# **Public Health Reports**

Vol. 55 • JULY 26, 1940 • No. 30

# PROTECTIVE OINTMENT FOR THE PREVENTION OF POISON IVY DERMATITIS<sup>1</sup>

By LOUIS SCHWARTZ, Medical Director, LEON H. WARREN, Acting Assistant Surgeon, and FREDERICK H. GOLDMAN, Associate Chemist, United States Public Health Service

Plants are one of the most frequent causes of contact dermatitis. In a compilation of 9,116 cases of occupational dermatitis reported to various State compensation boards, 10.7 percent were caused by plants. Rhus poisoning constitutes by far the largest percentage of these plant dermatoses. Field workers, such as farmers, horticulturists, gardeners, Civilian Conservation Corps workers, and men engaged in clearing land are the ones most often affected.

Many queries are received by the Public Health Service as to what means of protection should be used to prevent the occurrence of this form of dermatitis among field workers. While there are many efficient methods for the treatment of rhus poisoning, methods for its prevention are few. Desensitizing injections of ascending doses of rhus toxin have proven beneficial in some cases. These inoculations are more or less impracticable in field workers because they must be instituted some time before work in the field is begun, and because the labor turn-over would make it necessary to be continually desensitizing new men and render valueless, as far as the work is concerned, the injections given to the men who leave. Besides, careful medical supervision is necessary since the injections are sometimes attended with untoward reactions.

Various methods of prevention have been recommended: (1) Bathing the exposed parts with a strong solution of potassium permanganate; the objection to this method is that the stain is difficult to remove. (2) Applying to the exposed parts a 3-5 percent solution of ferric chloride in equal parts of glycerin and water; the objection to this is that persistent pigmentation of the skin may result from this procedure.

<sup>&</sup>lt;sup>1</sup> From the Office of Dermatoses Investigations, National Institute of Health. Paper delivered at the Third Annual Conference of Governmental Hygienists at the National Institute of Health, Bethesda, Md., May 1, 1940.

The active principle of rhus is urushiol, a mixture of o-dihydroxybenzenes with a normal 15 carbon atom side chain in position 3.

It occurred to one of us that if there could be found a chemical which would rapidly decompose or split up this complex radical, its irritating properties might be destroyed. Such a chemical, in order to be of use in the prevention of dermatitis, must itself be nonirritant. It has been known that potassium permanganate (a powerful oxidizing agent) will render urushiol harmless, but, as stated before, potassium permanganate discolors the skin. There are, however, a number of powerful, nonirritant, nonstaining oxidizing agents, such as sodium perborate, potassium chlorate, potassium periodate, and zinc peroxide.

We decided to test the detoxicant action of these oxidizing agents upon urushiol. For this purpose an extract of poison ivy, prepared in the following manner, was obtained from Lederle Laboratories.

Six pounds of poison ivy leaf were extracted with 12 quarts of acetone and between 11 and 12 liters of extract were obtained. Included in this extract was water from the green leaves. Not more than 5 percent of extract was lost in the process. Any loss of activity of the extracted irritant was avoided by never allowing the extract to dry through complete evaporation of the acetone. A small quantity of inert substance remaining in the acetone extract was insoluble in vegetable oils and was removed by warm filtration through a Berkfeld filter. This fraction was also insoluble in mineral oil. The original 3,000 gm. of leaf in 12,000 cc. represented a 1:4 dilution. This was evaporated down to 100 cc. (120 times as concentrated). Therefore, the extract was 30 times as concentrated as the leaf. One hundred cc. of extract contained 13.5 gm. of resin, and 1 drop contained 0.08 gm. of resin.

One drop of a mixture consisting of equal parts of a saturated aqueous solution of potassium chlorate and the acetone extract of the poison ivy resin was applied to the shorn abdomen of a guinea pig. Seventy-two hours later, circumscribed erythematous patches corresponding to the original spread of the resin mixture were observed on the abdomen of the guinea pig. Seventy-two hours later the adherent crust was removed and tested for solubility in acetone. Since it was insoluble in the latter, it is believed to have been desquamated skin rather than a film of the resin.

One drop of a mixture consisting of equal parts of a saturated aqueous solution of sodium perborate and the acetone extract of the poison ivy resin was applied to the shorn abdomen of another guinea pig, with reactions similar to the above. Results of this experiment seemed to indicate no perceptible difference in degree of detoxicant action of these two chemicals upon urushiol contained in the resin. The apparent failure of these oxidizing agents to exert a detoxicant action upon urushiol may have been due to the fact that there was not sufficient oxygen liberated from either of the two solutions to oxidize completely the amount of resin in the mixture tested. It is also possible that the 50-percent acetone solution caused the dermatitis. As the risk from dermatitis was not too hazardous, it was decided to experiment upon human beings.

A portion of the extract of the resin of poison ivy in acetone (containing 13 percent of the resin by weight) was mixed with equal parts of water (by volume). One drop of this mixture was placed on the flexor surface of the arm of one of the authors and allowed to evaporate, leaving a brown stain of resin on the skin. Forty hours later an erythematous macule ¼ inch in diameter appeared at the site of application.

A second portion of the ivy extract was mixed with equal parts of a saturated aqueous solution of sodium perborate. One drop of this mixture was placed on the flexor surface of the upper part of the forearm of one of us and allowed to evaporate, leaving a brown stain of resin on the skin. At the end of 72 hours there was no skin reaction at this site.

A third portion of the ivy extract was mixed with a saturated aqueous solution of potassium chlorate. One drop of this mixture was placed on the flexor surface of the lower part of the forearm of one of us and allowed to evaporate, leaving a brown stain of the resin on the skin. At the end of 48 hours there appeared an erythematous macule ½ inch in diameter at the above site. The first and second tests above were repeated on the flexor surface of the arms of 8 volunteers, using 1 drop of the mixture of ivy extract and equal parts of saturated aqueous solution of sodium perborate, with a control test of 1 drop of the mixture of ivy extract and equal parts of water.

One of us was not susceptible to poison ivy, and failed to react to either the detoxified or control tests. Two of us developed a spreading ervthema around the site of the control test within 24 hours, but no reaction to the detoxified resin. However, 8 and 9 days later, respectively, the two latter subjects developed an exacerbation of dermatitis at the site of the control test, together with a delayed reaction at the site of the previously negative tests to the detoxified The reactions covered a considerable portion of the arms and resin. became vesicular. They were flare-ups due to sensitization by application of the original unneutralized resin, together with the activation of a previously negative skin test site. Another subject was only slightly susceptible to the unneutralized resin and not at all to the neutralized resin. The remaining 5 subjects developed skin reactions of varying severity to the unneutralized resin and in each case markedly less reaction to the neutralized resin. One case developed a severe dermatitis with marked edema of the entire arm and forearm (fig. 1).

This experiment showed that the solution of sodium perborate had

some destructive action on the toxicity of urushiol, but that it did not completely detoxify it. This was thought to be due to the small amount of sodium perborate in the saturated solution. Since equal parts by volume of a saturated solution of sodium perborate did not liberate sufficient oxygen to neutralize completely the poison in the extract, we thought that if we incorporated the solid perborate in a vanishing cream and rubbed the vanishing cream on the skin, then the moisture from the skin would continue to liberate sufficient oxygen from the powdered sodium perborate to neutralize the action of whatever urushiol might come in contact with the skin. Moreover, the vanishing cream when rubbed into the skin would fill the pores and form a protective covering and prevent much of the poison from penetrating the skin. When the vanishing cream containing sodium perborate is rubbed into the skin, some of the sodium perborate remains on the surface of the skin and may oxidize whatever poison ivy comes in contact with it. Moreover, as the perspiration comes in contact with the vanishing cream in the pores of the skin, a soan is formed and the alkalinity of the soap liberates oxygen from the perborate while the soapy solution washes the poison ivv out of the skin from within outwards.

