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TYPHUS FEVER

THE EXPERIMENTAL TRANSMISSION OF ENDEMIC TYPHUS FEVER OF THE UNITED STATES BY THE RAT FLEA *Ceratophyllus fasciatus*

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In our first studies on the part played by rat fleas in the transmission of endemic typhus from rat to rat or from rat to man, two species of rat fleas were incriminated—*Xenopsylla cheopis* and *Ceratophyllus fasciatus*. The infectibility of *X. cheopis* with endemic typhus has been shown and the probable mechanism by which the infection is transmitted to man has been elucidated in a large measure in our more recent studies. That *C. fasciatus* is infectible with Mexican typhus virus was shown by Mooser and Castaneda.

To determine the ability of *C. fasciatus* to transmit endemic typhus the following experiment was performed:

A few fleas (*C. fasciatus*) were procured from rats trapped in Savannah, Ga. These fleas were placed in glass box C 10, furnished with a fresh white rat as a source of food supply and allowed to breed until many fleas were present in the box. Twelve fleas were then removed from this colony, emulsified in salt solution, and injected into two guinea pigs intraperitoneally. Neither of these animals developed any signs of typhus fever, nor were they found immune upon subsequent inoculation with typhus virus.

Being assured that our colony of this species of flea was noninfected, we then placed in box C 10 three white rats that had been freshly inoculated with endemic typhus virus. Fourteen days after the first, and six days after the last infected rat had been placed in the box, five fleas were removed from these rats, emulsified in salt solution, and injected intraperitoneally into two guinea pigs. One of these guinea pigs developed clinical endemic typhus after an incubation period of 10 days.

From this guinea pig a virus was recovered and studied in other animals. The identification of this strain of virus as endemic typhus virus was established by the six criteria on which we have come to rely for the identification of our experimental strains. These criteria are as follows:

1. Typical febrile reactions and typical scrotal involvement in guinea pigs.

2. Negative blood cultures from guinea pigs at the height of their reaction.
3. Intracellular rickettsia in smears made from the tunica vaginalis of guinea pigs reacting typically.
4. The development in rabbits of agglutinins for *B. proteus* X₁₉, type O.
5. Typical histologic lesions in the brains of guinea pigs.
6. Clear-cut cross-immunity between the unknown strain and known strains of typhus.

The recovery of typhus virus from fleas taken from box C 10 was twice repeated. In the first repetition, eight fleas were inoculated into guinea pigs, and in the second repetition, 10 fleas were used. In both instances a virus was recovered which produced the typical clinical picture in guinea pigs. One of these strains was not studied further, while the second was carried only until rickettsia had been found in smears of the tunica of reacting guinea pigs and a positive Weil-Felix had developed in one of the two rabbits inoculated.

A number of fleas were then removed from the typhus-infected colony in box C 10 and placed in box C 11. Three fresh white rats were then placed in box C 11 and allowed to remain 12, 13, and 14 days, respectively. On the days indicated, the rats were killed and their spleens were emulsified in salt solution and injected intraperitoneally into guinea pigs. From the white rat killed on the thirteenth day after his first exposure to infected fleas, a strain of virus was recovered and studied in other animals. This strain of virus was identified as the virus of endemic typhus by the criteria noted above.

(We are indebted to Passed Assistant Surgeon R. D. Lillie for histologic examination of brain specimens.)

CONCLUSION

Experimental transmission of the virus of endemic typhus from rat to rat by means of the rat flea *Ceratophyllus fasciatus* has been carried out in the laboratory.

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THE FORMATION OF ARSENOXIDE FROM THE ARSPHENAMINES IN THE LIVING ANIMAL AND IN TEST-TUBE OXIDATIONS

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Since the demonstration by Hata (1) and by numerous later investigators (2, 3) that arsphenamine and neoarsphenamine are, in the test tube, relatively nontoxic toward spirochetes and trypanosomes, it has been necessary to modify Ehrlich's view of a direct action of these drugs on the parasite. Several explanations of their action have been advanced, either postulating an action through stimulation of antibody formation or through a change of the drug within the host or the parasite to a compound of greater parasitocidal action.

Ehrlich and Bertheim (4) prepared arsphenamine by reduction of 3-amino-4-hydroxyphenyl arsenious oxide and obtained evidence that this "arsenoxide" is formed when arsphenamine is allowed to undergo oxidation in the air.

Arsenoxide was later found to be several hundred times as trypanocidal *in vitro* as the arsphenamines, and in 1920 Voegtlin and Smith (5) advanced the theory that the parasitocidal action of the arsphenamines is due to their conversion in the body of the host into a compound of the arsenoxide type.

In support of this theory, Voegtlin and Smith showed that in rats infected with *Tr. equiperdum* the trypanocidal action of intravenously injected arsphenamine and related arsenobenzene compounds is always preceded by a latent period of two to three hours, while with compounds of the arsenoxide type the trypanosomes begin to disappear from the blood immediately. It was further found that solutions of arsphenamine and neoarsphenamine that had undergone partial oxidation in air manifested a corresponding reduction of their latent period.

Since arsenoxide is also approximately ten times more toxic to the host than arsphenamine, and approximately twenty times more toxic than neoarsphenamine, the conversion of the arsphenamines in the body into arsenoxide would be of importance in the toxic effects produced by them upon higher animals. The evidence in favor of such a view has been summarized by Voegtlin (6).

Final proof of the arsenoxide theory has awaited more complete information concerning the formation of arsenoxide from arsphenamine *in vitro*, and the demonstration of arsenoxide in the tissues of animals following the injection of the arsphenamines.

We have developed a color test which will differentiate between the arsphenamines and arsenoxide. With this procedure, confirmatory evidence has been obtained that arsenoxide is formed from the arsphenamines, both in the living animal and also in test-tube oxidations.

THE TEST

It was observed that under certain conditions β -naphthoquinone sodium sulphonate would give a strong color reaction with arsenoxide and very little color with the arsphenamines. After a large number of experiments it was possible to develop these conditions so that a fair degree of specificity of the reaction for arsenoxide was obtained.

β -naphthoquinone has been employed by several investigators because of its ability to react with other compounds to form highly colored complexes. Ehrlich and Herter (7) and Herter (8) studied the color reactions between this dye and a large number of substances. Ehrlich and Bertheim (4) state that this dye reacts with arsenoxide, forming a dark red condensation product, soluble in alkali.

Folin (9) employed this naphthoquinone for the estimation of amino nitrogen in the blood, and Sullivan (10) has developed a highly specific color test for cysteine. It was while we were studying the reaction between cysteine and arsenoxide that the capacity of the latter substance for giving a color reaction with naphthoquinone was observed.

The procedure which we have finally employed is as follows:

Five c. c. of the aqueous solution containing arsenoxide is made neutral to litmus. Two c. c. of a 10 per cent aqueous solution of sodium cyanide is then added and the solution is mixed. There are then added 2 c. c. of a 0.25 per cent solution of β -naphthoquinone sodium sulphonate made up in a 10 per cent aqueous solution of sodium thiosulphate ($\text{Na}_2\text{S}_2\text{O}_3$). The addition of 0.2 c. c. of 50 per cent cadmium sulphate will approximately double the intensity of the color obtained, and we have usually added it at this point when testing for the low concentrations of arsenoxide found in tissues. It is best to make up the solution of naphthoquinone in thiosulphate 10 minutes before using and to employ especially clean glassware. If naphthoquinone thiosulphate, water, and glassware are satisfactory, the yellow color of the quinone will have almost completely disappeared after standing 10 minutes in thiosulphate solution. A thin layer of mineral oil is now run over the top of the solution being tested, and the test tubes are set aside at room temperature for 30 minutes, after which time 1 c. c. of a 20 per cent aqueous solution of sodium sulphite (Na_2SO_3) is added. The tube is gently shaken and the color is compared with a series of tubes containing known amounts of arsenoxide, upon which simultaneous tests have been run. From 0.15 mg. to 3 mg. of arsenoxide in 5 c. c. of solution can be estimated by this method. Direct comparison in a comparator block has been found more satisfactory than the use of the colorimeter where slight differences in shade of color were present. Accuracy to within 5 per cent can usually be obtained in this way.

THE SPECIFICITY OF THE TEST

While a complete specificity for arsenoxide is not claimed for this procedure, we have tested out a large number of arsenicals in dilute solution, and numerous protein-free extracts of various tissues, with negative results. The only compound which we know that gives a comparable color, and which might be present in the solutions or extracts tested, is ortho-aminophenol. By a modification of the test to be described in the following paragraphs, we have been able to distinguish between these two substances.

In Table 1 is listed the arsenic compounds which we have tested. Inorganic tri- or pentavalent arsenic gives no color, nor does it interfere with the color given by arsenoxide. The pentavalent compound corresponding to arsenoxide, 3-amino-4-hydroxyphenyl arsonic acid, gives no reddish color unless present in relatively high concentrations (0.1 per cent), considerably above any that we have dealt with, either in the tissue extracts or in the test-tube experiments. When either the amino or the hydroxy group is absent from arsenoxide, the reaction is negative. Freshly prepared solutions of arspenamine, neoarsphenamine, and sulpharsphenamine are negative with this reaction. Doctor Shonle, of Eli Lilly Laboratories, has kindly furnished us with several trivalent arsenicals of the arsenoxide type, as well as pentavalent aromatic arsenicals, containing either amino or hydroxy groups in the benzene ring. All of these compounds were negative.

TABLE 1.—Arsenical compounds tested with the naphthoquinone reaction

Compound	Parts per million	Color produced
3-amino-4-hydroxyphenyl arsenious oxide (arsenoxide) ¹	400	Red-brown.
Arspenamine.....	400	Yellow.
Neoarsphenamine.....	400	Do.
Sulpharsphenamine.....	400	Do.
4-dimethyl aminophenyl arsenious oxide ¹	400	Do.
4-hydroxyphenyl arsenious oxide ¹	400	Do.
p-aminophenyl arsenious oxide ²	300	Do.
p-acetylaminophenyl arsenious oxide ²	300	Do.
p-carbamino phenyl arsenious oxide ²	300	Do.
Arsenious oxide, As ₂ O ₃	1,000	Do.
Tryparsamide.....	400	Do.
Atoxyl.....	400	Do.
3-amino-4-hydroxyphenyl arsonic acid ¹	400	Do.
Phenylglycine arsonic acid ¹	400	Do.
3-nitro-4-hydroxyphenyl arsonic acid.....	400	Do.
Sodium p-acetyl aminophenyl arsiniate.....	400	Do.
p-propanol aminophenyl arsonic acid ²	400	Do.

¹ These compounds were prepared by Dr. J. M. Johnson of the National Institute of Health.

² Received from Dr. H. A. Shonle of Eli Lilly & Co. Laboratories.

Of the compounds of biological interest which we have studied, glutathione gives a negative reaction with the test, while cysteine in dilute solution gives a faint violet color which is practically eliminated when cadmium sulphate is employed in the test. All of the amino acids which we studied were negative, as well as other substances

which would normally be present in protein-free blood and tissue extracts. Tissue extracts, as well as protein-free urine, were uniformly negative (Table 2).

Of the aromatic compounds which do not contain arsenic, phenol does not give the color reaction; pyridine, aniline, and meta-aminophenol give a yellow color; while para-aminophenol gives a deep violet color quite distinct from that of arsenoxide.

TABLE 2.—*Miscellaneous substances tested with the naphthoquinone reaction. (Commercial samples of high purity)*

Substance	Parts per million	Color produced	Substance	Parts per million	Color produced
Cysteine.....	400	Violet. ¹	Urea.....	2,000	Yellow.
Glutathione.....	400	Yellow.	Creatinine.....	800	Do.
Phenyl alanine.....	800	Do.	Aniline.....	(?)	Do.
Glutamic acid.....	1,000	Do.	Pyridine.....	(?)	Do.
Glycol.....	400	Do.	Acetanilid.....	400	Do.
Histidine.....	500	Do.	o-aminophenol.....	400	Red-brown.
Tyrosine.....	1,000	Do.	p-aminophenol.....	400	Violet.
Tryptophane.....	400	Do.	m-aminophenol.....	400	Yellow.
Uric acid.....	800	Do.	Urine.....	Undiluted.	Do.

¹ Very faint if cadmium sulphate is employed in the test.

² 1 drop in 5 c. c.

³ 2 drops in 5 c. c.

Ortho-aminophenol gives a color similar to that of arsenoxide and of approximately the same intensity. Since this compound can be formed from arsenoxide by boiling with strong acids (11), it is necessary for the purpose of our experiments to distinguish between them. We have not been able to get rid of the color of the o-aminophenol and retain that of arsenoxide in the test but we have succeeded, by the use of stannous chloride, in modifying the procedure so that practically no red color is given by arsenoxide in dilute solution, while a strong red color is given by o-aminophenol. The procedure is as follows:

To 2 c. c. of 10 per cent sodium thiosulphate is added 10 drops of a 1 per cent stannous chloride solution in 1 per cent hydrochloric acid. Allow this mixture to stand ten minutes, when it becomes of a milky opacity. Then add this mixture to 5 c. c. of the neutral solution to be tested, mix, and add 2 c. c. of 10 per cent sodium cyanide and 4 drops of 10 per cent sodium hydroxide; mix, and allow to stand 10 minutes. Then add 1 c. c. of a freshly prepared 1 per cent aqueous solution of the naphthoquinone; mix, and in exactly 15 seconds add approximately 0.2 gm. of sodium sulphite as the powder, or in 20 per cent solution. Let the solution stand for 30 to 45 minutes, when it will have sufficiently cleared for color comparison. With this method 2 mg. of arsenoxide, or of the arsphenamines, give only a yellowish color, while similar amounts of o-aminophenol give a good red color. Amounts as small as 0.1 mg. of o-aminophenol in 5 c. c. can be detected. In the experiments described below we have applied this test for amino-

phenol to oxidized solutions of the arsphenamines which we have studied and to tissue extracts giving a positive reaction with the original test as described above. It has been possible to demonstrate that none of the color obtained in our experiments is due to *o*-aminophenol, but is due to arsenoxide.

