angina:	
Kansas	10
Montana	6
Oklahoma [1]	4
Oregon	8
Whooping cough:	
California	1,104
Idaho	3
Kansas	157
Oklahoma [1]	28
Oregon	23
Puerto Rico	101
Washington	19
Wisconsin	426
Yaws:	
Puerto Rico	2

[1] Exclusive of Oklahoma City and Tulsa.

WEEKLY REPORTS FROM CITIES

City reports for week ended December 24, 1932

State and city	Diphtheria cases	Influenza cases	Influenza deaths	Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
Maine:											
Portland	0	------	0	0	0	0	0	1	0	3	13
New Hampshire:											
Concord	0	------	0	0	0	1	0	2	0	0	13
Nashua	0	------	0	0	0	0	0	0	0	0	------
Vermont:											
Barre	0	------	0	0	0	0	0	1	0	0	6
Burlington	1	------	0	0	0	0	0	0	0	0	7
Massachusetts:											
Boston	14	2	2	26	23	62	0	6	1	61	214
Fall River	1	2	0	0	1	6	0	3	0	7	28
Springfield	0	------	0	9	0	9	0	0	0	5	32
Worcester	4	------	0	0	7	22	0	2	1	2	52
Rhode Island:											
Pawtucket	0	------	0	0	1	0	0	0	0	0	28
Providence	2	1	0	0	3	7	0	4	0	23	62
Connecticut:											
Bridgeport	0	2	0	14	7	9	0	0	0	8	32
Hartford	7	------	0	2	2	3	0	0	0	5	24
New Haven	1	------	1	0	3	0	0	0	0	5	36

City reports for week ended December 24, 1932—Continued

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
New York:											
Buffalo	3	2	6	3	38	29	0	8	0	14	203
New York	43	177	29	269	220	180	0	70	4	105	1,623
Rochester	1	88	2	3	11	15	0	1	0	4	90
Syracuse	0	------	0	0	5	19	0	1	0	4	56
New Jersey:											
Camden	0	2	1	0	8	7	0	2	0	0	55
Newark	3	23	0	33	8	20	0	6	0	13	------
Trenton	1	2	0	0	6	10	0	1	0	0	41
Pennsylvania:											
Philadelphia	5	14	6	16	40	103	0	31	3	5	461
Pittsburgh	5	64	28	1	65	41	0	11	0	12	289
Reading	1	------	0	33	2	3	0	1	1	3	29
Scranton	3	------	------	0	------	7	0	------	0	0	------
Ohio:											
Cincinnati	2	24	10	0	32	17	0	7	0	1	153
Cleveland	4	366	15	0	36	91	0	13	0	3	233
Columbus	5	9	8	278	10	9	0	6	0	0	93
Toledo	0	6	3	21	11	23	0	1	0	0	72
Indiana:											
Fort Wayne	4	------	0	1	5	0	0	2	0	0	34
Indianapolis	4	------	7	4	17	7	0	7	1	5	------
South Bend	0	------	2	0	1	6	0	0	0	1	13
Terre Haute	0	------	0	2	0	1	0	0	0	0	17
Illinois:											
Chicago	6	63	33	32	78	194	0	43	0	12	796
Springfield	------	------	------	------	------	------	------	------	------	------	------
Michigan:											
Detroit	17	95	9	51	36	89	0	20	0	62	275
Flint	1	50	1	2	2	4	0	0	0	5	17
Grand Rapids	0	------	1	5	3	4	0	1	0	30	41
Wisconsin:											
Kenosha	0	35	1	0	1	1	0	0	0	2	8
Madison	2	1	------	8	------	2	0	------	0	0	------
Milwaukee	1	18	6	3	12	15	0	5	0	16	128
Racine	2	2	2	0	0	11	0	0	0	0	16
Superior	0	------	0	1	0	0	0	0	0	0	8
Minnesota:											
Duluth	0	------	1	0	3	3	0	2	0	2	31
Minneapolis	1	4	19	30	18	19	0	1	0	0	152
St. Paul	0	5	4	1	14	14	0	4	0	15	105
Iowa:											
Des Moines	9	------	------	0	------	7	0	------	0	0	52
Sioux City	2	------	------	0	------	1	0	------	0	2	1
Waterloo	1	------	------	0	------	0	0	------	0	0	------
Missouri:											
Kansas City	2	6	2	32	33	23	0	9	0	1	147
St. Joseph	3	------	0	0	13	3	0	1	2	1	37
St. Louis	16	16	6	2	22	20	0	12	0	0	235
North Dakota:											
Fargo	0	------	1	0	2	0	0	0	0	0	9
Grand Forks	0	------	0	12	0	0	0	0	0	0	0
South Dakota:											
Aberdeen	1	------	------	0	------	0	0	------	0	4	------
Nebraska:											
Omaha	7	------	0	0	18	13	1	1	0	0	83
Kansas:											
Topeka	------	------	------	------	------	------	------	------	------	------	------
Wichita	0	------	0	0	5	7	0	1	0	0	24
Delaware:											
Wilmington	0	------	0	0	7	2	0	0	0	0	32
Maryland:											
Baltimore	6	181	7	2	55	56	0	12	0	21	253
Cumberland	0	3	0	0	1	0	0	1	0	0	12
Frederick	1	------	0	0	0	0	0	0	0	0	1
District of Columbia:											
Washington	2	54	4	2	32	10	0	11	0	6	173
Virginia:											
Lynchburg	2	------	1	1	4	1	0	0	0	2	18
Norfolk	2	11	0	0	7	4	0	1	1	0	46
Richmond	0	------	2	0	10	9	0	2	0	0	65
Roanoke	2	------	0	2	2	2	0	2	0	0	19
West Virginia:											
Charleston	1	27	2	0	5	0	0	1	0	0	12
Huntington	0	178	------	5	------	2	1	------	0	0	------
Wheeling	0	------	1	75	9	1	0	2	0	4	32