The protective action of such a vanishing cream containing 10 percent sodium perborate was tested on 9 volunteers. The protective cream was rubbed into the skin and on it was placed 1 drop of a solution of Lederle extract diluted with from equal parts to 1-10 parts of olive oil. This strength is from 3 to 15 times the concentration of the toxin contained in the fresh leaf.

The details of this experiment are as follows: A vanishing cream consisting of stearic acid (triple pressed) 200 gm., potassium hydroxide (sticks) 14 gm., water 800 cc., alcohol (90 percent) 40 cc., was compounded. To 50 gm. of the above cream, 5 gm. of sodium perborate were added. The resultant protective oxidizing cream was granular and did not rub well into the skin. A white deposit, probably of sodium perborate, was left on the surface of the skin.

A vial containing a mixture of equal parts of the acetone extract of the ivy resin and water was stoppered, shaken well, and the tip of the stopper touched to an area of skin of the forearm which had been previously coated with a film of the protective cream. It was noted that the acetone in the mixture dissolved the film of cream on the skin at the point of contact with the stopper. The application was allowed to dry. Two hours later the site was washed thoroughly with soap and water. Seventy-two hours later a vesicular skin reaction  $\frac{1}{2}$  inch in diameter developed at the site of application of the ivy resin. This corresponded to the area over which the resin was applied and where the cream had been dissolved by the acetone. There was



FIGURE 1.—Three subjects tested with extract of poison ivy mixed with equal parts of a solution of sodium perborate. The left-hand subject shows no reaction. The middle subject shows sovere reaction on the right arm and less severe on the left arm. The subject at the right shows a spreading reaction on the upper arm.

Public Health Reports, Vol. 55, No. 30, July 26, 1940



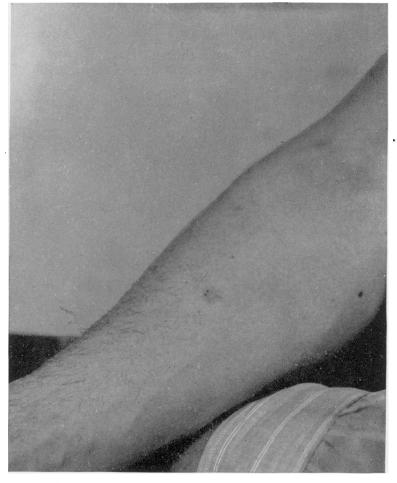


FIGURE 2.—Reaction at site of application of poison ivy extract applied over 10 percent sodium perborate in vanishing cream on a person highly susceptible to poison ivy.

no spread of the erythema beyond this area as there had been when no cream was applied.

In order to eliminate the solvent effect of the acetone on the cream, it was decided to dissolve the resin in mineral oil. Three drops of the acetone solution of the toxin were allowed to evaporate to dryness on a watch crystal and the remaining resin was dissolved in three drops of mineral oil. One drop of the solution was applied to the skin previously smeared with the cream. An erythema about  $\frac{1}{10}$  inch in diameter developed at the end of 72 hours localized to the site of the application of the solution (fig. 2).

A vanishing cream containing 10 percent sodium perborate was then tested for its protective action against ivy resin on the original 9 volunteers. Three drops of acetone extract of the resin were allowed to evaporate and the resin dissolved in 6 drops of olive oil. (This corresponds to 15 times the concentration of the toxin in the fresh leaf.) One drop of this solution was applied to an area of skin protected by the oxidant vanishing cream on 2 subjects and allowed to remain for 5 minutes, the excess removed by blotting, and the cream removed by washing with soap and water at the end of 4 hours. An erythema limited to site of the application of the drop of the solution developed at the end of 24 and 48 hours, respectively.

In three cases the resin was applied in the form of one drop of a mixture of equal parts of the acetone extract and water. This was washed off after 4 hours with soap and water. Only one of these developed a vesicular reaction limited to the site of the application of the drop of the solution.

In the remaining 4 cases, 3 drops of acetone extract of the resin were allowed to evaporate, the resin dissolved in 30 drops of olive oil, and 1 drop of this solution (three times the concentration of the irritant that is in the fresh leaf) allowed to remain for 2 minutes on the area protected by the oxidant vanishing cream. It was removed at the end of 4 hours by washing with soap and water. Only one of these developed any reaction and he had only an erythema about one-fourth inch in diameter. He was the subject who developed an edema and vesiculation of the entire arm when the ivy extract was applied to the unprotected skin.

On one of us the application of one drop of the solution of resin in one drop of olive oil was repeated at the end of 1 hour and again at the end of 2 hours to the area of skin originally protected by application of the oxidant vanishing cream, without renewal of the cream. When 4 hours had elapsed after the first of these three applications, the arm was washed with soap and water. No reactions resulted from this experiment.

In none of the subjects was there any spread of the skin reaction beyond the actual site of the application of the poison. Since six out of nine subjects developed no reaction when the toxin was applied over the protective cream, and since the person who reacted most severely to the application of the Lederle extract without the protective ointment developed only a slight erythema about one-fourth inch in diameter at the site of application of the toxin when the protective ointment was used, it is concluded that the vanishing cream containing perborate gives considerable protection against poison ivy dermatitis.

In order to decide whether both the alkali and the oxidizing agents are of value in this ointment, a portion of the Lederle extract was treated with 20 percent sodium hydrate and when all signs of reaction were over it was neutralized with acetic acid so that the alkali would not burn the skin. A drop of the solution was then applied to the skin and at the end of 3 days an area of erythema and vesicles about 1 inch long and one-half inch wide developed. This showed that urushiol cannot be inactivated by alkali alone.

Another portion of the Lederle extract was treated with a saturated solution of potassium periodate (about 2 percent) and applied as a skin test. There was no reaction to this, showing that oxidation inactivates urushiol. The crystals of potassium periodate were moistened and applied as a skin test. No reactions developed at this site, showing that potassium periodate itself is not a skin irritant. We know from previous skin tests that sodium perborate and zinc peroxide are not skin irritants. This experiment showed that it was the oxidant and not the alkali in the vanishing cream which inactivated the irritant principle of poison ivy.

In order to test the value of the protective cream against growing poison ivy, the hands and the right forearm of two of the susceptible subjects (one of whom was the most susceptible of all tested), were smeared with the protective ointment. Both subjects then plucked poison ivy leaves as they were found growing around a tree (figs. 3 and 4). In addition to this, the leaves were handled and pressed against the cream-protected areas of the forearms of both subjects, brushed up and down (with the other protected hand) and allowed to remain one-half hour on one subject, and several minutes on the other (figs. 5 and 6). One hour later the protective cream was washed off with water and no reactions followed.

That the more susceptible of the two subjects was still sensitive to poison ivy was shown by the fact that at the time the above experiment was performed he had a linear vesicular eruption on one of his arms from accidental contact with poison ivy. In order to verify the fact that the poison ivy leaves used in this experiment contained the active toxin and that the less susceptible subject was still sensitive, the following experiment was performed:

This subject applied some of the poison ivy leaves from the same



FIGURE 3.—Picking poison ivy after applying protective cream.



FIGURE 4.—Picking poison ivy after applying protective cream.



 $\label{eq:Figure 5.-Applying freshly picked poison ivy to the forearm of a susceptible subject covered with protective cream.$ 



FIGURE 6.—Applying freshly picked poison ivy to forearm of susceptible subject covered with protective cream.

plant shown in figures 3 and 4 to an unprotected portion of his left forearm. Thirty-six hours later there was considerable pruritus. At the end of 48 hours there was an area of erythema about 1½ inches long and 1 inch wide covered with minute vesicles at the site of the application of the fresh poison ivy leaf. This experiment proved that the ivy leaves contained the toxin and that the less susceptible of the two subjects still retained his sensitivity.