THE FORMATION OF ARSENOXIDE FROM THE OXIDATION OF THE ARSPHENAMINES IN VITRO

It was shown by Ehrlich and Bertheim (4) that arsphenamine was readily oxidized in the air, and by oxidizing arsphenamine with hydrogen peroxide they obtained crystalline 3-amino-4-hydroxyphenyl arsonic acid, so that arsenoxide must have been an intermediate stage. They gave further evidence of the presence of arsenoxide in arsphenamine samples by precipitation of arsphenamine in methyl alcohol with calcium carbonate, and by determination of the iodine titer of the filtrate; this method would not be specific for arsenoxide, and arsenoxide has not been actually isolated from arsphenamine by Ehrlich or by subsequent workers who have studied this problem. Ehrlich, with the above method (4,12), showed that the amount of arsenoxide increased in samples of arsphenamine exposed to air, and he attributed much of the toxicity of arsphenamine to this substance. The work of Voegtlin and Smith (5) demonstrated that solutions of the arsphenamines which were permitted to undergo oxidation *in vitro* manifested a much shorter latent period of action upon trypanosomes in infected rats. This gave biological evidence that arsenoxide was formed. They later studied the oxidation of arsphenamine and arsenoxide *in vitro* in the presence of varying amounts of alkali and by a modification of Ehrlich's iodine titration method showed that more arsenoxide was formed from arsphenamine when the oxidation proceeded slowly (in the presence of smaller amounts of alkali). The rate of oxidation of neoarsphenamine in water was also shown by them to be much more rapid than that of arsphenamine.

Since the work of Ehrlich (4) several investigators have studied the increase of toxicity of the arsphenamines when the solutions are exposed to air or aerated, especially Roth (13), Hunt (14), and Schamberg, Kolmer, and Raiziss (15). Smith (16) has shown that arsenoxide is many times more active than arsphenamine in its circulatory effects.

Fresh solutions of arsphenamine of good quality react negatively with the naphthoquinone test which we have described. The presence of considerable amounts of arsphenamine also inhibits the color reaction obtained with arsenoxide. It was therefore necessary to prove that the negative reaction obtained with arsphenamine is not due to the interfering action of sodium hydrosulphite ($\text{Na}_2\text{S}_2\text{O}_4$) or related

reducing substances used in the manufacture of arsphenamine. This can be simply shown by precipitation of arsphenamine from a solution, by a procedure which will not remove hydrosulphite; in this way it can be demonstrated that no substances interfering with the color reaction are present in the filtrate. Cadmium sulphate was found to be an effective precipitating agent for arsphenamine, while at the same time it will not precipitate hydrosulphite or related sulphites. By this procedure it was shown that a small amount of arsenoxide added to arsphenamine can be quantitatively recovered in filtrates. The following experiment will illustrate these findings:

Fifty mg. of arsphenamine hydrochloride are dissolved in 9 c. c. of water, 0.5 c. c. of 50 per cent cadmium sulphate is added, the solution is mixed, and 0.45 c. c. of normal sodium hydroxide is added drop by drop with shaking. The filtrate of this solution is negative with the naphthoquinone test. When 2 mg. of arsenoxide are added to arsphenamine it can be quantitatively recovered in the filtrate, revealing that no interfering substances are present. When a small amount of sodium hydrosulphite is put through this procedure the filtrate will actively inhibit the color reaction obtained with arsenoxide.

In the estimation of arsenoxide in the presence of arsphenamine, it is necessary to precipitate and remove the arsphenamine from the solution prior to applying the test, or else values too low will be obtained. We have employed cadmium precipitation in studying the presence of arsenoxide in arsphenamine solutions that are undergoing oxidation.

The formation of arsenoxide from the arsphenamines takes place with great ease in alkaline solution. With arsphenamine itself, experiments done at hydrogen ion concentrations near neutrality are complicated by its almost complete insolubility. However, if care is taken to employ a finely divided suspension, arsenoxide can be formed at pH 7.3 when a fine stream of oxygen is bubbled through a solution of arsphenamine at 38° C.

Experiments with arsphenamine were carried out as follows: Forty-eight mg. are dissolved in 2 c.c. of water, alkali added to make the disodium salt, and then phosphate buffer (Clark and Lubs) is slowly added with shaking to make a volume of 20 c.c. (m/100). Determinations of pH were now made with the glass electrode and alkali or acid added to bring the solution to the desired hydrogen ion concentration.

In Chart 1 is shown the formation of arsenoxide from the arsphenamines, as well as the oxidation of arsenoxide at pH 7.3. With arsphenamine and neoarsphenamine, cadmium precipitation (no alkali employed) was carried out and determinations were done upon the filtrates. The standards consisted of five tubes containing 0.1 to 0.5 c.c. of m/100 arsenoxide plus 0.25 c.c. of cadmium sulphate in

5 c.c. of aqueous solution. The final dilution of the solution was 1 to 10. Each sample was tested for o-aminophenol, and in no instance was a positive test obtained.

The concentration of arsenoxide reached is dependent upon its rate of formation and also upon its rate of oxidation. With arsphenamine at pH 7.3 the rate of formation is very slow, owing to solubility factors. This is evident from the curve obtained at pH 9.5, where high concentrations are rapidly reached (Chart 1).

The instability of neoarsphenamine is shown in that the rate of oxidation proceeds more rapidly than with the other compounds. Thirty-five per cent of arsenoxide is present after one-half hour of oxygenation, and 40 per cent after one hour. The high concentrations of arsenoxide reached are of considerable interest. Since a free

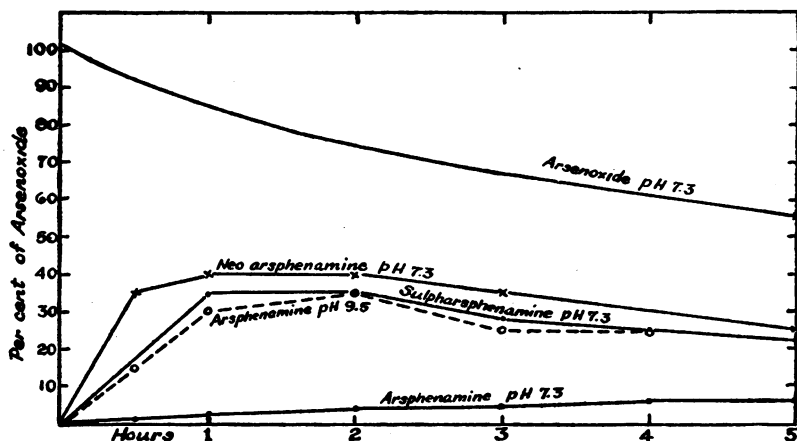


CHART 1.—The formation of arsenoxide from the arsphenamines, and the rate of oxidation of arsenoxide, when oxygen is bubbled through 0.01 molar solutions in phosphate buffer at 38° C.

amino group is essential for the color reaction with naphthoquinone, these findings suggest that the methylene sulphinate radical is split off from its amino linkage during the oxidation of neoarsphenamine. This evidence is borne out by the behavior of sulpharsphenamine. We have dealt with three products of this drug which were analyzed by Elvove's procedure (17) and were shown to be 53 per cent, 85 per cent, and 97 per cent disubstitution products. The rate of formation of arsenoxide was similar in all three products. With the 97 per cent disubstitution product the splitting off of the methylene sulphinate radical is essential to the formation of more than 3 per cent of free arsenoxide.¹ The oxidation experiments show that while the initial

¹ For absolute proof that the sulphinate radical is split off it must be shown that sulphonated arsenoxide reacts negatively with the color test. So far we have been unable to obtain such a compound.

rate is slower with sulpharsphenamine than with neoarsphenamine, the concentrations of arsenoxide reached within one to two hours are almost as high (Chart 1). In carrying out the tests upon sulpharsphenamine it was found that, with the product employed, concentrations of 2.0 mg. in 5 c.c. did not require cadmium precipitation for recovery of arsenoxide and in some of the experiments tests were carried out directly upon 0.5 c.c. of the 0.01 molar solution diluted to 5 c.c. with water. Unless the product has been shown not to inhibit the color reaction under these conditions, precipitation should be carried out.

COMPARISON OF THE COLOR TEST WITH TOXICITY TESTS UPON RATS

Since the toxicity of arsenoxide to rats is approximately ten times greater than that of arsphenamine, and approximately twenty times that of neoarsphenamine and sulpharsphenamine, a series of experiments was carried out to determine whether the quantity of arsenoxide, as shown by the naphthoquinone test, in oxidized solutions of the arsphenamines could be confirmed by demonstrating a corresponding increase in toxicity to rats. The minimum lethal dose of arsenoxide for rats under standard conditions is very sharply defined and has been previously established (18) by numerous experiments to be 26 mg. per kilo body weight.

With oxidized arsphenamine solutions there was very satisfactory correlation between the arsenoxide content and the toxic effects upon rats. A 0.5 per cent solution was made up in phosphate buffer as described above so that the final pH was approximately 10.5. After oxygen had been bubbled through this solution for 1 hour at 38° C., the arsenoxide content was estimated to be 40 per cent of the molar concentration of arsphenamine, or 2.16 mg. per c.c. With approximately half of the arsphenamine remaining in the solution, the minimum lethal dose should therefore be 54 mg. per kilo. Actually, at 45 mg. per kilo there was 25 per cent mortality, and at 60 mg. per kilo 100 per cent mortality (Table 3). The arsphenamine was brought completely into solution by adding a few drops of alkali immediately prior to the injections. The acute reactions, such as convulsions, lashing of the tail, etc., typical of arsenoxide, were present, and the average time of death with the 60 mg. dose was 92 minutes. With the smaller doses those that survived the first few hours recovered completely; none of the "late deaths" typical of some of the other arsenic compounds were observed.

TABLE 3.—*The toxicity to rats of a 0.5 per cent solution of arsphenamine in phosphate buffer (pH approximately 10.5) through which oxygen was bubbled for one hour at 38°C. The arsenoxide content by the color test was 2.16 mg. per c. c. (40 per cent of the arsphenamine), and the calculated M. L. D. was 54 mg. per kilo*

Rat weight	Dosage (mg. per kilo)	Time of death
110 grams.....	30	Survived.
92 grams.....	30	Do.
84 grams.....	30	Do.
100 grams.....	30	Do.
118 grams.....	45	155 minutes.
94 grams.....	45	Survived.
94 grams.....	45	Do.
96 grams.....	45	Do.
100 grams.....	60	120 minutes.
100 grams.....	60	160 minutes.
108 grams.....	60	120 minutes.
108 grams.....	60	30 minutes.

With neoarsphenamine in two series of experiments the acute toxicity was slightly in excess of that indicated by the amount of arsenoxide present, although the picture was complicated by the presence of late symptoms and late deaths. In one experiment at the end of an hour of oxidation in phosphate buffer of pH 7.3 at 38°C. the solution (0.5 per cent neoarsphenamine) contained 38 per cent of the neoarsphenamine as arsenoxide; and on this basis the theoretical minimum lethal dose for rats should have been 85 mg. per kilo. The acute toxicity (death within 24 hours) was found to be 65 mg. per kilo (Table 4). The majority of the rats injected with 37.5 to 50 mg. per kilo died three days later. Some of them had marked nervous symptoms, such as tremor and ataxia. In the other toxicity experiment a portion of the neoarsphenamine precipitated out during the course of the oxidation. At the end of an hour the solution was centrifuged and the tests were done upon the supernatant fluid. The arsenoxide content was estimated to be 1.4 mg. per c. c. and the theoretical M. L. D. should have been 18.5 c. c. per kilo. The M. L. D. as actually determined was found to be from 15 to 18.5 c. c. per kilo. With smaller doses the same late symptoms and frequent late deaths occurred. In all of the neoarsphenamine experiments acute reactions were produced, although they were not as marked as with arsphenamine. The toxicity experiments with oxidized solutions of neoarsphenamine lead us to conclude that besides the indicated amount of arsenoxide there is present some other compound of enhanced toxicity which produces symptoms different from those of arsenoxide. In this connection it is of interest that the production of delayed deaths in rats from fresh solutions of neoarsphenamine is well recognized and is taken into account in the biological standardization of this product.

TABLE 4.—*The toxicity to rats of a 0.5 per cent neoarsphenamine solution in phosphate buffer pH 7.3, through which oxygen was bubbled for one hour at 38° C. Arsenozide content by color test=1.37 mg. per c. c. (33 per cent); Theoretical M. L. D.=85 mg. per kilo*

Rat weight	Dosage (mg. per kilo)	Time of death
100 grams.....	93	5 minutes.
102 grams.....	93	15 minutes.
104 grams.....	93	2 minutes.
110 grams.....	93	5 minutes.
104 grams.....	75	17 minutes.
104 grams.....	75	18 minutes.
108 grams.....	75	12 hours.
110 grams.....	75	22 minutes.
125 grams.....	75	16 hours.
100 grams.....	65	10 minutes.
112 grams.....	65	15 minutes.
112 grams.....	65	16 hours.
116 grams.....	65	5 minutes.
102 grams.....	50	16 hours.
104 grams.....	50	3 days.
106 grams.....	50	3½ days.
108 grams.....	50	3 days.
80 grams.....	37.5	Survived.
92 grams.....	37.5	3 days.
110 grams.....	37.5	20 hours.