City reports for week ended December 24, 1932—Continued

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
North Carolina:											
Raleigh	0	------	0	1	0	1	0	1	0	0	------
Wilmington	1	------	0	0	5	0	0	0	1	0	7
Winston-Salem	0	------	0	1	5	0	0	0	0	0	17
South Carolina:											
Charleston	0	94	0	0	2	0	0	2	0	0	22
Columbia	1	------	0	0	0	1	0	0	0	0	------
Greenville	0	------	0	1	0	0	0	0	0	0	------
Georgia:											
Atlanta	3	702	22	0	13	4	0	5	0	5	105
Brunswick	1	------	0	0	0	1	0	0	0	0	3
Savannah	1	57	3	0	6	1	0	2	0	1	36
Florida:											
Miami	1	------	0	0	3	0	0	0	0	0	23
Tampa	------	------	------	------	------	------	------	------	------	------	------
Kentucky:											
Covington	------	------	------	------	------	------	------	------	------	------	------
Lexington	0	71	3	0	7	1	0	5	0	0	35
Louisville	2	23	2	0	18	9	0	2	0	0	79
Tennessee:											
Memphis	1	------	14	0	18	5	0	4	0	0	105
Nashville	0	------	10	0	11	2	0	6	0	0	59
Alabama:											
Birmingham	3	194	15	0	8	1	0	2	0	1	84
Mobile	1	3	1	0	4	3	0	0	0	0	22
Montgomery	0	66	------	0	------	2	0	------	0	0	------
Arkansas:											
Fort Smith	------	------	------	------	------	------	------	------	------	------	------
Little Rock	0	70	1	0	13	0	0	2	0	0	16
Louisiana:											
New Orleans	13	------	0	0	18	4	0	13	1	0	192
Shreveport	0	------	3	0	15	1	0	3	0	0	51
Oklahoma:											
Tulsa	0	------	------	0	------	2	0	------	0	2	------
Texas:											
Dallas	12	179	25	0	19	13	0	3	0	0	108
Fort Worth	2	------	7	2	13	7	0	3	0	0	------
Galveston	0	------	0	0	10	0	0	0	0	0	34
Houston	8	------	5	1	10	10	0	5	0	0	68
San Antonio	5	3	15	0	13	2	0	8	0	0	81
Montana:											
Billings	0	------	0	0	0	0	0	0	0	0	7
Great Falls	0	------	0	113	1	0	0	0	0	0	10
Helena	0	252	1	0	0	0	0	0	0	0	8
Missoula	0	171	1	0	0	0	0	0	0	0	8
Idaho:											
Boise	0	------	0	6	2	0	5	0	0	0	5
Colorado:											
Denver	4	186	16	5	27	14	0	5	0	1	108
Pueblo	0	------	1	0	2	1	0	0	0	1	13
New Mexico:											
Albuquerque	0	------	0	0	3	0	0	4	0	4	12
Arizona:											
Phoenix	0	------	0	0	8	0	0	4	0	0	------
Utah:											
Salt Lake City	1	------	5	1	1	1	0	1	0	0	51
Nevada:											
Reno	0	------	0	0	0	0	0	0	0	0	4
Washington:											
Seattle	1	------	------	0	------	3	0	------	1	10	------
Spokane	0	------	------	0	------	3	1	------	1	0	------
Tacoma	0	------	0	0	2	3	0	0	0	0	34
Oregon:											
Portland	0	165	3	0	7	5	0	1	0	0	80
Salem	0	119	------	5	------	1	0	------	0	0	------
California:											
Los Angeles	18	198	7	26	27	33	2	30	1	21	362
Sacramento	1	3	0	0	6	0	0	2	0	1	26
San Francisco	0	119	13	2	16	6	0	13	1	14	205