#### CONCLUSIONS

1. An alkaline vanishing cream containing a nonirritant oxidizing agent, such as sodium perborate or potassium periodate is an effective preventive against poison ivy dermatitis.

2. It should be well rubbed into the skin of the arms and face of workers before exposure to poison ivy. This procedure leaves a deposit of the powdered oxidant on the skin.

3. The protective cream should be allowed to remain on until the noon hour when it should be removed by washing with soap and water; this will emulsify the vanishing cream in the pores of the skin and wash away from within outward whatever toxin may be in the pores or on the skin.

4. The cream should be reapplied again after the lunch hour and again washed off in the evening when work is over.

5. This vanishing cream should be freshly prepared at least once in 2 weeks to avoid deterioration. However, the cream used in our experiments was slightly discolored but still active after 1 month.

#### ACKNOWLEDGMENTS

We wish to acknowledge gratefully the cooperation of Drs. Sayers, Neal, Castberg, Dreessen, Mr. Reinhart, and Mr. Schayer, who at no small inconvenience and discomfort submitted themselves for the patch tests which are the bases of this study.

# EFFECT OF SYNTHETIC PANTOTHENIC ACID ON ADRENAL HEMORRHAGE, ATROPHY, AND NECROSIS IN RATS

By FLOYD S. DAFT, Biochemist, W. H. SEBRELL, Surgeon, National Institute of Health, United States Public Health Service, S. H. BABCOCK, Jr., and T. H. JUKES, University of California

In a previous article Daft and Sebrell (1) reported hemorrhagic necrosis of the adrenal glands of rats on deficient diets, apparently due to some unidentified dietary factor. It was noted, further, that when rats received an adequate amount of pyridoxine  $(B_6)$  without "filtrate factor" the incidence of adrenal necrosis was very high; while the animals given a crude fuller's earth filtrate from liver or rice polishings did not have adrenal necrosis. Our observations have been extended and it has been found that adrenal hemorrhage or necrosis occurs in almost 100 percent of our rats on a vitamin B complex deficient diet when they receive a supplement containing crystalline pyridoxine but no "filtrate factor," and the experiment is allowed to proceed to death of the animals. As fractionation of the active "filtrate factor" concentrates progressed, it became evident also that the factor preventing adrenal necrosis followed pantothenic acid; but until synthetic pantothenic acid could be obtained, we could not be certain that this active factor was not an accompanying impurity. We wish to report at this time that rats given synthetic pantothenic acid show arrest and repair of the degenerative process in the adrenal glands.

#### EXPERIMENT

Pantothenic acid was prepared from alpha-hydroxy-beta-betadimethyl-gamma-butyro-lactone and beta-alanine by coupling directly with the aid of sodium hydroxide according to the method described by Babcock and Jukes (2). The solution thus prepared was standardized by means of chick assay (3). In this method one chick unit has been shown to correspond to 14 micrograms of natural pantothenic acid (4) or to 28 micrograms of synthetic *dl*-pantothenic acid (2).

Forty-eight albino rats at weaning were placed on our diet No. 461, which consists of leached and alcohol extracted casein, 18 percent, cod liver oil, 2 percent, Wesson oil, 3 percent, Osborne and Mendel salt mixture, 4 percent, and sucrose, 73 percent. After a depletion period of 10 days to 2 weeks, when the weights of the animals were stationary or declining, each was given a daily supplement of 20 micrograms of riboflavin, 15 micrograms of thiamin chloride, 10 micrograms of pyridoxine hydrochloride, 2 milligrams of choline, and 1 milligram of nicotinic acid. There was an immediate and rapid gain in weight which was sustained, however, for only 2 or 3 weeks. After 6 to 10 weeks on the deficient diet, 26 of the animals showed evidence of nosebleed. 7 had a sticky exudate on the eyelids which at times closed the eyes entirely, 3 had depilation about the nose and mouth, and 1 had "spectacled eyes." The rats were then divided into groups: 31 were treated with synthetic pantothenic acid, while 17 litter mates were not so treated. In order to make this curative test as severe as possible the rats appearing to be in the worst condition (from clinical symptoms and weight curves) were treated: the animals which seemed in best condition were used as the untreated controls. Six rats were sacrificed after receiving 6 daily doses of 100 micrograms of synthetic pantothenic acid; 7 rats. after 10 daily doses; and 17, after 14 daily doses. One additional

rat was sacrificed after receiving 14 daily doses, each of 200 micrograms. All of the untreated control animals were sacrificed at the same time that their litter mates completed the 14 days of synthetic pantothenic acid treatment.

The symptoms of nosebleed, ocular exudate, "spectacled eyes," and depilation not only continued to increase in severity in the untreated animals, but also developed in additional rats in this group. These symptoms in the rats treated with synthetic pantothenic acid either disappeared entirely or decreased in severity; and no symptoms developed while the animals were being treated.

The influence of synthetic pantothenic acid on the histopathology of the adrenals is striking.<sup>1</sup> Congestion, fibrosis, scarring, and hemosiderin deposition were found singly or in combination in the adrenals of all the 44 animals examined.<sup>2</sup> Necrosis, atrophy, and hemorrhage, however, were found in only 1 adrenal of 1 rat <sup>3</sup> of the 28 treated animals. Ten of the 16 untreated rats showed one or more of these adrenal lesions. Three showed adrenal hemorrhage, atrophy, and necrosis, 2 showed hemorrhage and necrosis, 2 hemorrhage and atrophy, 2 atrophy, and 1 hemorrhage. Fourteen showed marked fat depletion of the adrenals while none of the treated animals gave a similar finding. If the hormones of the adrenal cortex are intimately associated with the fat, as many investigators believe, then the increased fat in the glands of the treated animals probably indicates a return toward normal function.

#### DISCUSSION

Necrosis and hemorrhage of the adrenal glands of rats on deficient diets have been noted in several laboratories and have been reported from at least two. György, Goldblatt, Miller, and Fulton (5) mentioned this condition in connection with a failure of hematopoiesis. Daft and Sebrell (1) reported that adrenal necrosis occurred on "filtrate factor" deficient diets without the bone-marrow and blood-cell changes noted by György et al.<sup>4</sup> In an accompanying paper, Nelson (6) described the histopathology of the adrenal gland.

Morgan and Simms (7) have reported that in rats deprived of the anti-grey-hair vitamin, the zona reticularis of the adrenal glands degenerated and that there were heavy deposits of yellow pigment and connective tissue and excess vascularity. In an earlier note (8), they

<sup>&</sup>lt;sup>1</sup> All of the histological examinations were made by Passed Assistant Surgeon L. L. Ashburn and our remarks are based on his findings. The details, together with a description of accompanying histopathology of other organs, are reported in "The Effect of Administration of Pantothenic Acid on the Histopathology of the Filtrate Factor Deficiency State in Rats," the following article in this issue of the Public Health Reports.

<sup>&</sup>lt;sup>2</sup> The adrenals from 4 rats—1 in the untreated group, 2 treated for 6 days, and 1 treated for 14 days—were lost and therefore not examined.

<sup>&</sup>lt;sup>3</sup> From gross examination and the histological findings in the other adrenal (used for fat stain), it is probable that this one came by mistake from another animal.

<sup>&</sup>lt;sup>4</sup> A much larger number of rats than reported in the previous paper have now been studied with the same general results.

spoke of atrophy as the change which they noted in the adrenal glands. In reference to hair depigmentation, they say (7), "The concurrent skin and gland changes seen in the rat may well be suspected as being products of the same mechanism."