With oxidized solutions of sulpharsphenamine the enhanced toxicity to rats was entirely sufficient to account for the estimated amount of arsenoxide present; as with neoarsphenamine the toxicity was slightly in excess of that anticipated. In one experiment after an hour of bubbling oxygen through the 0.5 per cent solution in phosphate buffer of pH 7.3 at 38° C., the arsenoxide content was 1.216 mg. per c. c., or 32 per cent of the molar concentration of the sulpharsphenamine. The theoretical minimum lethal dose was 90 mg. per kilo and the actual M. L. D. was 70 mg. per kilo (Table 5).

In another similar experiment the theoretical M. L. D. was 90 mg. per kilo, and that determined was 85 mg. per kilo. Typical and marked acute reactions were produced in all cases. Delayed deaths were not observed in these experiments but the number of surviving animals was too small to be conclusive.

TABLE 5.—*The toxicity to rats of a 0.5 per cent sulpharsphenamine solution in phosphate buffer pH 7.3, through which oxygen was bubbled for one hour at 38° C. Arsenoxide content by color test=1.218 mg. per c. c. (32 per cent). Theoretical M. L. D.=90 mg. per kilo*

Rat weight	Dosage (mg. per kilo)	Time of death
80 grams.....	100	8 minutes.
94 grams.....	100	5 minutes.
100 grams.....	100	13 minutes.
104 grams.....	100	5 minutes.
86 grams.....	85	10 minutes.
90 grams.....	85	10 minutes.
94 grams.....	85	10 minutes.
94 grams.....	85	7 minutes.
102 grams.....	85	3 minutes.
102 grams.....	85	50 minutes.
92 grams.....	70	16 hours.
100 grams.....	70	5 hours.
100 grams.....	70	20 hours.
104 grams.....	70	16 hours.
84 grams.....	50	Survived.
84 grams.....	50	16 hours.
98 grams.....	50	12 hours.
100 grams.....	50	16 hours.

ARSENOXIDE IN THE TISSUES OF ANIMALS FOLLOWING THE INJECTION OF ARSPHENAMINES

The first difficulty to be met in this phase of the work was the satisfactory extraction of arsenoxide from tissues. When arsenoxide was added to an organ it was found impossible to recover more than a trace of it with any of the protein precipitants generally employed. Our recent experience (19) had shown that arsenoxide combines firmly with the fixed sulphhydryl groups of the proteins. Further knowledge from the work of Voegtlin, Johnson, and Rosenthal (20) was at hand for the great affinity of certain heavy metals for these SH groups, particularly silver, cadmium, and lead. Experiments revealed that fairly good yields could be obtained when considerable amounts of these metals were employed along with trichloroacetic acid as a protein precipitant. Silver was the most effective and the easiest to remove from the acid solution. The technique finally employed was as follows:

Five grams of the organ is rapidly minced with fine scissors and ground with sand in a mortar for one or two minutes. Five c. c. of 20 per cent trichloroacetic acid is now added, and the mixture is ground one or two minutes longer and allowed to stand for 15 minutes. Ten c. c. of 4 per cent silver nitrate is now slowly added while stirring with

the pestle, and then 3 c. c. of methyl alcohol is added drop by drop, with stirring. The mixture is now filtered, away from bright light, into 5 c. c. of 8 per cent sodium chloride in a 25 c. c. graduated cylinder, and the final volume is recorded. This step precipitates out the silver and the solution is now filtered again, after which the naphthoquinone tests are performed upon the neutralized filtrate. Filtration should be rapid, and in both stages the filtrates should be perfectly clear and free from hemoglobin. By this procedure from 80 to 100 per cent of arsenoxide can be recovered when 2.5 to 5 mg. are added to 5 grams of organ. Standards should preferably be made up in filtrates from normal organs. Five tubes containing from 0.2 to 0.6 mg. of arsenoxide were employed. Two tenths c. c. of 50 per cent cadmium sulphate were added to both standards and unknowns.

The final filtrate usually represents a dilution such that 6 c. c. is equivalent to 1 gram of organ. Since this requires at least 0.25 mg. of arsenoxide per gram of organ to give a color reaction strong enough for quantitative determinations, it was necessary to employ maximum doses of the arsphenamines in order to reach this concentration of arsenoxide in the tissues. In this connection it must be clearly brought out that the negative tests obtained in some of the following experiments do not mean that arsenoxide is absent, but that it is present in concentrations too low to be detected by this method.

RESULTS WITH ARSPHENAMINE

From 200 to 250 mg. per kilogram of freshly alkalized arsphenamine was administered to rats by slow injection (10 to 15 minutes) into the femoral vein. The rats were killed by decapitation (to permit maximum exsanguination) at various intervals after the injection and the organs to be examined were removed immediately and treated as described in the previous paragraphs. In testing for arsenoxide in the kidneys or spleen, the corresponding organs of two rats were usually combined to give sufficient tissue for the analysis.

The results of 14 such experiments on rat livers are given in Table 6. Tests done within one hour following the injection were negative for arsenoxide. This is good substantiation of the specificity of the reaction; for Fordyce, Rosen, and Meyers (21) have shown that the maximum arsenic concentration occurs in the liver during this period. This finding is also in accord with the results of Voegtlin and Smith (5) demonstrating that a latent period of 1 to 3 hours is necessary, following the intravenous injection of arsphenamine in the rat, before the trypanocidal action becomes manifest.

TABLE 6.—Arsenoxide in liver following injection of arsphenamine

Rat weight	Dosage of arsphenamine (mg. per kilo)	Interval following injection	Arsenoxide in liver (mg. per gm.)	Approximate percentage of total arsphenamine
156 grams.....	250	10 minutes.....	None.....	-----
180 grams.....	250	¼ hour.....	None.....	-----
160 grams.....	250	1 hour.....	None.....	-----
156 grams.....	200	do.....	None.....	-----
140 grams.....	250	2½ hours.....	0.25.....	5.3
135 grams.....	250	do.....	0.35.....	7.0
108 grams.....	250	do.....	0.35.....	8.8
136 grams.....	250	3 hours.....	0.45.....	9.0
150 grams.....	250	do.....	0.3.....	6.1
200 grams.....	200	do.....	0.4.....	10.0
104 grams.....	250	3½ hours.....	0.5.....	12.0
100 grams.....	200	do.....	0.4.....	10.0
130 grams.....	250	4 hours.....	0.4.....	8.0
160 grams.....	200	4½ hours.....	0.5.....	10.0

Following the injection of arsphenamine, all of the tests done upon the rat liver at intervals of 2½ hours or later yielded positive reactions. The maximum concentrations occurred within 3 to 4 hours. The extracts were all tested for o-amino-phenol with negative results in every instance. Because of the large doses of arsphenamine employed, the rats were usually severely ill; when fatalities occurred, these animals were usually not employed for the study.

Although the arsenic content of the spleen has been shown to be as high as or higher than that of the liver following arsphenamine injections (21, 22, 23) concentrations of arsenoxide sufficient to be detected by the naphthoquinone test were not present in any of the animals studied. Tests done from ½ hour to 3 hours following the injection were all negative (Table 7). Likewise, tests done upon the kidneys and upon the blood serum of rats and rabbits following arsphenamine injections were entirely negative.

TABLE 7.—The inability to demonstrate arsenoxide in the spleen, kidneys, and blood serum, following arsphenamine injections

Animal	Organ	Dosage of arsphenamine (mg. per kilo)	Interval following injection	Arsenoxide color test
Rat.....	Spleen.....	250	¼ hour.....	Negative.
Do.....	do.....	250	2 hours.....	Do.
Do.....	do.....	250	3 hours.....	Do.
Do.....	do.....	250	do.....	Do.
Rat.....	Kidneys.....	250	¼ hour.....	Negative.
Do.....	do.....	250	3 hours.....	Do.
Do.....	do.....	250	3½ hours.....	Do.
Rat.....	Blood serum.....	250	3 hours.....	Negative.
Rabbit.....	do.....	200	10 minutes.....	Do.
Do.....	do.....	200	1½ hours.....	Do.
Do.....	do.....	200	do.....	Do.
Do.....	Blood plasma.....	200	2 hours.....	Do.
Do.....	do.....	200	3 hours.....	Do.

Different results were obtained with nearsphenamine. Although a dosage one-third greater (on a basis of arsenic content) was used, no arsenoxide could be detected in the liver (Table 8). In the kidney a negative test was obtained 20 minutes after injection, while in five experiments where tests were done from 1¼ to 4½ hours after injection the reactions were all positive (Table 9). The concentrations reached were similar to those in the liver following arsphenamine, but the relative weights of the kidneys made the total amounts of arsenoxide present much less. No arsenoxide could be detected in the spleen following the nearsphenamine injections. All of the above-described extracts were tested for o-aminophenol with negative results.

TABLE 8.—*The inability to demonstrate arsenoxide in the liver and spleen following injection of nearsphenamine in rats*

Organ	Dosage of nearsphenamine (mg. per kilo)	Interval following injection	Arsenoxide color test
Liver.....	500	10 minutes.....	Negative.
Do.....	500	1 hour.....	Do.
Do.....	500	2 hours.....	Do.
Do.....	500	3 hours.....	Do.
Do.....	500	3¼ hours.....	Do.
Do.....	500	4½ hours.....	Do.
Spleen ¹	500	2½ hours.....	Negative.
Do. ¹	500	4 hours.....	Do.

¹ Organs combined for the determination.

TABLE 9.—*Arsenoxide in kidneys following injection of nearsphenamine in rats (the organs of two animals combined for each determination)*

Weight of rat	Dosage of nearsphenamine (mg. per kilo)	Interval following injection	Arsenoxide in kidneys (mg. per gm.)	Approximate percentage of total nearsphenamine
100}	500	20 minutes.....	Negative.	-----
140}		1¼ hours.....		
136}	500	2½ hours.....	.5	1.5
204}		----- do.....		
164}	500	4 hours.....	Trace.	-----
160}		4½ hours.....		
140}	500	-----	-----	-----
114}		-----		
162}	500	-----	-----	-----
130}		-----		
125}	-----	-----	-----	-----

CONTROL EXPERIMENTS

For the sake of completeness we are including some of the control experiments designed to show that any chemical changes which might be produced in the body by the action of arsenic itself, as well as the

presence of the arsphenamines in the tissues or extracts, play no part in the production of the color reaction.

1. In five experiments, from 6 to 10 mg. of arsphenamine were added to 5 gm. of freshly removed liver, which was then macerated, ground with sand, and extracted by the usual procedure. The extracts were all negative with the naphthoquinone test. Likewise, 10 mg. of neoarsphenamine added to liver gave negative results. Twenty mg. of neoarsphenamine were added to 5 gm. of kidney and treated in this manner. The filtrate gave a pale orange-yellow color with the test.

2. To 5 c. c. of the filtrate obtained from a normal liver 1.2 mg. of arsphenamine was added. A negative color test was obtained.

3. A filtrate from the kidneys of a rat injected with neoarsphenamine showed 0.5 mg. per gram of arsenoxide. The test for *o*-aminophenol was negative; 0.05 mg. of *o*-aminophenol was now added to 5 c. c. of the filtrate. A test for *o*-aminophenol compared to a standard showed quantitative recovery.

4. Seven rats were injected with a toxic dose (10 c. c. of $n/200$ solution per kilo) of arsenious oxide, As_2O_3 . Naphthoquinone tests done upon the liver at 10 minutes, $2\frac{1}{2}$ hours, 3 hours, 4 hours, and 18 hours, were negative. To 5 gm. of the liver removed at 3 hours and at 4 hours were added 6 mg. arsphenamine each, and extracted. The extracts were both negative.

DISCUSSION

The recovery of considerable amounts of arsenoxide from the liver of the rat following the injection of arsphenamine and from the kidney following neoarsphenamine, confirms the theory of Voegtlin and Smith that arsenoxide is formed from these compounds in the living body. In harmony with their view, that this compound is responsible for the trypanocidal action, is the fact that the interval of time required before arsenoxide can be demonstrated in the tissues is similar to the latent period required by these drugs before their trypanocidal action in the body is manifest.

It will also be observed from results obtained following the injection of lethal doses of arsphenamine that there is fixed in the liver an amount of arsenoxide which would be fatal if injected intravenously. This evidence supports the view of Voegtlin (6) that the toxicity of arsphenamine to the host is due to arsenoxide. Dale (24) has emphasized the importance of the slow liberation into the circulation of arsenoxide from arsphenamine as a basis for therapeutic efficiency and safety to the host. Jackson and Raap (25) showed that the severe circulatory disturbances resulting from the peripheral intravenous injection of arsphenamine were largely absent if the drug

was injected into the portal vein. The general circulatory effects and pulmonary lesions produced by arsenoxide play an important rôle in the acute toxicity of this compound and its formation from arsphenamine in certain tissues, with subsequent fixation there, would permit larger amounts to be present in the body without general toxic effects.

The actual concentrations of arsenoxide present in the tissues may be slightly higher than those obtained in our experiments, since complete recovery of arsenoxide added to an organ is not always possible. One factor that may be responsible for the incomplete recovery may be the presence of glutathione, which we have found inhibits the color reaction if present in considerable amounts. However, nitroprusside tests upon the filtrates of normal and arsenic-containing organs have been negative, which leads us to believe that under these conditions the glutathione is precipitated out by the silver used in the extraction.