City reports for week ended December 24, 1932—Continued

State and city	Meningococcus meningitis		Poliomyelitis cases	State and city	Meningococcus meningitis		Poliomyelitis cases
	Cases	Deaths			Cases	Deaths	
Massachusetts:				Missouri:			
Boston	1	2	0	Kansas City	1	0	0
Rhode Island:				St. Joseph	2	0	0
Providence	1	0	0	St. Louis	2	0	0
New York:				District of Columbia:			
New York	5	2	0	Washington	0	1	0
New Jersey:				Georgia:			
Newark	1	1	0	Atlanta	1	0	0
Pennsylvania:				Kentucky:			
Philadelphia	0	1	0	Louisville	0	0	1
Pittsburgh	1	1	0				
Ohio:				Louisiana:			
Cleveland	1	0	0	New Orleans	1	1	0
Toledo	0	1	0				
Indiana:				Washington:			
Indianapolis	4	1	0	Seattle	1	0	1
Illinois:				California:			
Chicago	10	2	0	Los Angeles	2	0	0
Michigan:							
Detroit	2	0	0				

Lethargic encephalitis.—Cases: Toledo, 1; Chicago, 1; Birmingham, 1.
Dengue.—Cases: Charleston, S. C., 2.
Pellagra.—Cases: Savannah, 1; New Orleans, 1; Los Angeles, 1.
Typhus fever.—Cases: Savannah, 1; Mobile, 1.

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FOREIGN AND INSULAR

CANADA

Provinces—Communicable diseases—Week ended December 17, 1932.—The Department of Pensions and National Health of Canada reports cases of certain communicable diseases for the week ended December 17, 1932, as follows:

Disease	Nova Scotia	New Brunswick	Quebec	Ontario	Manitoba	Saskatchewan	Alberta	British Columbia	Total
Cerebrospinal meningitis			1						1
Chicken pox	2		115	334	68	4	2	48	573
Diphtheria	1	5	25	15	8	4	1		59
Dysentery								3	3
Erysipelas			4	2	4				10
Influenza	9		2	200	1			731	943
Lethargic encephalitis				1	1				2
Measles	3	12	59	431	1		4	23	533
Mumps				59	11	7		1	78
Pneumonia				25		2		13	40
Poliomyelitis		1	1						2
Scarlet fever	10	4	61	85	32	8	3	20	223
Smallpox				10					10
Trachoma					4			5	9
Tuberculosis	1	15	38	59	23	14	2	22	174
Typhoid fever		6	8	1	1	2		1	19
Whooping cough			114	76	37	8		28	263