Our experimental diet presumably contains little or no anti-greyhair vitamin. There is definite evidence of repair of the adrenal lesions following treatment with synthetic pantothenic acid. There can be little doubt of the activity of pantothenic acid, as shown by these experiments; it is possible that other substances as well may be concerned in the production or repair of adrenal damage due to dietary insufficiencies.

The clinical symptoms in rats due to pantothenic acid deficiency have not been completely studied. The treatment with synthetic pantothenic acid was followed by definite improvement in the symptoms of nosebleed, sticky exudate on the evelids, "spectacled eves," and depilation about the nose and mouth.

#### SUMMARY

The adrenal glands of 44 rats were studied histologically. All of these animals received our basic diet and a supplement containing thiamin, flavin, pyridoxine, nicotinic acid, and choline. Sixteen of the rats received no pantothenic acid; 4 received 100 micrograms of synthetic pantothenic acid daily during the last 6 days of the experiment: 7 received 100 micrograms daily for the last 10 days; 16 received 100 micrograms daily for the last 14 days; and 1 received 200 micrograms daily for the last 14 days.

The rats were killed after 52 to 84 days on the deficient basic diet. Ten of the 16 untreated animals had hemorrhage, necrosis, or atrophy of the adrenal glands or a combination of these lesions; 14 showed marked fat depletion of the adrenals. Only 1 adrenal of 1 rat of 28 litter mates given synthetic pantothenic acid had hemorrhage, atrophy, or necrosis; only 4 of these animals showed even moderate fat depletion of the adrenals and this was in patchy areas.

#### CONCLUSION

Repair or prevention of adrenal hemorrhage, atrophy, and necrosis in rats is brought about by a daily dose of 100 micrograms of synthetic pantothenic acid for 6 to 14 days. This is presumptive evidence that a deficiency of pantothenic acid is at least one of the causes of these adrenal lesions in rats on deficient diets.

#### REFERENCES

- Daft, Floyd S., and Sebrell, W. H.: Hemorrhagic adrenal necrosis in rats on deficient diets. Pub. Health Rep., 54: 2247 (1939).
   Babcock, S. H., Jr., and Jukes, T. H.: The biological activity of synthetic pantothenic acid. J. Am. Chem. Soc., 62: 1628 (1940).

- (3) Jukes, T. H.: Further observations on the assay, distribution, and properties of the filtrate factor. J. Biol. Chem., 117: 11 (1937).
- (4) Jukes, T. H.: The pantothenic acid requirement of the chick. J. Biol. Chem., 129: 225 (1939).
- (5) György, Paul, Goldblatt, Harry, Miller, F. R., and Fulton, R. P.: Panmyelophthisis with hemorrhagic manifestations in rats on a nutritional basis. J. Exp. Med., 66: 579 (1937).
- (6) Nelson, A. A.: Hemorrhagic cortical necrosis of adrenals in rates of a hutritional diets. Pub. Health Rep., 54: 2250 (1939).
  (7) Morgan, Agnes Fay, and Simms, Helen Davison: Greying of fur and other distances for the providence of the second distance of
- (7) Morgan, Agnes Fay, and Simms, Helen Davison: Greying of fur and other disturbances in several species due to a vitamin deficiency. J. Nutrition, 19: 233 (1940).
- (8) Morgan, Agnes Fay, and Simms, Helen Davison: Adrenal atrophy and senescence produced by a vitamin deficiency. Science, 89: 565 (1939).

# THE EFFECT OF ADMINISTRATION OF PANTOTHENIC ACID ON THE HISTOPATHOLOGY OF THE FILTRATE FACTOR DEFICIENCY STATE IN RATS <sup>1</sup>

By L. L. ASHBURN, Passed Assistant Surgeon, United States Public Health Service

This report is based largely on the 48 animals described in the paper by Daft, Sebrell, Babcock, and Jukes (1), with some general remarks based on experience gathered from a large series of animals (unpublished data) which were allowed to die while being fed on a diet deficient in the "filtrate factor."

The purpose of this experiment was to determine the effect of pantothenic acid on the clinical symptoms and pathologic lesions of rats on this deficient diet. Details of the experimental procedure are given in the preceding paper (1). This report deals only with the pathologic aspects of the problem.

Tissues were fixed in Orth's fluid and stained by alum hematoxylin Romanowsky (2) and iron hematoxylin Van Gieson methods. One adrenal from each animal was impregnated with 1 percent osmic acid, one part, and potassium bichromate, two parts, immediately after fixation with Orth's fluid, and examined for its fat content.

Nelson (3), from this laboratory, has described the character of the adrenal and associated lesions in rats dying on this deficient diet. The animals reported in his study had died, presumably of the deficiency; hence, advanced and extensive lesions were the common finding. The 16 control (untreated) animals of the present study were killed after being on the deficient diet for 56 to 84 days.

#### MICROSCOPIC FINDINGS

#### ADRENALS FROM UNTREATED ANIMALS

Congestion, hemorrhage, atrophy, necrosis, scarring, fibrosis, hemosiderin deposition, and cortical fat depletion occurred as independent or combined lesions in all of the 16 controls.

<sup>\*</sup> From the Division of Pathology, National Institute of Health.

Congestion.—Congestion was usually slight to moderate in degree and most often involved only the inner third of the cortex. In some glands, congestion was diffuse and marked or occurred as small foci scattered in the fascicular zone. It was slight in 1 animal, moderate in 7, and marked in 2.

*Hemorrhage.*—Hemorrhagic foci were usually small and most often located in the inner third of the cortex. They occurred with less frequency in the outer cortical layers, with or without concurrent involvement of the inner zone. Hemorrhage was slight in 3 animals, moderate in 3, and marked in 2.

Atrophy.—Cell atrophy was a fairly common finding. Sometimes this was manifest only by a reduction in cell size, with slight widening of spaces between the cell rows, and occurred usually as small foci in the inner part of the fascicular zone. Diffuse involvement of the inner half to two-thirds of this zone was occasionally seen, with moderate to marked reduction in number and size of cortex cells. In such areas, stroma was not collapsed and a spongy appearance was produced. Atrophy was present occasionally in areas of hemorrhage, without necrosis; here, cells and cell rows were isolated from one another with reduction in cell size. Slight atrophy was seen in 4 cases; it was moderately extensive in 2 and marked in 1.

Necrosis.-The most common location for small areas of necrosis was the inner cortex at one or both ends of the oval medulla, and usually it involved only a small part of the adjacent fascicular zone. In such an area, cells were seen only as pale oxyphilic masses, nuclei being absent or karyorrhectic. Seen in a later stage, these lesions showed amorphous oxyphilic debris, a few macrophages and slight fibroblast proliferation and fibrosis. From this slight involvement all grades were seen up to subtotal cortical necrosis, with only a few small cells remaining in the glomerular zone. In contrast or in addition to this focal or diffuse necrosis, some animals showed a few isolated cells, scattered throughout the fascicular zone, which were slightly enlarged, had quite oxyphilic cytoplasm, and pyknotic or karvorrhectic nuclei. In the early and small lesions, capillaries were still recognizable and patent; in larger areas of necrosis they were reduced in number, having become necrotic or compressed and unrecognizable. Necrosis was slight in 2 animals, moderate in 2, and marked in 1. This latter animal also showed moderate calcium deposition, much of which was deposited as a surface incrustation on the necrotic cells. Calcification of necrotic debris was a fairly common finding in the animals allowed to die of the deficiency. However, in this group it was seen only cnce.

Scarring.—In agreement with the location of foci of necrosis, scars were most often found at the junction of the fascicular and reticular zones. They were usually small, dense, and narrow or linear, with their long axes parallel to the capsule. In all of this group and in a larger series studied previously, linear scars, when present, always showed this orientation. Oval or irregularly rounded scars were less often present; these were usually located at one or both ends of the oval medulla and were much less dense than those described above. Two scars were found in each of 2 glands and 1 scar in each of 3.