The differences in distribution of arsenoxide following arsphenamine and neoarsphenamine injections are of particular interest in view of a similar distribution of the pathological changes produced by them. Arsphenamine produces lesions principally in the liver, while with neoarsphenamine the pathological changes are chiefly in the kidney (Kolmer and Lucke (26)). Since the studies of Fordyce, Rosen, and Myers (21) reveal that the concentrations of arsenic which are reached in these organs are very similar when large doses of either arsphenamine or neoarsphenamine are injected into rats, our experiments would suggest that it is not the total arsenic but the arsenic in the form of arsenoxide that plays an important part in the production of these pathological effects.

The naphthoquinone reaction can be applied as a corollary to tests of toxicity upon commercial samples of arsphenamine. We are investigating the problem of toxicity of samples of arsphenamine as related to their arsenoxide content. A future communication will be published upon this subject.

SUMMARY

A color reaction with 1, 2 naphthoquinone-4-sodium sulphonate has been developed which distinguishes arsenoxide (3-amino-4-hydroxyphenyl arsenious oxide) in dilute solution from all other arsenicals tested. Among all of the other compounds studied, the only one which reacts similarly is ortho-aminophenol. By a modification of the test it is possible to distinguish between these two compounds.

This test has been employed to study quantitatively the formation of arsenoxide from solutions of arsphenamine, neoarsphenamine, and sulpharsphenamine when oxygen is bubbled through them. With

arsphenamine this proceeds slowly at pH 7.3, owing to solubility factors, but rapidly at pH 9.5 or above.

With sulpharsphenamine, and particularly with neoarsphenamine, the formation of arsenoxide is very rapid at pH 7.3. The high concentrations of arsenoxide reached give evidence that with both compounds the sulphur radical is split off from the amino group during the process of oxidation.

Toxicity tests upon rats with the oxidized solutions of the arsphenamines substantiate the quantitative estimations of arsenoxide yielded by the color reaction.

A method has been developed whereby from 80 to 100 per cent of arsenoxide added to tissues can be recovered from them in protein-free extracts. With this method of extraction it has been possible to demonstrate the presence of arsenoxide in the tissues of rats following the injection of large doses of arsphenamine and neoarsphenamine.

From 10 to 12 per cent of the injected arsphenamine can be recovered as arsenoxide from the liver of the rat if the estimations are made from 3 to 4 hours after injection. No arsenoxide could be detected in other tissues after arsphenamine, although the lack of sensitivity of the test makes it possible that quantities insufficient to detect are present.

Following the injection of large doses of neoarsphenamine, arsenoxide could be recovered only from the kidney of the rat. The highest concentration of arsenoxide reached in this organ was 0.5 mg. per gram of kidney. This is comparable to the concentration reached in the liver after arsphenamine injections.

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DEATHS DURING WEEK ENDED APRIL 2, 1932

Summary of information received by telegraph from industrial insurance companies for the week ended April 2, 1932, and corresponding week of 1931. (From the Weekly Health Index, issued by the Bureau of the Census, Department of Commerce)

	Week ended Apr. 2, 1932	Correspond- ing week, 1931
Policies in force.....	73, 717, 468	75, 139, 274
Number of death claims.....	18, 540	13, 411
Death claims per 1,000 policies in force, annual rate.....	13. 1	9. 3
Death claims per 1,000 policies, first 13 weeks of year, annual rate.....	10. 4	11. 1

Deaths¹ from all causes in certain large cities of the United States during the week ended April 2, 1932, infant mortality, annual death rate, and comparison with corresponding week of 1931. (From the Weekly Health Index, issued by the Bureau of the Census, Department of Commerce)

[The rates published in this summary are based upon mid-year population estimates derived from the 1930 census]

City	Week ended Apr. 2, 1932				Corresponding week, 1931		Death rate ² for the first 13 weeks	
	Total deaths	Death rate ²	Deaths under 1 year	Infant mortality rate ³	Death rate ²	Deaths under 1 year	1932	1931
Total (84 cities).....	9, 427	13. 5	673	• 58	13. 0	7•2	12. 7	14. 0
Akron.....	32	6. 3	3	37	6. 7	5	7. 8	8. 5
Albany.....	44	17. 6	1	20	12. 9	1	15. 1	15. 3
Atlanta.....	71	13. 1	5	49	13. 9	12	14. 2	16. 3
White.....	39	10. 9	4	59	9. 6	3	11. 1	13. 1
Colored.....	32	17. 5	1	29	22. 4	9	20. 3	22. 6
Baltimore.....	254	16. 2	24	85	15. 6	18	15. 0	17. 5
White.....	191	14. 9	13	59	13. 6	13	13. 9	16. 1
Colored.....	63	21. 9	11	177	24. 9	5	19. 9	23. 8
Birmingham.....	60	11. 3	3	31	19. 7	7	12. 3	15. 8
White.....	29	8. 8	0	0	15. 3	5	10. 1	12. 2
Colored.....	31	15. 4	3	81	26. 9	2	15. 8	21. 7
Boston.....	250	16. 6	20	60	14. 7	22	15. 6	16. 5
Bridgeport.....	35	12. 4	1	18	11. 3	1	12. 2	13. 2
Buffalo.....	191	17. 0	9	43	14. 4	13	14. 2	15. 6
Cambridge.....	33	15. 1	2	41	12. 8	2	14. 2	13. 8
Camden.....	52	22. 8	4	70	15. 3	2	16. 1	18. 2
Canton.....	25	12. 1	2	50	13. 2	4	10. 9	11. 4
Chicago.....	744	11. 0	58	57	11. 0	73	11. 1	12. 0
Cincinnati.....	140	15. 8	4	26	17. 2	11	16. 9	18. 2
Cleveland.....	229	13. 0	23	75	13. 5	15	12. 0	12. 8
Columbus.....	77	13. 4	5	50	17. 1	4	14. 7	15. 3
Dallas.....	63	11. 7	4	-----	14. 5	4	11. 7	12. 7
White.....	42	9. 4	3	-----	12. 7	3	10. 9	11. 1
Colored.....	21	22. 5	1	-----	23. 1	1	15. 7	20. 6
Dayton.....	47	10. 3	1	14	13. 5	5	11. 9	12. 7
Denver.....	85	15. 1	2	20	15. 4	5	16. 9	15. 9
Des Moines.....	51	18. 3	4	69	9. 0	1	12. 6	12. 4
Detroit.....	296	9. 1	29	52	9. 9	34	8. 8	9. 9
Duluth.....	26	13. 3	3	87	7. 2	0	10. 3	11. 6
El Paso.....	27	13. 2	1	-----	20. 4	5	15. 0	18. 1
Erie.....	28	12. 3	2	42	10. 6	2	12. 2	11. 7
Evansville.....	19	9. 4	0	0	10. 0	1	10. 2	12. 1
Fall River.....	25	11. 3	1	27	14. 9	2	13. 1	14. 1
Flint.....	35	10. 8	3	44	6. 0	4	9. 1	7. 9
Fort Wayne.....	26	11. 2	3	77	10. 1	0	11. 1	12. 0
Fort Worth.....	39	11. 9	1	-----	13. 4	3	11. 0	12. 0
White.....	27	9. 8	1	-----	14. 5	3	10. 5	11. 6
Colored.....	12	23. 5	0	-----	7. 7	0	13. 7	14. 0
Grand Rapids.....	36	10. 8	1	17	8. 2	1	9. 9	9. 7
Houston.....	91	14. 7	6	-----	10. 3	6	11. 3	11. 8
White.....	63	13. 8	5	-----	9. 0	3	10. 6	10. 8
Colored.....	28	17. 1	1	-----	13. 8	3	13. 0	14. 5
Indianapolis.....	94	13. 1	2	16	12. 5	5	14. 1	15. 5
White.....	80	12. 7	2	18	11. 4	4	13. 6	14. 9
Colored.....	14	15. 9	0	0	20. 8	1	17. 8	19. 9
Jersey City.....	78	12. 7	6	50	14. 7	14	12. 1	14. 0
Kansas City, Kans.....	35	14. 8	2	44	14. 8	1	13. 6	16. 1
White.....	29	15. 1	1	27	15. 2	1	13. 2	14. 8
Colored.....	6	13. 2	1	178	13. 3	0	15. 1	21. 3
Kansas City, Mo.....	141	17. 7	15	120	14. 4	10	13. 5	15. 5
Knoxville.....	34	15. 9	1	25	14. 8	2	13. 1	14. 7
White.....	22	12. 3	1	28	14. 8	2	11. 9	13. 7
Colored.....	12	34. 3	0	0	14. 6	0	19. 1	19. 8
Long Beach.....	20	6. 5	0	0	9. 2	0	10. 3	10. 5
Los Angeles.....	277	10. 5	25	74	9. 2	16	11. 9	11. 6
Louisville.....	119	20. 2	4	37	17. 9	11	14. 9	17. 8
White.....	95	19. 0	4	42	16. 6	7	13. 2	16. 0
Colored.....	24	26. 3	0	0	25. 1	4	23. 8	28. 1
Lowell.....	35	18. 3	3	78	16. 1	2	15. 0	15. 2
Lynn.....	19	9. 6	1	28	12. 2	1	12. 0	12. 9
Memphis.....	101	20. 0	11	120	22. 6	14	17. 2	18. 3
White.....	44	14. 1	3	51	19. 9	6	13. 0	15. 5
Colored.....	57	29. 6	8	241	26. 9	8	23. 8	22. 9
Miami.....	26	11. 9	4	112	9. 7	1	12. 8	14. 4
White.....	21	12. 4	1	39	8. 4	0	12. 1	13. 6
Colored.....	5	10. 3	3	302	14. 4	1	15. 3	17. 1

See footnotes at end of table.

Deaths¹ from all causes in certain large cities of the United States during the week ended April 2, 1932, infant mortality, annual death rate, and comparison with corresponding week of 1931. (From the Weekly Health Index, issued by the Bureau of the Census, Department of Commerce)—Continued

City	Week ended Apr. 2, 1932				Corresponding week, 1931		Death rate ² for the first 13 weeks	
	Total deaths	Death rate ³	Deaths under 1 year	Infant mortality rate ³	Death rate ³	Deaths under 1 year	1932	1931
Milwaukee.....	114	9.9	8	38	9.6	11	9.6	10.9
Minneapolis.....	116	12.6	6	39	11.9	13	11.7	12.4
Nashville ⁴	46	15.3	3	45	23.1	6	15.1	18.8
White.....	30	13.8	2	39	15.3	5	14.4	16.0
Colored.....	16	19.5	1	62	43.9	1	17.2	26.2
New Bedford ⁵	35	18.3	3	86	13.0	6	13.8	13.3
New Haven.....	63	20.2	2	40	10.9	3	13.9	13.6
New Orleans ⁶	138	15.2	10	57	18.0	19	16.0	19.4
White.....	84	13.0	4	35	15.2	7	13.5	16.0
Colored.....	54	20.5	6	98	24.8	12	22.1	27.8
New York.....	1,775	12.9	146	65	12.8	148	12.1	13.6
Bronx Borough.....	244	9.2	20	58	9.0	25	9.1	9.8
Brooklyn Borough.....	609	11.9	52	58	11.9	52	11.3	12.7
Manhattan Borough.....	691	20.3	60	86	19.9	53	18.3	20.7
Queens Borough.....	177	7.6	10	42	7.5	17	7.7	8.8
Richmond Borough.....	54	16.9	4	79	16.0	1	15.1	14.6
Newark, N. J.....	124	14.5	10	55	13.0	12	12.1	13.8
Oakland.....	66	11.5	1	13	11.1	2	11.7	12.0
Oklahoma City.....	39	9.9	0	0	16.4	7	10.4	12.1
Omaha.....	66	15.8	7	79	13.5	2	15.4	14.8
Paterson.....	38	14.3	11	200	11.3	3	13.7	16.2
Peoria.....	24	11.3	1	28	16.4	1	12.8	14.1
Philadelphia.....	747	19.7	43	66	13.7	62	13.9	16.2
Pittsburgh.....	166	12.7	16	73	15.5	15	15.0	18.0
Portland, Oreg.....	75	12.6	3	38	12.1	3	12.6	13.0
Providence.....	104	21.2	9	87	14.1	12	15.7	15.4
Richmond ⁶	53	14.9	1	15	15.6	4	14.9	18.0
White.....	26	10.3	0	0	14.7	1	12.3	15.2
Colored.....	27	26.7	1	46	17.7	3	21.6	24.9
Rochester.....	78	12.2	9	86	12.4	6	12.6	14.0
St. Louis.....	286	18.0	13	46	18.2	10	14.8	18.5
St. Paul.....	59	11.0	2	32	11.5	4	11.2	11.9
Salt Lake City ⁴	25	9.0	2	31	10.6	4	11.8	12.7
San Diego.....	51	16.3	0	0	17.0	3	16.6	15.7
San Francisco.....	155	12.2	5	35	12.4	5	13.9	14.6
Schenectady.....	26	14.1	3	87	10.3	1	11.5	12.2
Seattle.....	62	11.4	6	60	15.4	5	12.3	13.4
Somerville.....	31	15.2	1	40	11.9	2	10.4	11.4
South Bend.....	19	8.9	0	0	9.2	3	8.1	9.4
Spokane.....	32	14.3	1	27	13.9	0	18.0	13.3
Springfield, Mass.....	32	10.8	2	34	14.0	7	11.9	13.9
Syracuse.....	56	13.5	4	52	11.5	4	12.3	12.9
Tacoma.....	23	11.1	2	55	13.1	2	12.4	15.0
Tampa ⁶	21	10.2	1	29	12.9	3	12.5	14.9
White.....	15	9.2	0	0	11.3	1	12.2	13.6
Colored.....	6	13.8	1	158	18.8	2	13.8	19.9
Toledo.....	63	10.9	6	65	13.9	7	13.0	13.9
Trenton.....	67	28.2	5	99	21.5	7	17.5	19.6
Utica.....	40	20.3	4	114	17.8	0	16.0	16.7
Washington, D. C. ⁶	188	19.9	10	56	14.8	9	17.6	18.6
White.....	115	16.8	2	16	12.2	5	15.9	16.1
Colored.....	73	27.9	8	142	21.6	4	22.0	28.1
Waterbury.....	25	12.9	1	33	10.9	7	10.5	11.3
Wilmington, Del. ⁷	57	28.0	3	68	16.6	4	16.5	16.8
Worcester.....	62	13.7	6	84	14.0	9	13.7	15.2
Yonkers.....	17	6.3	0	0	4.1	3	7.9	10.4
Youngstown.....	41	12.2	7	114	13.0	8	11.2	11.9

¹ Deaths of nonresidents are included. Stillbirths are excluded.