Ontario Province—Communicable diseases—Four weeks ended November 26, 1932.—The Department of Health of the Province of Ontario reports certain communicable diseases for the four weeks ended November 26, 1932, as follows:

Disease	Cases	Deaths	Disease	Cases	Deaths
Cerebrospinal meningitis	1		Pneumonia		74
Chicken pox	1,140		Poliomyelitis	21	
Diphtheria	126	4	Puerperal septicemia	1	1
Dysentery	1		Scarlet fever	240	1
Erysipelas	8		Septic sore throat	3	
German measles	23		Syphilis	237	
Gonorrhea	359		Tuberculosis	110	28
Influenza	50	1	Trench mouth	1	
Lethargic encephalitis	1		Typhoid fever	42	
Measles	997	1	Undulant fever	10	
Mumps	308		Whooping cough	324	1
Paratyphoid fever	3				

LATVIA

Communicable diseases—August–October, 1932.—During the months of August, September, and October, 1932, cases of certain communicable diseases were reported in Latvia as follows:

Disease	Cases			Disease	Cases		
	August	September	October		August	September	October
Botulism	1			Mumps	63	14	36
Cerebrospinal meningitis	8	4	2	Paratyphoid fever	27	15	14
Diphtheria	40	85	143	Poliomyelitis	1	1	6
Dysentery	16	12	1	Puerperal fever	6	8	9
Erysipelas	16	20	20	Scarlet fever	20	37	52
Influenza	51	57	68	Tetanus	2		3
Leprosy			2	Trachoma	38	62	35
Lethargic encephalitis	1			Typhoid fever	75	101	86
Measles	16	7	270	Whooping cough	103	74	65

PUERTO RICO

Communicable diseases—Four weeks ended December 3, 1932.—During the four weeks ended December 3, 1932, cases of certain communicable diseases were reported in Puerto Rico as follows:

Disease	Cases	Disease	Cases
		Ophthalmia neonatorum	6
Bronchitis	97	Paratyphoid fever	4
Broncho-pneumonia	5	Pellagra	2
Chicken pox	22	Pneumonia	7
Diphtheria	36	Puerperal fever	1
Dysentery	2,612	Syphilis	247
Filariasis	3	Tetanus	7
Framboesia, tropical	2	Tetanus, infantile	7
Impetigo contagiosa	5	Trachoma	10
Influenza	388	Tuberculosis	349
Malaria	4,187	Typhoid fever	12
Measles	135	Whooping cough	98
Mumps	28		

CHOLERA, PLAGUE, SMALLPOX, TYPHUS FEVER, AND YELLOW FEVER

(NOTE.—A table giving current information of the world prevalence of the quarantinable diseases appeared in the Public Health Reports for December 30, 1932, pp. 2382–2394. A similar cumulative table will appear in the Public Health Reports to be issued January 27, 1933, and thereafter, at least for the time being, in the issue published on the last Friday of each month.)

Cholera

Philippine Islands.—During the week ended December 31, 1932, 14 cases of cholera with 5 deaths were reported in Leyte Province, Philippine Islands, and 68 cases with 54 deaths in Samar Province.

Plague

Argentina.—On December 16, 1932, two fatal cases of plague were reported in the Province of Cordoba. It was stated that a total of 27 cases of plague had been reported in the Province of Salta.

Egypt—Alexandria.—A fatal case of plague was reported at Alexandria, Egypt, during the week ended December 17, 1932.

Smallpox

Ceylon—Colombo.—From November 30 to December 29, 1932, 47 cases of smallpox were reported at Colombo, Ceylon.

China—Canton.—During the week ended December 24, 1932, 213 cases of smallpox with 8 deaths were reported at Canton, China.

Egypt—Alexandria.—During the two weeks ended December 24, 1932, 110 cases of smallpox with 36 deaths were reported at Alexandria Egypt.

Yellow Fever

Brazil—Ceara State.—On November 2, 1932, a case of yellow fever was reported at Lavras, State of Ceara, Brazil.

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