Fibrosis.—Fibrosis was one of the most common findings, being present in 15 animals. It was very slight in 2, slight in 7, moderate in 5, and marked in 1. The fibrosis occurred regularly in the inner cortex and, when slight in degree, was present only in the juxtamedullary zone. It did not give the appearance of a scar, rather collagen fibers of varying thickness ramified between cells producing a fine fibrous feltwork. As this process progressed, the enclosed cells were reduced in number. The end stage was a fairly dense fibrous band in a juxtamedullary position. Besides the fibrosis of this inner zone, a very thin, occasionally incomplete fibrous capsule enclosed areas of calcification, and less frequently necrotic foci were similarly encapsulated.

Hemosiderin deposition .- The presence of hemosiderin pigmentation was determined by Perls' reaction for ferric iron. It was found in all of the 16 animals, being very slight in 2, slight in 6, moderate in in 5, and marked in 3. When it was present in very slight or slight amounts it was located only in the inner zone, often juxtamedullary or in scars. In 2 of the 5 animals showing a moderate amount, a little was also present in the fascicular zone. In one adrenal showing a large amount of hemosiderin, much of it was in the periadrenal fat, capsule, and fascicular zone, while the medulla contained a smaller amount. It was present in scars in larger amounts than in areas of necrosis. When it occurred in the latter location or associated with calcified foci, the hemosiderin was usually peripheral in position, having much the same physical relationship to these lesions as did hemorrhage. Most of the hemosiderin was phagocytosed, particularly that in the inner zone. When associated with hemorrhage or necrosis, it occurred both free and in macrophages.

Glomerular zone.—This zone in animals that died on the deficient diet was frequently absent, absent in stretches, or was quite thin. When it was absent the large cells of the fascicular zone abutted directly on the capsule. This condition was seen in only 1 animal of the present study. In 1, this zone was absent due to necrosis. It was thin in 4 and normal in 10.

Fascicular zone.—In general, the most striking cell alteration, other than necrosis, was decreased cytoplasmic vacuolation. This was seen in 14 animals, being moderate in 5 and marked in 9. In the 9 animals, cell cytoplasm was homogeneous and stained lightly basophilic to amphophilic. Cells appeared normal in one animal and in one other there was marked diffuse atrophy.

Reticular zone.—This zone was distinct, with fairly sharp peripheral limitation, in 6 of the 16 animals. Cells were small, with relatively scanty cytoplasm. Their nuclei were essentially similar to those of the fascicular zone, though the proximity of nuclei to each other made this zone quite dark as compared to the middle layer. In 8 animals this inner zone was quite different. In these, cytoplasm was oxyphilic and slightly to moderately increased in amount; consequently nuclei showed wider separation and the two inner zones were no longer distinct. One additional animal showed an inner zone essentially similar to that just described, except that in variably sized foci the smaller type cells were present. In 1 animal there was no difference in cell type between the two inner zones.

Fat content.—One adrenal of each animal was studied after having been stained with osmic acid. It was realized that some fat might be lost in the process of dehydration and clearing. This was of no serious concern, however, since the purpose of this study was to allow comparison of distribution and amount of fat in the treated and untreated groups.

A number of adrenals from normal rats were studied to establish a basis for comparison. There was, of course, some variation in the fat content, but an average was taken as a standard, and on this basis the adrenals of the treated and untreated animals were rated as normal, or as showing varying grades of fat depletion. This method was not necessarily accurate for any given animal, but served quite satisfactorily for group comparison. Since alteration was present in varying degrees in different layers, they will be described separately.

The amount of fat in the glomerular zone was normal in 7 animals; reduction was slight in 3, moderate in 2, marked in 3, and subtotal in 1.

In the fascicular zone, the fat depletion was quite striking. This zone of 1 animal was normal. Reduction was slight in 1, marked in 5, and subtotal in 9.

The inner zone showed no fat in 9 animals and very small to small amounts in the remaining 7.

In the adrenals containing a normal amount of fat, it occurred as small black to dark gray droplets of fairly uniform size, occupying most of the cell cytoplasm. When fat depletion was moderate to marked, there was considerable variation in size, many being quite large, particularly in the inner zone where some appeared to be outside of cells. In the necrotic areas, fat was rarely seen; at most, only a few cells contained small to moderate amounts. Many of these cells were very probably phagocytes, but in the osmic acid preparation these were not readily distinguished from fat-containing corter: cells. The fact that in many glands the glomerular zones contained normal or almost normal amounts of fat, when it was absent or largely depleted in the remainder of the cortex, is not explained, but may bear some relationship to the possibly different functions of the three layers.

Gross and microscopic observation showed that in animals that died on the deficient diet, there was marked loss of abdominal fat. When marked, there was a disappearance of fat cells; however, when only slight fat loss was present, microscopic study showed a decrease in size of cells and a reduction in number of droplets per cell, the cell cytoplasm not occupied by fat draplets being homogeneous and oxyphilic. Periadrenal fat was examined in 16 animals. It was normal in 3. Fat reduction was slight in 9, moderate in 2, and marked in 2.

#### ADRENALS FROM ANIMALS TREATED WITH PANTOTHENIC ACID

The general statements concerning the various types of adrenal lesions seen in the control animals apply equally to those of the treated groups and need not be reported. Here, only their type and number or extent will be given. There were 31 treated animals divided into groups of 6, 7, and 18, which were treated for 6, 10, and 14 days, respectively. From a pathological standpoint, there is not a sufficient difference between the groups to warrant separate description. In 3 animals the adrenal was lost or the preparation was unsatisfactory for study.

Congestion.—Congestion was present in 12 of the 28 animals; slight in 7 and moderate in 2, being limited to the inner zone in all except 1, in which it also occurred focally in the fascicular zone. This animal is the one referred to under the next heading.

Hemorrhage, atrophy, and necrosis.—These occurred in only 1 animal. This gland occasioned much surprise when examined microscopically. The amount of damage present should have been easily recognizable on gross examination. However, at autopsy the adrenal glands were recorded as normal. The osmic acid preparation showed normal fat content. These facts strongly indicate an unfortunate mislabeling of the adrenal gland while it was being prepared for microscopic examination.

Calcification.—Calcification was present in 4 animals, slight in 2, and moderate in 2.

Scarring.—Nine glands showed scarring, 1 scar in each of 3, 2 scars in 3, and 3 scars in 4 glands.

Fibrosis.—Fibrosis was very slight in 6 glands, slight in 12, and moderate in 8.

Hemosiderin.—Hemosiderin was present in 22 animals. The deposition was very slight in 1, slight in 7, moderate in 11, and marked in 5. In 7 of these, hemosiderin was present in all layers of cortex and capsule. In only 2 of these were focal cortical lesions found (calcification). This is of particular interest owing to the fact that in previous observations large amounts of hemosiderin were found in the capsule and glomerular and fascicular zones only in cases which showed hemorrhagic or necrotic foci in outer cortex. This indicates that the damage to these glands had been much greater than that found following treatment.

Glomerular zone.—This zone was intact in all animals except one. In this rat a short stretch had an appearance similar to the fascicular zone.

Fascicular zone.—Cells of this layer were vacualated and oxyphilic in all but one animal. This is the one referred to under the heading "necrosis" and showed cells with homogeneous, lightly basophilic cytoplasm essentially similar to those of the deficient animals.

Inner zone.—In 17 animals the cells of this layer were small; cytoplasm was scanty and less oxyphilic than the cells of fascicular zone. On low magnification this zone was dark owing to the concentration of nuclei. In 7 additional animals there was only a slight variation of this picture. In these animals, isolated or grouped large oxyphilic cells were seen in small numbers. In 3, there were none of the small cells present, cells being large and oxyphilic which made the separation of the fascicular and inner zones indistinct.