² These rates represent annual rates per 1,000 population, as estimated for 1932 and 1931 by the arithmetical method.

³ Deaths under 1 year of age per 1,000 estimated live births. Cities left blank are not in the registration area for births.

⁴ Data for 80 cities.

⁵ Deaths for week ended Friday.

⁶ For the cities for which deaths are shown by color the percentages of colored population in 1930 were as follows: Atlanta, 33; Baltimore, 18; Birmingham, 38; Dallas, 17; Fort Worth, 16; Houston, 27; Indianapolis, 12; Kansas City, Kans., 19; Knoxville, 16; Louisville, 16; Memphis, 38; Miami, 23; Nashville, 28; New Orleans, 29; Richmond, 29; Tampa, 21; and Washington, D. C., 27.

⁷ Population Apr. 1, 1930; decreased 1929 to 1930, no estimate made.

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 April 9, 1932, and April 11, 1931

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended April 9, 1932, and April 11, 1931

Division and State	Diphtheria		Influenza		Measles		Meningococcus meningitis	
	Week ended Apr. 9, 1932	Week ended Apr. 11, 1931	Week ended Apr. 9, 1932	Week ended Apr. 11, 1931	Week ended Apr. 9, 1932	Week ended Apr. 11, 1931	Week ended Apr. 9, 1932	Week ended Apr. 11, 1931
New England States:								
Maine.....	1	3	7	6	246	18	0	0
New Hampshire.....					13	38	0	0
Vermont.....	3				73	3	0	0
Massachusetts.....	30	52	12	8	661	478	5	3
Rhode Island.....	5	4		1	133	40	1	0
Connecticut ¹	6	8	19	5	112	795	2	0
Middle Atlantic States:								
New York.....	111	119	160	120	2,484	2,137	7	17
New Jersey.....	29	51	67	24	573	920	0	8
Pennsylvania.....	90	96			1,947	4,740	5	14
East North Central States:								
Ohio.....	35	52	71	115	820	852	1	2
Indiana.....	36	28	138	32	83	953	9	15
Illinois.....	104	146	86	18	1,649	1,650	0	27
Michigan.....	14	15	28	18	2,294	93	2	13
Wisconsin.....	3	9	390	58	1,007	682	1	4
West North Central States:								
Minnesota.....	12	7	5	1	61	137	0	2
Iowa.....	3	3			3	19	2	2
Missouri.....	15	26	34	30	60	447	1	12
North Dakota.....		4			52	84	0	0
South Dakota.....		5	2	5	14	168	0	0
Nebraska.....	2	16			1	7	0	1
Kansas.....	10	9	12	3	270	23	2	3
South Atlantic States:								
Delaware.....		3	7		2	228	0	0
Maryland ¹	10	16	303	40	46	1,396	3	4
District of Columbia.....	7	6	3	3	9	373	2	0
Virginia.....							4	4
West Virginia.....	16	8	367	168	419	94	5	1
North Carolina.....	22	20	108	32	428	1,015	1	2
South Carolina.....	6	4	2,262	1,153	118	105	0	4
Georgia ¹	15	6	209	410	33	146	1	1
Florida.....	6	6	5	68	6	260	2	1

¹ Typhus fever, 10 cases: 1 case in Connecticut, 7 cases in Georgia, and 2 cases in Alabama.

² New York City only.

³ Week ended Friday.

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended April 9, 1932, and April 11, 1931—Continued

Division and State	Diphtheria		Influenza		Measles		Meningococcus meningitis	
	Week ended Apr. 9, 1932	Week ended Apr. 11, 1931	Week ended Apr. 9, 1932	Week ended Apr. 11, 1931	Week ended Apr. 9, 1932	Week ended Apr. 11, 1931	Week ended Apr. 9, 1932	Week ended Apr. 11, 1931
East South Central States:								
Kentucky.....	12		469		53	362	0	4
Tennessee.....	3	2	739	206	209	51	3	2
Alabama ¹	18	12	294	345	10	483	1	5
Mississippi.....	0	2					0	2
West South Central States:								
Arkansas.....	3	2	198	209		24	0	1
Louisiana.....	28	15	37	57	27	8	1	2
Oklahoma ¹	7	19	231	170	10	29	3	2
Texas.....	39	20	625	77	57	67	0	1
Mountain States:								
Montana.....	5	3	13		138	72	0	0
Idaho.....	1	5	3	4		5	0	1
Wyoming.....		1			4	3	0	0
Colorado.....	4	10			139	139	0	0
New Mexico.....	10	1	1	6	50	46	0	2
Arizona.....	1		9	3	2	31	0	0
Utah ¹	2			6		2	0	1
Pacific States:								
Washington.....	1	10		37	513	35	0	0
Oregon.....	4	6	65	72	332	113	0	1
California.....	62	70	62	100	534	1,532	5	7
Total	802	900	7,000	3,510	13,702	20,892	69	168

Division and State	Polioomyelitis		Scarlet fever		Smallpox		Typhoid fever	
	Week ended Apr. 9, 1932	Week ended Apr. 11, 1931	Week ended Apr. 9, 1932	Week ended Apr. 11, 1931	Week ended Apr. 9, 1932	Week ended Apr. 11, 1931	Week ended Apr. 9, 1932	Week ended Apr. 11, 1931
New England States:								
Maine.....	0	0	21	37	0	0	0	2
New Hampshire.....	0	0	32	0	0	0	0	0
Vermont.....	0	0	7	2	3	1	0	0
Massachusetts.....	0	0	500	342	0	0	1	0
Rhode Island.....	0	0	71	56	0	0	0	0
Connecticut ¹	1	0	85	55	0	0	1	1
Middle Atlantic States:								
New York.....	1	5	1,442	932	0	5	6	16
New Jersey.....	1	0	282	287	0	0	2	3
Pennsylvania.....	3	0	578	640	0	0	7	11
East North Central States:								
Ohio.....	1	0	351	490	45	78	5	1
Indiana.....	1	0	173	320	12	91	0	2
Illinois.....	1	1	439	512	10	62	5	7
Michigan.....	1	0	436	280	13	31	11	2
Wisconsin.....	1	1	106	123	3	5	1	0
West North Central States:								
Minnesota.....	0	0	124	82	1	6	0	1
Iowa.....	0	1	26	119	27	73	3	0
Missouri.....	0	0	62	269	18	30	1	0
North Dakota.....	0	1	26	28	3	14	0	6
South Dakota.....	1	0	4	31	2	19	0	0
Nebraska.....	0	0	31	38	11	116	0	0
Kansas.....	0	1	70	66	6	116	0	2
South Atlantic States:								
Delaware.....	1	0	11	31	0	0	0	0
Maryland ¹	0	0	155	71	0	0	6	6
District of Columbia.....	1	0	23	20	0	0	0	1
Virginia.....								
West Virginia.....	1	0	26	44	3	2	1	4
North Carolina.....	0	1	44	30	1	2	7	2
South Carolina.....	0	1	9	8	0	0	0	0
Georgia ¹	0	1	7	107	0	0	11	2
Florida.....	0	0	6	2	0	1	15	3

¹ Typhus fever, 10 cases: 1 case in Connecticut, 7 cases in Georgia, and 2 cases in Alabama.

² Week ended Friday.

⁴ Figures for 1932 are exclusive of Oklahoma City and Tulsa.

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended April 9, 1932, and April 11, 1931—Continued

Division and State	Poliomyelitis		Scarlet fever		Smallpox		Typhoid fever	
	Week ended Apr. 9, 1932	Week ended Apr. 11, 1931	Week ended Apr. 9, 1932	Week ended Apr. 11, 1931	Week ended Apr. 9, 1932	Week ended Apr. 11, 1931	Week ended Apr. 9, 1932	Week ended Apr. 11, 1931
East South Central States:								
Kentucky.....	1	0	63	84	9	33	8	1
Tennessee.....	1	0	32	35	14	9	7	6
Alabama ¹	0	0	14	16	11	16	6	3
Mississippi.....	0	1	13	21	23	64	2	5
West South Central States:								
Arkansas.....	0	0	5	21	6	39	2	5
Louisiana.....	0	1	15	18	6	40	16	3
Oklahoma ¹	0	0	28	45	5	104	1	3
Texas.....	0	1	62	42	113	40	3	4
Mountain States:								
Montana.....	0	0	10	20	0	6	1	1
Idaho.....	3	1	3	9	0	4	0	2
Wyoming.....	0	0	6	13	0	1	2	0
Colorado.....	0	0	30	23	3	5	1	0
New Mexico.....	0	0	18	10	0	0	4	1
Arizona.....	0	0	11	0	0	1	1	0
Utah ¹	0	0	8	7	0	0	0	0
Pacific States:								
Washington.....	0	0	38	48	29	51	1	3
Oregon.....	0	0	20	9	8	21	3	3
California.....	0	4	161	111	7	42	10	10
Total.....	20	20	5,696	5,551	392	1,060	157	124

¹ Typhus fever, 10 cases: 1 case in Connecticut, 7 cases in Georgia, and 2 cases in Alabama.

¹ Week ended Friday.

* Figures for 1932 are exclusive of Oklahoma City and Tulsa.

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
<i>February, 1932</i>										
Kansas.....	15	81	101		520		0	245	10	7
Mississippi.....	2	71	3,585	1,486	24	283	2	43	119	32
<i>March, 1932</i>										
Arizona.....	4	11	257		7	2	1	33	1	4
Connecticut.....	2	25	203		978		0	546	8	1
District of Columbia.....	11	45	36		10	1	1	146	0	
Florida.....		48	30	15	16	8	1	27	1	38
Georgia.....	5	34	692	38	159	24	0	33		42
Iowa.....	8	50			13		2	263	100	7
Nebraska.....	3	30	112		70		0	138	43	3
New Hampshire.....		5					0	156		
New Mexico.....	3	54	709	1	380	2	0	47	2	2

The following table gives the number of new admissions for the month of July, 1930, by psychoses:

Psychoses	Male	Female	Total
1. Traumatic psychoses.....	18	14	32
2. Senile psychoses.....	160	126	286
3. Psychoses with cerebral arteriosclerosis.....	210	114	324
4. General paralysis.....	241	53	294
5. Psychoses with cerebral syphilis.....	37	10	47
6. Psychoses with Huntington's chorea.....	1	3	4
7. Psychoses with brain tumor.....	2	2	4
8. Psychoses with other brain or nervous disease.....	36	10	46
9. Alcoholic psychoses.....	116	13	129
10. Psychoses due to drugs and other exogenous toxins.....	19	9	28
11. Psychoses with pellagra.....	15	34	49
12. Psychoses with other somatic diseases.....	28	49	77
13. Manic-depressive psychoses.....	210	291	501
14. Involution melancholia.....	22	44	66
15. Dementia præcox (schizophrenia).....	407	326	733
16. Paranoia and paranoid conditions.....	37	32	69
17. Epileptic psychoses.....	40	43	83
18. Psychoneuroses and neuroses.....	28	60	88
19. Psychoses with psychopathic personality.....	32	8	40
20. Psychoses with mental deficiency.....	64	52	116
21. Undiagnosed psychoses.....	131	81	212
22. Without psychosis.....	190	64	254
Total.....	2,044	1,438	3,482

During the month of July, 1930, there were 3,482 new admissions to the hospitals, 58.7 per cent of these new admissions being males and 41.3 per cent females, the ratio being 142 males per 100 females. Four hundred and sixty-six of the new admissions were reported as being undiagnosed or without psychosis. There were 3,016 new admissions for whom provisional diagnoses were made. Of these 3,016 patients, cases of dementia præcox constituted 24.3 per cent; manic-depressive psychoses, 16.6 per cent; psychoses with cerebral arteriosclerosis, 10.7 per cent; general paralysis, 9.7 per cent; and senile psychoses, 9.5 per cent. These five classes accounted for 2,138 new patients, 70.9 per cent of the new admissions for whom diagnoses were made.

The following table shows the number of patients in the hospitals and on parole on July 31, 1930.

	Male	Female	Total
Patients on books last day of month:			
In hospitals.....	86,452	76,186	162,638
On parole or otherwise absent but still on books.....	8,475	6,915	15,390
Total.....	94,927	83,101	178,028

Of the 178,028 patients, 8,475 males and 6,915 females were on parole or otherwise absent but still on the books at the end of the month—8.9 per cent of the males, 8.3 per cent of the females, and 8.6 per cent of the total number of patients.