Fat content.—The glomerular zone was normal in 13 animals. Reduction of fat was slight in 10, moderate in 4, and marked in 1.

The fascicular zone was normal in 6 animals. Reduction of fat was slight in 19 and moderate in 3.

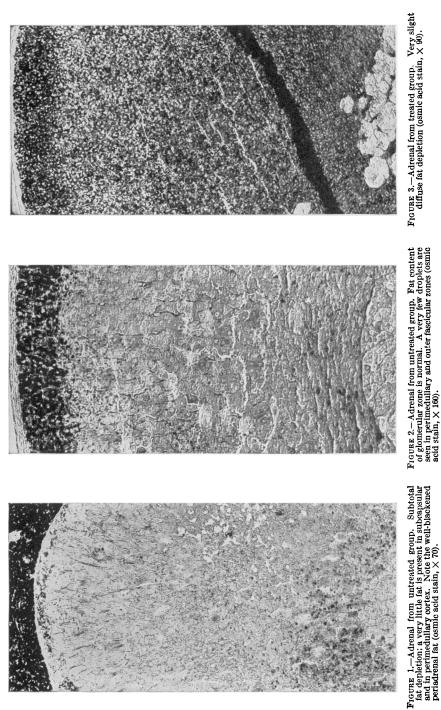
In the inner zone, fat was present in small amounts in 25 animals, in 4 of which most of it occurred as large globules. Three animals showed little or no fat in this layer.

Periadrenal fat was normal in 16, slightly reduced in 11, and in 1 animal it was not examined.

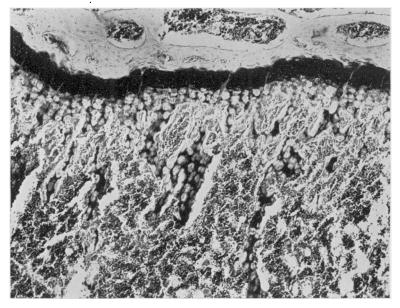
#### MICROSCOPIC CHANGES IN OTHER ORGANS

The only organs examined in this study, other than the adrenal gland, were the testis, spleen, pancreas, and tibia. Other organs have been examined in a previous study. In these, the absence of lesions or the occurrence of apparently unrelated conditions justified their exclusion from this study. The pituitary gland, thymus, and thyroid will be studied in future work on this subject.

Spleen.—The spleen was examined in all animals. In agreement with the findings reported by Nelson (2), there was no significant alteration in this organ other than the occurrence of hemosiderin. In the untreated animals, a small amount was present in 13 and a moderate amount in 4. In the treated group, it was absent or present in insignificant amounts in 12, present in very small amounts in 7, in PLATE I



Public Health Reports, Vol. 55, No. 30, July 26, 1940



 $\label{eq:FIGURE 4.-Upper epiphyseal cartilage and adjacent cancellous bone of tibia from animal that died on deficient diet, aged 75 days. Cartilage measures 65 \mu. There is no active epiphyseal bone growth.$ 

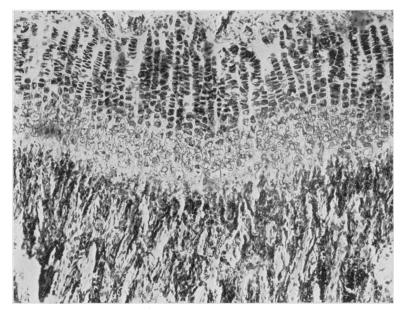


FIGURE 5.—Upper epiphyseal cartilage and adjacent cancellous bone of tibia (treated animal, aged 80 days). Cartilage measures 312µ. Note the thick hypertrophic cell layer and the osteoblasts lining the numerous thin cartilaginous and bony cancelli. small amounts in 11, and in a moderate amount in 1. Although there was more hemosiderin in the treated than in the untreated group, the amount in individual spleens had no constant relationship to the presence or degree of hemorrhage or the amount of hemosiderin in the adrenal.

Testes.—The testes from 20 animals were examined. Of the 14 in the treated group, large, multinucleated, or otherwise abnormal spermatids were present in 8. A few were seen in 3 testes, a moderate number in 2, and in 3 testes they were numerous. Of the 6 testes examined from the untreated controls, these abnormal cells were numerous in 1 and a few were present in 3 others.

Spermatozoa were present in small numbers in 3, and moderate numbers in 4 of the 14 testes examined from the treated group. Of the 6 testes from untreated controls, 1 showed a few spermatozoa and 3 a moderate number. There is a considerable difference in the number of testes with spermatozoa between the 3 treated groups. The 4 testes from the group treated for 6 days showed no spermatozoa. Two of the 4 from the group treated for 10 days showed a few spermatozoa. Of the 7 from the 14-day group, 4 showed a moderate number, and 1 a few spermatozoa. In both groups of animals, the testes showed a few spermatocytes and spermatids which were oxyphilic and occasionally necrotic. The epididymis was not examined in a sufficient number of rats to be significant.

*Bone.*—When studying a large series of animals that had died on a diet deficient in the filtrate factor, it was noted that the upper epiphyseal cartilage of the tibia was quite thin. This finding led to the study of this cartilage in this experiment.

The average thickness of the cartilage in the 17 untreated controls was 203µ. The hypertrophic layer (preparatory zone) averaged about three cells thick, measuring  $71\mu$ . The cartilage of the 31 treated animals averaged  $311\mu$ , with a hypertrophic layer averaging  $108\mu$  in thickness. This latter zone was usually four or more cells thick. In both the treated and untreated groups the cancellous zone showed slight to moderate congestion and numerous large osteoblasts. In most animals the periosteum of the tibial shaft was cellular and of moderate thickness. It is of interest to compare the above findings with similar features of animals that died while on the deficient diet (unpublished data). The average age of these animals was slightly less than that of the treated group. In these, the cartilage averaged 118µ in thickness; the hypertrophic layer was absent or very thin, being only one to two cells thick. Many animals showed absence or marked retardation in epiphyseal bone growth, cancelli being short and present in very small numbers. Osteoblasts were absent in 4. present in small numbers in 6, and in moderate numbers in 5. In 237922\*-40-2

most animals, the periosteum of the tibial shaft was seen only as a layer of fibrous tissue with very few small spindle cells. In 1 animal it was thick.

Pancreas.—Pancreas was examined in 5 of the treated and 4 of the untreated animals; it was normal in all.

#### DISCUSSION

The frequency and degree of adrenal congestion was somewhat greater in the control than in the treated animals. However, no significance is given to the fact since congestion is not necessarily associated with pathological processes. It may merely be a part of an increased or attempted increase in functional activity.

A striking difference is seen when the groups are compared with respect to hemorrhage, atrophy, and necrosis. In the deficient animals, these lesions occurred singly or in combination in 10 of the 16 animals. All 3 types of lesions were seen in 3, hemorrhage and necrosis in 2, hemorrhage and atrophy in 2, atrophy in 2, and hemorrhage in 1. Of the 28 treated animals, only 1 showed active lesions. Reasons were given under the heading "necrosis" for the belief that this adrenal was mislabeled and did not belong in this treated group. It is apparent from this and previous studies of this deficiency that calcification is preceded by necrosis; hence, necrosis had occurred in at least 3 other treated animals. There was no associated hemorrhage or atrophy, which suggests that the necrosis had occurred previous to the beginning of treatment.

Scarring occurred with about equal frequency in the two groups, a finding that was expected since small foci of necrosis do not ordinarily increase in size. Rather the debris is phagocytosed or organized, resulting in the small scars. Fibrosis and hemosiderin deposition, though present in almost all animals of both groups, were slightly more pronounced in the untreated animals. These changes, particularly fibrosis, in the majority of instances were confined to the inner zone, and when in this location were not associated with hemorrhage or necrosis. It is believed that this type of alteration is a continuous process and in the case of fibrosis, irreversible. It was impossible to determine whether or not hemosiderin continued to be deposited during the period of treatment. However, it is believed that the 14 days (longest period) of treatment was too short a time for the previously formed hemosiderin to have been removed.

There was a distinct difference in the appearance of cells of the fascicular and inner zones between treated and untreated groups. In the treated group cells of the fascicular zone showed, except in one case, normal vacuolated oxyphilic cytoplasm, whereas in the untreated group this type of cell was present in only one case. The appearance of the cells of the inner zone was considered within normal limits in about 30 percent of the untreated animals and in about 88 percent of the treated group.

One of the most striking differences between the adrenals of the two groups is their fat content, particularly in the fascicular zone. Fourteen of the untreated animals showed marked to subtotal depletion of fat in this layer. In the treated group this zone was normal in 6 animals and fat was only slightly depleted in 18 others. The remaining 4 showed moderate depletion in patchy areas. The relationship of cortical fat to cortical hormone is still a much debated question. However, there appears to be general agreement that the relation is close and that the functional state of the gland can be gauged by its fat content (4). With this view as a criterion, it is evident that the adrenal cortices of most animals on the deficient diet have partly or completely lost the ability to produce the specific hormone. By the same reasoning, it is apparent that this function is largely restored by treating the deficient animals with pantothenic acid.

Pantothenic acid did not cause the testes to regain their normal appearance or function in 14 days, although the analysis of the animals by length of treatment suggests a trend in this direction. Two male rats, not a part of this experiment proper, but which had been handled in the same manner, were continued on the treatment for 38 days. When killed their testes showed no abnormal cells and spermatozoa were numerous. These testes were considered to be normal.

On the deficient diet rats lose weight or fail to grow. When the animals are treated with pantothenic acid the tibial epiphyseal cartilages are hyperactive, which suggests that skeletal growth is resumed or accelerated.

Many aspects of this and previous studies indicate that we are dealing with various degrees of adrenal cortical insufficiency due to a vitamin deficiency. Reduced fat content of adrenal cortex, reduced rate or lack of growth, loss of abdominal fat and reduced testicular function all lead to the consideration of such a probability. Such lesions as visceral congestion and generalized lymphoid hyperplasia described as occurring in experimental chronic adrenal cortical insufficiency were not observed. With reference to thymus hyperplasia, which has been described as occurring in chronic cortical insufficiency (5), it should be stated that most of our animals were under 90 days of age and all were under 107 days. Since these animals were too young to show thymic involution, this organ was not examined.

From this experiment and previous experience, it was noted that hemorrhage and necrosis frequently occurred independently. Not infrequently animals die without showing either hemorrhage or necrosis, other forms of adrenal alteration being present. Although the term "hemorrhagic adrenal necrosis" designates the prominent pathological feature of this deficiency, a more inclusive title should be found for this condition when more is known concerning the pathologic physiology.

#### SUMMARY AND CONCLUSION

The adrenal lesions, abnormal testicular function, and cartilage hypoplasia occurring in rats fed on a diet deficient in the "filtrate factor" are markedly affected by supplementing the diet with 100  $\gamma$ of pantothenic acid daily. Arrest and repair of adrenal lesions occur, testicular function is improved, and skeletal growth is accelerated.

It is suggested that rats develop a partial or complete adrenal cortical insufficiency on the deficient diet.

#### REFERENCES

- (1) Daft, Floyd S., Sebrell, W. H., Babcock, S. H., Jr., and Jukes, T. H.: Effect of synthetic pantothenic acid on adrenal hemorrhage and necrosis in rats. Preceding paper in this issue of Public Health Reports.
- (2) Lillie, R. D.: Romanowsky staining with buffered solutions. III. Extension of the method to Romanowsky stains in general. Stain Technology, 1940. (In press.)
- (3) Nelson, A.: Hemorrhagic cortical necrosis of adrenals in rats on deficient diets. Pub. Health Rep., 54: 2250 (1939).
  (4) Zwemer, R. L.: A study of adrenal cortex morphology. Am. J. Pathol., 12:
- 111 (1936).
- (5) Jaffe, Henry L.: The effect of bilateral suprarenalectomy on the life of rats. Am. J. Physiol., 78: 453 (1926).

# **PROVISIONAL MORTALITY RATES FOR THE FIRST QUARTER** OF 1940

The mortality rates in this report are based upon preliminary data for 30 States, the District of Columbia, Alaska, and Hawaii for the first 3 months of 1940. Comparative data are presented for the same States for the 2 previous years.

This report is made possible through a cooperative arrangement with the respective States, which voluntarily furnish provisional quarterly and annual tabulations of current birth and death records. The reports are analyzed and published by the United States Public Health Service.

Because of lack of uniformity in the method of classifying deaths according to cause as well as some delay in filing certificates, these data are preliminary and may differ in some instances from the final figures subsequently published by the Bureau of the Census.

In the past, these preliminary reports have accurately reflected the trend in mortality rates for the country as a whole. Some deviation from the final figures for individual States may be expected because of the provisional nature of the information. However, it is believed that the trend of mortality within each State is correctly represented. Comparisons of specific causes for different States are subject to error

because of differences in tabulation procedure and completeness of reporting. Such comparisons should be based upon the final figures published by the Bureau of the Census.

The causes of death were classified according to the latest revision of the International List and the numbers in parentheses after each cause are the code numbers of this revision. The number of States reporting for the past quarter is smaller than usual, probably as a result of the change in classification of causes of death which most States are now making.

In general the mortality record of the first 3 months of this year has been favorable. The crude death rate, 11.7 per 1,000 population, was slightly less than the corresponding rate for 1939, 11.8 per 1,000 population, although it was somewhat higher than in 1938. The States reporting a higher death rate in 1940 than in 1939 were all in the South or West.

The principal infectious and contagious diseases were all less prevalent than last year, and for all except influenza the mortality rates were less than those reported in either 1938 or 1939. A minor epidemic of influenza during January and February was responsible for maintaining the death rate from this disease at a relatively high level. The decline in the mortality rate from tuberculosis continued unchecked so that the rate for the first quarter of 1940 was about 5 percent less than that for the corresponding period in 1939. The largest decline was reported for pneumonia, the death rate from this disease being nearly 20 percent less than during the first quarter of 1939. Although it is still too soon to form a definite opinion, it is thought that the use of recently discovered methods of therapy undoubtedly contributed to this decline. Only 4 of the 31 States reported a higher death rate in 1940 than in 1939.

Poliomyelitis was the only communicable disease with a higher death rate than in 1939. The increase was insignificant since this disease is most prevalent during the last half of the year. Increases were reported for the principal diseases of late adult life, cancer, cerebral hemorrhage, diabetes, heart disease, and nephritis. In part, at least, these increases result from an increased proportion of old persons in the population.

The death rate from accidental causes was also higher than in 1938 or 1939. Seventeen of the 31 States reported a higher death rate from automobile accidents than in 1939. The increase was about 3 percent.

The downward trend in the infant and maternal mortality rates continued unchecked; the maternal mortality rate, 3.9 per 1,000 live births, was 15 percent less than that for the first quarter of 1938.

The birth rate, 16.3 per 1,000 population, was slightly higher than that reported during the corresponding period of the two previous years.