INFLUENZA CASE RATES, MARCH 13 TO APRIL 9, 1932

In the table following are presented the influenza case rates, by weeks, per 100,000 population, annual basis, in geographic groups of States, as indicated by weekly reports, for the four weeks from March 13 to April 9, 1932, and similar rates for the corresponding period of 1931. The rates are calculated in groups and as a whole on the reported cases and estimated populations of 35 States, the District of Columbia, and New York City. The States included are the same as shown for a similar table on pages 571 and 572, of the PUBLIC HEALTH REPORTS of March 4, 1932. Complete figures are not available for the States which are omitted from the table. Similar rates for the period from February 21 to March 12, 1932, are shown on page 736 of the PUBLIC HEALTH REPORTS of March 25, 1932.

Influenza case rates per 100,000 population

	Week ended--							
	1932				1931			
	Mar. 19	Mar. 26	Apr. 2	Apr. 9	Mar. 21	Mar. 28	Apr. 4	Apr. 11
35 States.....	615	409	453	378	252	206	239	187
New England.....	86	54	215	29	60	41	24	15
Middle Atlantic.....	206	120	93	58	46	22	28	20
East North Central.....	365	203	287	144	63	97	64	49
West North Central.....	35	42	27	27	53	51	39	20
South Atlantic.....	940	1,095	1,201	1,279	819	1,098	888	724
East South Central.....	2,481	1,344	1,417	984	456	677	418	362
West South Central.....	674	392	400	473	249	248	232	215
Mountain.....	2,057	180	132	65	58	150	451	33
Pacific.....	255	213	139	96	485	343	202	132

GENERAL CURRENT SUMMARY AND WEEKLY REPORTS FROM CITIES

The 96 cities reporting cases used in the following table are situated in all parts of the country and have an estimated aggregate population of more than 33,800,000. The estimated population of the 89 cities reporting deaths is more than 32,240,000. The estimated expectancy is based on the experience of the last nine years, excluding epidemics.

Weeks ended April 2, 1932, and April 4, 1931

	1932	1931	Estimated expectancy
<i>Cases reported</i>			
Diphtheria:			
46 States.....	862	852	
96 cities.....	306	340	745
Measles:			
45 States.....	15,729	19,091	
96 cities.....	5,504	7,185	
Meningococcus meningitis:			
46 States.....	111	154	
96 cities.....	52	86	
Poliomyelitis:			
46 States.....	20	19	
Scarlet fever:			
46 States.....	6,724	5,731	
96 cities.....	2,677	2,364	1,567
Smallpox:			
46 States.....	381	1,006	
96 cities.....	26	88	63
Typhoid fever:			
46 States.....	153	115	
96 cities.....	33	25	25
<i>Deaths reported</i>			
Influenza and pneumonia:			
89 cities.....	1,212	1,183	
Smallpox:			
89 cities.....	0	0	

City reports for week ended April 2, 1932

The "estimated expectancy" given for diphtheria, poliomyelitis, scarlet fever, smallpox, and typhoid fever is the result of an attempt to ascertain from previous occurrence the number of cases of the disease under consideration that may be expected to occur during a certain week in the absence of epidemics. It is based on reports to the Public Health Service during the past nine years. It is in most instances the median number of cases reported in the corresponding weeks of the preceding years. When the reports include several epidemics, or when for other reasons the median is unsatisfactory, the epidemic periods are excluded, and the estimated expectancy is the mean number of cases reported for the week during non-epidemic years.

If the reports have not been received for the full nine years, data are used for as many years as possible, but no year earlier than 1923 is included. In obtaining the estimated expectancy, the figures are smoothed when necessary to avoid abrupt deviation from the usual trend. For some of the diseases given in the table the available data were not sufficient to make it practicable to compute the estimated expectancy.

Division, State, and city	Chick- en pox, cases re- ported	Diphtheria		Influenza		Meas- les, cases re- ported	Mumps, cases re- ported	Pneu- monia, deaths, re- ported
		Cases, esti- mated expect- ancy	Cases reported	Cases reported	Deaths reported			
NEW ENGLAND								
Maine:								
Portland	2	0	0	1	0	43	8	4
New Hampshire:								
Concord	0	0	0	0	0	0	0	0
Manchester	0	0	0	0	2	0	0	3
Nashua	0	0	0	0	0	0	0	0
Vermont:								
Barre	0	0	0	0	0	0	0	1
Burlington	0	0	0	0	0	2	0	0
Massachusetts:								
Boston	51	27	12	1	2	65	80	26
Fall River	1	3	0	2	0	65	1	2
Springfield	26	3	0	0	0	38	16	1
Worcester	3	3	0	2	0	0	15	8
Rhode Island:								
Pawtucket	1	7	0	0	0	0	0	0
Providence	2	1	4	1	1	97	7	15
Connecticut:								
Bridgeport	0	5	0	0	1	9	0	6
Hartford	1	4	0	0	0	0	0	0
New Haven	16	0	0	5	3	2	3	3
MIDDLE ATLANTIC								
New York:								
Buffalo	16	10	3	0	2	11	1	28
New York	175	212	81	113	37	175	135	262
Rochester	6	6	0	2	0	255	5	5
Syracuse	3	3	1	0	0	640	3	6
New Jersey:								
Camden	5	4	0	3	6	1	1	8
Newark	41	16	4	18	2	25	44	12
Trenton	0	2	1	5	4	2	1	11
Pennsylvania:								
Philadelphia	128	57	7	37	20	5	76	97
Pittsburgh	34	14	3	3	5	290	44	24
Reading	34	2	0	0	1	1	2	5
EAST NORTH CENTRAL								
Ohio:								
Cincinnati	12	7	3	0	7	0	0	17
Cleveland	95	23	5	70	6	814	72	22
Columbus	3	3	1	4	4	2	0	6
Toledo	11	4	1	1	1	43	3	4
Indiana:								
Fort Wayne	2	2	2	0	1	0	0	2
Indianapolis	14	3	1	0	1	12	48	10
South Bend	1	0	0	0	0	1	0	2
Terre Haute	0	0	1	0	0	0	0	2

City reports for week ended April 2, 1932—Continued

Division, State, and city	Chick- en pox, cases re- ported	Diphtheria		Influenza		Meas- les, cases re- ported	Mumps, cases re- ported	Pneu- monia, deaths, re- ported
		Cases, esti- mated expect- ancy	Cases reported	Cases reported	Deaths reported			
EAST NORTH CENTRAL—continued								
Illinois:								
Chicago.....	99	90	27	18	10	469	21	67
Springfield.....	12	1	0	5	0	0	10	7
Michigan:								
Detroit.....	49	40	6	15	7	217	24	33
Flint.....	7	2	1	21	0	159	76	4
Grand Rapids.....	13	0	0		3	109	27	4
Wisconsin:								
Kenosha.....	3	0	0		0	0	0	0
Madison.....	12	1	1			0	0	
Milwaukee.....	91	12	2	2	2	756	26	12
Racine.....	25	2	0		0	100	70	0
Superior.....	5	0	0		0	2	25	1
WEST NORTH CENTRAL								
Minnesota:								
Duluth.....	0	0	1		0	1	0	3
Minneapolis.....	11	11	3		2	4	35	14
St. Paul.....	3	4	0		0	3	27	3
Iowa:								
Davenport.....	8	0	1			0	0	
Des Moines.....	0	1	2			0	0	
Sioux City.....	2	1	0			1	0	
Waterloo.....	4	0	0			1	0	
Missouri:								
Kansas City.....	12	4	6		0	0	5	24
St. Joseph.....	1	0	4			0	0	2
St. Louis.....	29	31	15	5	2	3	6	13
North Dakota:								
Fargo.....	1	0	0		2	35	0	0
South Dakota:								
Aberdeen.....	0	0	1			19	0	
Sioux Falls.....	0	0	0			0	0	
Nebraska:								
Omaha.....	5	3	3		0	1	8	3
Kansas:								
Topeka.....	22	0	2	2	0	0	7	1
Wichita.....	10	1	2		0	161	0	3
SOUTH ATLANTIC								
Delaware:								
Wilmington.....	0	3	1		0	0	1	21
Maryland:								
Baltimore.....	103	19	3	25	7	2	81	31
Cumberland.....	0	0	0	2	1	3	0	2
Frederick.....	0	0	0	9	0	3	0	0
District of Columbia:								
Washington.....	39	12	9	3	4	3	0	21
Virginia:								
Lynchburg.....	22	1	0		0	1	0	3
Norfolk.....	16	1	3		0	0	0	2
Richmond.....	1	2	0		0	0	0	4
Roanoke.....	0	0	0		0	0	0	1
West Virginia:								
Charleston.....	2	1	2	4	0	73	0	3
Wheeling.....	1	0	0		0	4	1	2
North Carolina:								
Raleigh.....	5	0	0		0	20	0	3
Wilmington.....	0	0	0		0	0	0	2
Winston-Salem.....	20	0	1	6	0	2	3	3
South Carolina:								
Charleston.....	0	0	0	157	2	0	0	8
Columbia.....	0	0	0		0	0	0	0
Greenville.....	1	0	0		0	3	0	0
Georgia:								
Atlanta.....	3	2	1	21	1	9	1	10
Brunswick.....	3	0	0		0	0	0	0
Savannah.....	2	0	0	23	2	5	0	5

City reports for week ended April 2, 1932—Continued

Division, State, and city	Chick- en pox, cases re- ported	Diphtheria		Influenza		Meas- les, cases re- ported	Mumps, cases re- ported	Pneu- monia, deaths, re- ported
		Cases, esti- mated expect- ancy	Cases reported	Cases reported	Deaths reported			
SOUTH ATLANTIC— continued								
Florida:								
Miami.....	5	2	0	1	0	3	0	3
Tampa.....	3	1	2	2	3	0	0	1
EAST SOUTH CENTRAL								
Kentucky:								
Covington.....	0	0	0	1	0	0	0	2
Lexington.....	4	0	0	2	0	15	0	2
Tennessee:								
Memphis.....	9	3	0	3	3	0	1	17
Nashville.....	1	0	1	1	0	0	0	5
Alabama:								
Birmingham.....	5	2	0	21	1	1	10	4
Mobile.....	1	1	0	1	1	0	0	3
Montgomery.....	2	0	0	1	0	0	5	-----
WEST SOUTH CENTRAL								
Arkansas:								
Fort Smith.....	0	0	1	-----	0	0	0	-----
Little Rock.....	7	1	0	-----	3	0	4	5
Louisiana:								
New Orleans.....	0	11	26	9	0	0	0	5
Shreveport.....	2	0	0	-----	0	12	8	7
Oklahoma:								
Muskogee.....	1	-----	0	-----	0	5	2	0
Oklahoma City..	0	1	4	34	0	14	5	9
Texas:								
Dallas.....	7	5	13	8	8	51	3	9
Fort Worth.....	4	4	4	-----	2	1	0	9
Galveston.....	0	0	0	-----	0	0	0	2
Houston.....	3	4	7	-----	1	0	0	14
San Antonio.....	1	3	1	-----	0	0	0	9
MOUNTAIN								
Montana:								
Billings.....	1	0	0	-----	0	1	0	0
Great Falls.....	3	0	0	258	0	1	0	1
Helena.....	0	0	0	-----	0	2	0	0
Missoula.....	0	0	0	2	2	0	0	2
Idaho:								
Boise.....	0	0	0	-----	0	0	0	0
Colorado:								
Denver.....	23	7	1	-----	4	72	32	7
Pueblo.....	28	0	0	-----	1	0	0	3
New Mexico:								
Albuquerque.....	1	0	0	-----	0	32	10	0
Arizona:								
Phoenix.....	0	-----	0	-----	0	0	0	1
Utah:								
Salt Lake City..	39	2	1	-----	1	1	4	1
Nevada:								
Reno.....	0	0	0	-----	0	0	0	0
PACIFIC								
Washington:								
Seattle.....	25	2	0	-----	-----	341	7	-----
Spokane.....	12	1	0	-----	-----	0	0	-----
Tacoma.....	4	1	0	-----	0	42	0	5
Oregon:								
Portland.....	11	6	0	-----	2	78	6	7
Salem.....	7	0	0	1	-----	0	2	-----
California:								
Los Angeles.....	166	33	26	60	0	6	31	11
Sacramento.....	32	2	1	-----	0	66	1	9
San Francisco.....	89	12	3	1	1	208	5	13

City reports for week ended April 2, 1932—Continued

Division, State, and city	Scarlet fever		Smallpox			Tuber- culosis, deaths reported	Typhoid fever			Whoop- ing cough, cases reported	Deaths, all causes
	Cases, esti- mated expect- ancy	Cases re- ported	Cases, esti- mated expect- ancy	Cases re- ported	Deaths re- ported		Cases, esti- mated expect- ancy	Cases re- ported	Deaths re- ported		
WEST NORTH CENTRAL											
Minnesota:											
Duluth.....	8	3	0	0	0	3	0	0	0	0	26
Minneapolis.....	34	42	0	0	0	1	0	0	0	40	116
St. Paul.....	30	12	0	0	0	0	0	0	0	16	63
Iowa:											
Davenport.....	2	7	2	0	0	0	0	0	0	0	0
Des Moines.....	10	13	2	0	0	0	0	0	0	0	51
Sioux City.....	0	3	1	0	0	0	0	0	0	3	0
Waterloo.....	2	0	1	0	0	0	1	0	0	6	0
Missouri:											
Kansas City.....	25	21	1	0	0	7	0	0	0	39	141
St. Joseph.....	3	3	0	0	0	1	0	0	0	0	35
St. Louis.....	48	12	3	0	0	13	0	0	0	34	286
North Dakota:											
Fargo.....	1	3	0	0	0	0	0	0	0	0	6
South Dakota:											
Aberdeen.....	0	0	0	0	0	0	0	0	0	0	0
Sioux Falls.....	1	0	0	0	0	0	0	0	0	0	10
Nebraska:											
Omaha.....	5	6	4	1	0	3	0	0	0	7	66
Kansas:											
Topeka.....	4	2	1	0	0	0	0	0	0	42	23
Wichita.....	5	1	1	0	0	0	0	0	0	3	0
SOUTH ATLANTIC											
Delaware:											
Wilmington.....	6	15	0	0	0	0	0	0	0	4	57
Maryland:											
Baltimore.....	40	77	0	0	0	12	2	1	0	103	254
Cumberland.....	0	5	0	0	0	0	0	0	0	0	9
Frederick.....	0	0	0	0	0	0	0	0	0	2	5
District of Colum- bia:											
Washington.....	25	32	0	0	0	15	1	1	1	19	188
Virginia:											
Lynchburg.....	0	4	0	0	0	0	0	0	0	20	11
Norfolk.....	1	4	0	0	0	1	0	0	0	14	0
Richmond.....	4	3	0	0	0	5	0	0	0	0	39
Roanoke.....	1	6	0	0	0	0	0	0	0	2	15
West Virginia:											
Charleston.....	1	0	0	0	0	0	1	0	0	7	20
Wheeling.....	2	2	0	0	0	0	0	0	0	16	17
North Carolina:											
Raleigh.....	1	0	0	0	0	0	0	0	0	1	16
Wilmington.....	0	0	0	0	0	0	0	0	0	3	12
Winston-Salem.....	1	28	1	0	0	0	0	0	0	26	16
South Carolina:											
Charleston.....	0	1	0	0	0	3	1	0	0	0	30
Columbia.....	0	0	0	0	0	0	0	0	0	0	0
Greenville.....	0	3	0	0	0	0	0	0	0	1	0
Georgia:											
Atlanta.....	8	2	1	0	0	1	0	0	0	12	71
Brunswick.....	0	0	0	0	0	0	0	0	0	0	2
Savannah.....	1	0	0	0	0	2	1	2	0	0	41
Florida:											
Miami.....	1	0	0	0	0	1	0	1	0	0	26
Tampa.....	1	1	0	0	0	2	1	0	0	0	19
EAST SOUTH CENTRAL											
Kentucky:											
Covington.....	2	0	0	0	0	1	0	0	0	0	18
Lexington.....	0	0	0	0	0	2	0	0	0	6	14

City reports for week ended April 8, 1932—Continued

Division, State, and city	Scarlet fever		Smallpox			Tuber- culo- sis, deaths re- ported	Typhoid fever			Whoop- ing cough, cases re- ported	Deaths, all causes
	Cases, esti- mated expect- ancy	Cases re- ported	Cases, esti- mated expect- ancy	Cases re- ported	Deaths re- ported		Cases, esti- mated expect- ancy	Cases re- ported	Deaths re- ported		
EAST SOUTH CENTRAL—CON.											
Tennessee:											
Memphis.....	13	9	2	1	0	7	1	0	0	24	101
Nashville.....	3	3	0	0	0	5	0	0	0	0	46
Alabama:											
Birmingham...	4	0	1	0	9	4	1	0	0	7	60
Mobile.....	0	3	0	5	0	2	0	1	0	0	24
Montgomery...	0	1	0	0			0	0		0	
WEST SOUTH CENTRAL											
Arkansas:											
Fort Smith.....	0	0	0	0			0	0		1	
Little Rock.....	1	0	1	1	0	0	0	0	0	1	8
Louisiana:											
New Orleans...	11	7	1	0	0	10	2	0	0	0	138
Shreveport.....	1	1	1	0	0	2	0	0	1	4	34
Oklahoma:											
Muskogee.....		2		2				0		0	
Oklahoma City.....	5	8	2	2	0	1	0	0	0	8	38
Texas:											
Dallas.....	5	1	1	0	0	1	0	2	1	7	63
Fort Worth.....	2	3	6	3	0	2	0	1	0	0	39
Galveston.....	0	0	0	0	0	3	0	0	0	0	15
Houston.....	1	5	2	0	0	7	0	0	0	0	91
San Antonio.....	1	0	1	0	0	9	0	2	2	0	89
MOUNTAIN											
Montana:											
Billings.....	1	0	0	0	0	0	0	0	0	0	12
Great Falls.....	2	0	0	0	0	0	0	0	0	0	10
Helena.....	0	0	0	0	0	0	0	0	0	0	2
Missoula.....	0	1	0	0	0	0	0	0	0	0	10
Idaho:											
Boise.....	0	0	0	3	0	0	0	0	0	0	4
Colorado:											
Denver.....	15	11	0	0	0	6	0	0	0	15	80
Pueblo.....	1	0	0	0	0	0	0	0	0	1	10
New Mexico:											
Albuquerque...	0	2	0	0	0	4	0	0	0	0	9
Arizona:											
Phoenix.....	1	0	1	0	0	5	0	0	0	0	
Utah:											
Salt Lake City.....	2	3	0	0	0	1	0	0	1	5	25
Nevada:											
Reno.....	0	0	0	0	0	0	0	0	0	0	1
PACIFIC											
Washington:											
Seattle.....	9	5	3	1			0	3		8	
Spokane.....	6	1	3	0			0	0		1	
Tacoma.....	2	0	3	2	0	0	0	0	0	2	23
Oregon:											
Portland.....	5	5	9	6	0	1	0	0	0	3	75
Salem.....	0	0	0	0			0	0		1	
California:											
Los Angeles...	39	48	4	0	0	30	1	3	2	45	277
Sacramento.....	3	0	0	0	0	4	0	0	0	5	29
San Francisco...	23	10	1	4	0	8	1	3	1	6	155

City reports for week ended April 2, 1932—Continued

Division, State, and city	Meningo- coccus meningitis		Lethargic en- cephalitis		Pellagra		Polomyelitis (infan- tile paralysis)		
	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases esti- mated expect- ancy	Cases	Deaths
NEW ENGLAND									
Massachusetts:									
Boston.....	1	1	0	0	0	0	0	0	0
MIDDLE ATLANTIC									
New York:									
Buffalo.....	0	3	0	0	0	0	0	0	0
New York.....	8	6	0	0	0	0	0	0	0
Rochester.....	0	0	1	0	0	0	0	0	0
New Jersey:									
Trenton.....	1	1	1	1	0	0	0	0	0
Pennsylvania:									
Philadelphia.....	6	5	3	3	0	0	0	1	0
Pittsburgh.....	4	1	0	0	0	0	0	0	0
EAST NORTH CENTRAL									
Ohio:									
Cleveland.....	4	1	0	0	0	0	0	1	0
Indiana:									
Indianapolis.....	8	2	0	0	0	0	0	0	0
Illinois:									
Chicago.....	5	4	1	0	0	0	0	0	0
Michigan:									
Detroit.....	2	3	0	0	0	0	0	0	0
Grand Rapids.....	1	1	0	0	0	0	0	0	0
Wisconsin:									
Madison.....	0	0	0	0	0	0	0	1	0
Racine.....	0	1	0	0	0	0	0	0	0
WEST NORTH CENTRAL									
Minnesota:									
Minneapolis.....	1	0	0	0	0	0	0	0	0
St. Paul.....	1	0	0	0	0	0	0	0	0
Iowa:									
Des Moines.....	1	0	0	0	0	0	0	0	0
Missouri:									
St. Louis.....	1	1	0	0	0	0	0	0	0
Nebraska:									
Omaha.....	0	1	0	0	0	0	0	0	0
Kansas:									
Wichita.....	1	0	0	0	0	0	0	0	0
SOUTH ATLANTIC ¹									
District of Columbia:									
Washington.....	2	0	0	0	0	0	1	0	0
West Virginia:									
Charleston.....	1	1	0	0	0	0	0	0	0
Wheeling.....	0	0	0	0	0	0	0	1	0
North Carolina:									
Raleigh.....	0	0	0	0	1	0	0	0	0
Winston-Salem.....	0	0	0	0	2	0	0	0	0
South Carolina:									
Charleston ²	0	0	0	0	3	0	0	0	0
Georgia: ³									
Savannah ¹	0	0	0	0	4	1	0	0	0
EAST SOUTH CENTRAL									
Tennessee:									
Memphis.....	0	1	0	1	0	0	0	0	0
Alabama:									
Montgomery.....	0	0	0	0	1	0	0	0	0

See footnote at end of table.

City reports for week ended April 2, 1932—Continued

Division, State, and city	Meningococcus meningitis		Lethargic encephalitis		Pellagra		Polio-myelitis (infantile paralysis)		
	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases estimated expectancy	Cases	Deaths
WEST SOUTH CENTRAL									
Louisiana:									
New Orleans.....	0	0	0	0	1	1	0	0	0
Oklahoma:									
Oklahoma City.....	0	0	0	0	0	1	0	0	0
Texas:									
Dallas.....	0	0	0	0	1	1	0	0	0
Fort Worth.....	0	0	0	0	0	1	0	0	0
San Antonio.....	1	0	0	0	0	0	0	0	0
MOUNTAIN									
Montana:									
Great Falls.....	1	0	0	0	0	0	0	0	0
PACIFIC									
California:									
Los Angeles.....	2	0	0	0	0	0	0	0	0
San Francisco.....	1	1	0	0	0	0	0	0	0

¹ Delayed report.

² Dengue, 2 cases at Charleston, S. C.

³ Typhus fever, 3 cases: 1 case at Atlanta, Ga.; 1 case at Savannah, Ga.; and 1 case at Tampa, Fla.

The following table gives the rates per 100,000 population for 98 cities for the 5-week period ended April 2, 1932, compared with those for a like period ended April 4, 1931. The population figures used in computing the rates are estimated mid-year populations for 1931 and 1932, respectively, derived from the 1930 census. The 98 cities reporting cases have an estimated aggregate population of more than 34,000,000. The 91 cities reporting deaths have more than 32,400,000 estimated population.

Summary of weekly reports from cities, February 23 to April 2, 1932—Annual rates per 100,000 population, compared with rates for the corresponding period of 1931¹

DIPHTHERIA CASE RATES

	Week ended—									
	Mar. 5, 1932	Mar. 7, 1931	Mar. 12, 1932	Mar. 14, 1931	Mar. 19, 1932	Mar. 21, 1931	Mar. 26, 1932	Mar. 28, 1931	Apr. 2, 1932	Apr. 4, 1931
98 cities	62	73	59	65	62	65	52	78	47	53
New England.....	48	106	53	79	65	67	65	70	43	46
Middle Atlantic.....	63	61	56	67	54	64	56	63	44	48
East North Central.....	66	75	54	72	48	72	31	82	29	64
West North Central.....	49	71	74	63	95	73	55	163	78	42
South Atlantic.....	78	93	59	53	49	73	60	61	37	47
East South Central.....	35	29	46	35	12	23	6	76	6	29
West South Central.....	102	118	135	68	162	71	112	64	158	85
Mountain.....	9	61	26	26	43	17	9	87	17	44
Pacific.....	57	63	44	55	89	51	70	69	57	53

MEASLES CASE RATES

98 cities	698	769	171	947	732	1,041	727	1,208	851	1,122
New England.....	1,740	909	901	1,346	860	1,527	599	1,479	863	1,106
Middle Atlantic.....	504	874	644	1,026	578	1,158	598	1,321	621	1,250
East North Central.....	919	369	938	582	1,167	558	1,203	722	1,573	726
West North Central.....	241	643	165	595	316	492	186	651	398	532
South Atlantic.....	424	2,241	286	2,758	302	3,448	232	3,885	245	3,814
East South Central.....	17	1,045	58	1,157	23	1,004	19	1,650	6	1,515
West South Central.....	257	68	99	37	40	51	158	47	208	88
Mountain.....	198	1,321	509	1,462	358	1,288	603	1,140	664	661
Pacific.....	1,313	347	1,205	357	1,443	394	1,440	519	1,262	359

SCARLET FEVER CASE RATES

98 cities	475	345	481	375	488	389	478	403	414	371
New England.....	666	527	709	589	724	676	731	697	744	577
Middle Atlantic.....	777	359	799	389	786	392	755	454	632	404
East North Central.....	382	346	382	399	394	395	397	378	345	377
West North Central.....	231	492	178	518	195	589	197	560	206	585
South Atlantic.....	312	354	327	311	371	342	382	311	345	291
East South Central.....	87	406	81	482	110	487	100	564	92	399
West South Central.....	66	71	79	95	89	102	49	78	46	95
Mountain.....	155	306	172	400	215	305	233	209	129	157
Pacific.....	158	122	135	96	147	110	133	104	122	92

SMALLPOX CASE RATES

98 cities	4	13	5	19	5	22	4	17	4	14
New England.....	10	0	0	0	0	0	0	0	3	0
Middle Atlantic.....	0	0	0	0	0	0	0	0	0	0
East North Central.....	7	15	5	9	4	8	2	7	4	9
West North Central.....	6	57	11	132	17	130	17	89	2	78
South Atlantic.....	6	0	0	0	0	0	0	4	0	2
East South Central.....	17	23	46	0	12	12	38	12	35	12
West South Central.....	7	47	0	61	13	95	0	78	3	71
Mountain.....	0	17	17	17	17	9	0	44	26	0
Pacific.....	4	12	13	41	11	43	15	22	13	16

See footnotes at end of table.

Summary of weekly reports from cities, February 28 to April 2, 1932—Annual rates per 100,000 population, compared with rates for the corresponding period of 1931—Continued

TYPHOID FEVER CASE RATES

	Week ended—									
	Mar. 5, 1932	Mar. 7, 1931	Mar. 12, 1932	Mar. 14, 1931	Mar. 19, 1932	Mar. 21, 1931	Mar. 26, 1932	Mar. 28, 1931	Apr. 2, 1932	Apr. 4, 1931
98 cities.....	6	4	5	3	4	4	5	4	5	4
New England.....	5	5	0	0	2	2	5	2	0	2
Middle Atlantic.....	4	3	3	2	1	2	3	2	3	3
East North Central.....	6	1	1	2	2	2	3	2	4	2
West North Central.....	0	11	2	0	2	8	4	2	2	4
South Atlantic.....	20	12	25	6	2	16	12	12	8	14
East South Central.....	17	18	6	18	29	0	19	0	6	0
West South Central.....	16	0	10	14	23	10	20	7	13	10
Mountain.....	0	0	9	0	17	0	9	0	0	9
Pacific.....	0	2	8	4	2	8	6	10	17	2

INFLUENZA DEATH RATES

	37	44	37	34	37	32	36	29	29	23
91 cities.....	37	44	37	34	37	32	36	29	29	23
New England.....	17	19	19	36	10	19	17	14	19	2
Middle Atlantic.....	42	32	47	23	39	23	36	20	34	17
East North Central.....	41	48	39	28	40	28	41	25	24	18
West North Central.....	32	59	15	50	32	47	23	35	17	12
South Atlantic.....	33	73	39	57	49	49	36	32	39	40
East South Central.....	83	140	25	102	50	115	44	127	55	127
West South Central.....	71	52	37	55	61	35	84	55	40	69
Mountain.....	34	44	26	35	43	35	43	61	69	26
Pacific.....	12	34	7	36	12	34	6	41	2	14

PNEUMONIA DEATH RATES

	189	194	193	191	188	184	193	180	167	171
91 cities.....	189	194	193	191	188	184	193	180	167	171
New England.....	192	185	194	147	156	183	225	156	162	127
Middle Atlantic.....	221	229	250	214	238	216	243	220	203	223
East North Central.....	158	154	131	139	133	132	119	125	113	120
West North Central.....	241	218	215	159	192	215	239	178	204	150
South Atlantic.....	196	265	224	332	233	269	272	263	235	222
East South Central.....	169	229	182	242	201	210	201	191	194	172
West South Central.....	172	149	148	211	205	180	199	211	172	238
Mountain.....	196	131	207	236	232	122	138	131	121	157
Pacific.....	102	101	118	125	93	101	72	98	88	53

¹ The figures given in this table are rates for 100,000 population, annual basis, and not the number of cases reported. Populations used are estimated as of July 1, 1932 and 1931, respectively.

² Columbia, S. C., and Montgomery, Ala., not included.

³ Pawtucket, R. I., and Hartford, Conn., not included.

⁴ Columbia, S. C., not included.

⁵ Montgomery, Ala., not included.

FOREIGN AND INSULAR

INFLUENZA IN EUROPE

*England and Wales.*¹—The number of deaths from influenza reported in 117 great towns in England and Wales, including London, fell from 292 during the week ended March 12, 1932, to 117 during the following week. The number of cases of acute primary pneumonia and acute influenzal pneumonia reported in England and Wales was 2,074 during the week ended March 5; 1,924 during the following week; and 1,718 during the week ended March 19.

Germany.—The accompanying table gives the number of deaths from influenza reported in 50 great towns of Germany during the 3 weeks ended March 5, 1932. The corresponding general mortality rates are also given.

Week ended—	Number of deaths from influenza	General death rate per 1,000 population
Feb. 20, 1932.....	50	11.4
Feb. 27.....	65	11.5
Mar. 5.....	76	11.4

*Switzerland.*¹—The number of cases of influenza reported in Switzerland fell from 6,420 to 4,221 during the week ended March 19, 1932. In districts of over 10,000 population, 85 deaths from influenza were reported during the week ended March 12, as compared with 55 during the preceding week.

CANADA

Provinces—Communicable diseases—Week ended March 26, 1932.—The Department of Pensions and National Health of Canada reports cases of certain communicable diseases for the week ended March 26, 1932, as follows:

Province	Cerebro-spinal fever	Influenza	Lethargic encephalitis	Polio-myelitis	Smallpox	Typhoid fever
Prince Edward Island *.....						
Nova Scotia.....		22				2
New Brunswick.....						13
Quebec.....				3		2
Ontario.....	3	183	2		1	1
Manitoba.....					1	
Saskatchewan *.....						1
Alberta.....						
British Columbia.....					2	
Total.....	3	205	2	3	3	24

* No case of any disease included in the table was reported during the week.

¹ See also PUBLIC HEALTH REPORTS, vol. 47, No. 15, April 8, 1932, p. 863.

Quebec Province—Communicable diseases—Week ended March 26, 1932.—The Bureau of Health of the Province of Quebec, Canada, reports cases of certain communicable diseases for the week ended March 26, 1932, as follows:

Diseases	Cases	Disease	Cases
Chicken pox.....	76	Poliomyelitis.....	3
Diphtheria.....	26	Puerperal fever.....	1
Erysipelas.....	6	Scarlet fever.....	135
German measles.....	4	Tuberculosis, pulmonary.....	95
Measles.....	260	Typhoid fever.....	18
Ophthalmia neonatorum.....	2	Whooping cough.....	23

CHINA

Meningitis.—According to recent information, cerebrospinal meningitis has been reported in Hong Kong, Canton, and Macao, China, as follows:

	Cases	Deaths
Hong Kong:		
Two weeks ended Mar. 19, 1932.....	5	2
Week ended Mar. 26.....	3	1
Week ended Apr. 2.....	13	1
Canton:		
Week ended Mar. 5, 1932.....		1
Week ended Mar. 12.....	12	1
Week ended Mar. 19.....	7	3
Week ended Mar. 26.....	11	2
Week ended Apr. 2.....	14	1
Macao:		
Two weeks ended Mar. 5, 1932.....	34	10
Week ended Mar. 19.....	82	45
Week ended Mar. 26.....	94	33
Week ended Apr. 2.....	115	94

EGYPT

*Cerebrospinal meningitis.*¹—The number of cases of cerebrospinal meningitis, with deaths, reported in Egypt during the month of February, 1932, is given in the accompanying table. During the week ended March 3, 1932, there was a decrease in the number of cases reported, but the deaths numbered 200. During the first four weeks of the year, 125 of the 196 cases reported occurred in Cairo, and most of the remaining cases occurred in the provinces of Lower Egypt. Since the seasonal maximum is usually reached in April, it was thought unlikely that any further increase in the disease would take place.

Week ended—	Cases	Deaths
Feb. 4, 1932.....	77	24
Feb. 11.....	104	37
Feb. 18.....	252	89
Feb. 25.....	394	162

¹ See also PUBLIC HEALTH REPORTS, vol. 47, No. 15, April 8, 1932, p. 865.

JAMAICA

Communicable diseases—Four weeks ended March 26, 1932.—During the four weeks ended March 26, 1932, cases of certain communicable diseases were reported in Kingston, Jamaica, and in the island of Jamaica outside of Kingston, as follows:

Disease	Kingston	Other localities	Disease	Kingston	Other localities
Cerebrospinal meningitis.....		1	Paratyphoid fever.....		2
Chicken pox.....	24	30	Poliomyelitis.....	1	
Dysentery.....	1	3	Puerperal fever.....		2
Erysipelas.....	1		Tuberculosis.....	42	71
Leprosy.....	1		Typhoid fever.....	8	52

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

PLAGUE

[C indicates cases; D, deaths; P, present]

Place	Sept. 20- Oct. 17, 1931	Oct. 18- Nov. 14, 1931	Nov. 15- Dec. 12, 1931	Week ended—													
				December, 1931					January, 1932					February, 1932		March, 1932	
				19	26	2	9	16	23	30	6	13	20	27	5	12	19
Argentina: Cordoba Province ¹																	
Azores:																	
San Miguel Island.....			5														
Tercera Island.....			1														
Tercera Island.....			16														
Tercera Island.....			6														
Belgian Congo.....																	
British East Africa (see also table below):																	
Tanganyika.....	13				10												
Uganda.....	5				10												
Uganda.....	276	218	145	9	13					14	10	7	7	1			
Uganda.....	270	211	138	24	15	10	13			14	6	6	5	1			
Canary Islands: Palma Island—Los Lanos.....										8							
Canary Islands: Palma Island—Los Lanos.....										5	3						
Ceylon: Colombo.....	4		1		4					1		1		2	1	1	
Ceylon: Colombo.....	3		1		1					1		1		1	1	1	
Ceylon: Colombo.....			1		1												
Chile: Santiago.....																	
Chile: Santiago.....																	
Chile: Santiago.....																	
China:																	
Plague-infected rats.....																	
Plague-infected rats.....																	
Kwang Chow Wan.....																	
Kwang Chow Wan.....																	
Kwang Chow Wan.....																	
Shansi Province ¹																	
Shansi Province.....																	
Shansi Province.....																	
Dutch East Indies:																	
Java—																	
Surabaya.....										1	1						
Surabaya.....										1	1						
Java and Madura.....	325	512	702	179	151	136	121	102	127	126	144	118	144	118	144	144	
Java and Madura.....	113	139	198	64	54	36	46	34	34	48	54	65	60	60	60	60	
West Java.....	113	139	198	64	54	36	46	34	34	48	54	64	56	56	56	56	

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

PLAGUE—Continued

[C indicates cases; D, deaths; P, present]

Place	Sept. 24 17 1931	Oct. 18 14 1931	Nov. 15 12 1931	Week ended—													
				December, 1931			January, 1932			February, 1932			March, 1932				
				19	26	3	9	16	23	30	6	13	20	27	5	12	19
Indo-China (see table below).																	
Iraq:																	
Baghdad.....		2	7	1					1	2	1	1	1				
.....		2	2	2													
Mandhan.....		3	1	1													
Madagascar (see also table below):																	
Tamatave.....	1	1	2	1													
Morocco.....	18	2	11														
.....	8	6															
Peru (see table below).																	
Senegal (see table below).																	
Siam.....	4	5	5	1					P	1					1	4	1
.....	3	2	2	1												1	2
Spain, Hospital—Barcelona Province.....	2	7															
Syria: Beirut.....	1	1															
Tunisia: Tunis.....	3	1															
Union of South Africa: Orange Free State.....	1								P								
.....	P									P						2	

Place	September, 1931	October, 1931	November, 1931	December, 1931	January, 1932	February, 1932	March, 1932
British East Africa (see also table above): Kenya.....	C 14	64	44	41	17	33
Ecuador:							
Provinces—							
Chimborazo.....	C 13	2	8	8	13
Loja.....	C 4	11	2	11
Loja.....	C 4	4	3	16	1	12
Indo-China.....	D 4	1	9	9	1	6
Madagascar (see also table above):							
Ambohitra Province.....	C 1	8	39	142	166
Antsirabe Province.....	D 1	5	37	121	152
Maevatanana Province.....	D 19	17	27	56	53
Antsirabe Province.....	D 19	17	27	51	51
Maevatanana Province.....	D 4	4
Miarinarivo Province.....	C 14	18	10	14	15
Moramanga Province.....	D 12	16	6	14	15
Moramanga Province.....	C 12	13	25	30	13
Tananarive Province.....	D 11	11	25	29	13
Tananarive Province.....	C 65	120	146	245	263
Tananarive Province.....	D 63	117	178	241	196
Tananarive Province.....	C 2	8	27	21	11
Tananarive Province.....	D 2	7	11	9
Peru.....							
Departments—							
Cajamarca.....	C 12	7	16	1	2
Cajamarca.....	D 8	8	6	7	1
Cajamarca.....	C 13	6	2
Dakar ¹	D 8	2
Dakar ¹	C 46	4
Dakar ¹	D 31	4
Dourbel ¹	C 13
Dourbel ¹	D 6
Longa ¹	C 10	1
Longa ¹	D 4	2
Rufisque ¹	C 1	7	12
Rufisque ¹	D 1	1	2
Thies ¹	C 12	7	16	1
Thies ¹	D 8	6	7
Yombel ¹	C 1
Yombel ¹	D 7
Yombel ¹	C 1

¹ Reports incomplete.

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

YELLOW FEVER—Continued

[C indicates cases; D, deaths; P, present]

Place	Week ended—												
	January, 1932			February, 1932			March, 1932			April, 1932			
	16	23	30	6	13	20	27	5	12	19	26	2	9
Gold Coast:													
Avudua.....													
Cape Coast.....	C												
Dagomba District.....	C												
Kete Krachi.....	C	1											
Salaga.....	C												
Tamale.....	D												
Yapei.....	D	2											
Ivory Coast: Tehini.....	C												
Nigeria.....	C												
Senegal:	D												
St. Louis.....	C	1											
Tibes.....	C	1											
Sudan (French): Macina—Kayo Circle.....	D	1											
Togo (French): Atakpame—Anle Circle.....	D	2											
Upper Volta:	D	1											
Banfora.....	D	1											
Dedougou.....	C	2											
Diabakoko.....	C	1											
Ouagadougou.....	C	2											
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