2	
-sp	
ğ	
Ĩ	
R	
ېن. د به	
<u>چ</u> .	
5	
6	
ž.	
P	
õ	
68	
Ę	
<u>र</u>	
ž	
7 1	
ž	,
2ta	
ซ	
la'	
<u>ē</u> .	
<u>ي</u> .	
20	
ē,	
_e	
at,	
ē	
du	
8	
4	2
pith	0.00
), with	0.0010
140, with	0.001t
1940, with	10/10
of 1940, with	10000
he of 1940, with	e-2011
nths of 1940, with	10/10/1
months of 1940, with	e
3 months of 1940, with	ennait
st 3 months of 1940, with	en11011
first 3 months of 1940, with	410/14
he first 3 months of 1940, with	e#U016
n the first 3 months of 1940, with	
s in the first 3 months of 1940, with	
ses in the first 3 months of 1940, with	
auses in the first 3 months of 1940, with	
t causes in the first 3 months of 1940, with	
rin causes in the first 3 months of 1940, with	
rtain causes in the first 3 months of 1940, with	
certain causes in the first 3 months of 1940, with	
m certain causes in the first 3 months of 1940, with	
0	
0	
ity from c	
rtality from c	
rtality from c	
mortality from c	
nal mortality from c	
mortality from c	
visional mortality from c	
onal mortality from c	

5	а
•	<u>ه</u>
3	2
	~
•	
•	

I	(1100° P° C)	4 60 69	14.7 16.6 18.0	~~~~	- 26	91-9	
	Automobile socidents	01 20 20 01 20 20	401-	333		1881 1982	220
	All accidents, including automobile accidents	688	228	156. 137. 815.	***	258	****
	Nephritis, all forms (i30-132)	4.28	68.8 58.3 58.3	12.5 12.5 19.3	8.88 8.88 8.89	8.08 8.08 8.08	88.89
	Diarrhes and entertits under 2 years (119)	6144 0 8 90	844 14 14 14 14 14 14 14 14 14 14 14 14 1	335	8.8 7.7	899 940	4 53 F
	Diseases of the digestive system (115-129)	52.8 54.0 54.0		1273 1004 1004	52.28 27.28 2.28	60.1 63.9 75.4	40.6 40.6 40.6
	Pneumonia, all forms (107-109)	87.3 107.5 104.7	57.0 78.4 78.6	180. 1 180. 9 366. 9	8 8 1 8 1 8	118.1 154.9 144.1	8.08
asds)	Diseases of the heart (90-95)	<b>333.8</b> 318.0 285.2	* 182.5 * 183.0 * 169.4	196.1 205.9 302.6	424.7 420.5 396.0	322.4 276.5 249.0	280.4 280.1 276.3
d lænat	Cerebral hemorrhage, embolism, and throm- bosis (83a, b)	102.4 95.8 88.7	67.13 66.03 64.13	78.0 49.9 225.3	100.0 102.6 89.9	95.4 105.6 98.5	128.88
ion (ar	Disbetes mellitus (61)	20.5 25.9 25.9	88.8 89.12	E.S.E	28.2 4.7 28.2	18.9 19.5 17.4	888
opulat	Cancer, all forms (45-55)	122.1 119.3 114.9	104.9 101.9 94.9	90.0 99.0 100.4	164.6 153.7 147.5	127.8 113.1 117.0	163.5 146.5 141.3
Desth rate per 100,000 population (amual basis)	Tuberculosis, all forms (13-22)	64 45 64 66 65 64 0 8 8 64	45.8 47.4 47.7	<b>444</b> . 2 324. 4 740. 3	62.6 64.5 71.5	56.5 64.15 64.1	38.3 37.6
e Der 1(	Cerebrospinal (meningo- coccus) meningitis (6)	1.2		EEE	1.2		3.5 .5
sth rat	Acute infectious enceph- alitis (lethargic) (37)	0.5		E.E	<u>,</u>	***	EE .
Å	Acute polioencephalitis (36) polioencephalitis (36)			EEE	4.0.4	31.1 1.1	333
	Influenza (grippe) (33)	31.2 32.8 21.1	16.6 18.7 4	13.8°	13 8 9 1 1 8	<u>888</u>	8.0 4.7
	Diphtheris (10)	49894 1911 - 19	51.3	e°e	5.1.1 53	014 10 01 1 10	11. 11.
	Whooping cough (9)	ະເອີຍ ເອີຍ ເອີຍ ເອີຍ ເອີຍ		87.0 173.8 8.0	1.00 L	8.0 40 10	1.8
	Scarlet fever (8)	0.9 1.3		EE.	С <sup>9,9</sup> .		£
	(35) 251289 M	4.1.24		<b>පි</b> ල ද		01-i-4i	3.12
	Typhoid fever (1-2)	0.5	10 4 00	EEE	1.3		EEE
Rate per 1,000 live burths	Maternal mortality	8.4.4 6.1 6.1		Eui a	0000 010100 010100	01 10 10 10	0 00 00 01 01 01
Rat bur	Total intant mortality	822		-	442		***
rths) per	Births (exclusive of stillbi 1,000 population (annus	16.3 16.1 15.6		***	15.8 15.3 15.5		5 5 5 5 5 5 5 5 5 5 5 5 5 5 5 5 5 5 5
s) ) bobn <del>ja</del> -	All causes, rate per 1,000 tion (annual basis	11.7 11.8 11.2	ත්ත්ත් දෙන ත	80.08 80.08 80.08	13.5	12.5	111
	State and period	31 States: 1 1940	1940	1940. 1839. 1838. California: •	1839 1839 1938 Colorado:	1940. 1889. Connectiout:	1940. 1839. 1838.

1349

July 26, 1940

1 12.2 8 20.3 1 21.7	0 16.7 7 18.3 1 21.1	2011 10 10 10 10 10 10 10 10 10 10 10 10	2 7.6 4 11.3 0 9.7	6 18.9 5 25.7 4 24.3	8 8 7 8 8 7 9 3 7 9 3	8888 8888 846	3 15.4 3 15.9 6 15.5	6 17.9 1 16.9 9 22.2	5 16.9 8 18.7 1 17.2	5 25.2 217.6 7 18.8	5 19.6 8 12.3 6 14.7	4 20.3 38 18.7 4 21.9
8698 8698	202 255 5255	8 9 1 7 8 1 1 3 8 1 1 3 8 1 4 8 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1	3338 2436	5×3	233	893	523	688	833	ଞ୍ଚଞ୍ଚ	888	8233
38138 138138	141	<u>8833</u>	88 22 28	8828	103.3 112.5 100.0	84.1 67.8 64.7	72.7 60.9 67.4	108.4 104.0 109.2	71.5 66.6 65.3	131.3 124.4 106.7	87.2 81.1 96.0	153. 153.
36.3	5.6 7.6 5.1	4.9 11.1 5.5	5.1 10.4 17.7	0101-1 440	1.5 3.0 3.0	000 000	1.2 1.7 3.1	1.9 2.8 2.6	4.5.4	7.8 8.1 8.1	7.5 4.7 7.1	99 ¥ 99
53.3 54.0 34.1	79.2 79.5 2.2	80.1 83.4 78.8	43.8 55.6 61.1	49. 7 54. 6 60. 0	61.0 58.4 61.6	EEE	47.4 59.7 60.7	57.2 52.3 58.2	48.2 46.3 34.6	67.1 62.2 60.7	53.2 49.0 56.7	55.2 52.2 56.1
92.9 145.0 114.5	134.9 110.4 152.0	110.5 83.1 111.0	48.1 57.3 71.7	55.2 106.0 118.4	76.2 103.7 92.8	97.3 138.7 103.8	77.9 91.1 96.4	56.3 95.0 95.0 95.0 95.0 95.0 95.0 95.0 95.0	100.6 119.8 104.6	174. 6 149. 8 152. 3	71.4 113.6 105.9	125.6 136.5 135.1
431. 1 417. 9 400. 9	421.4 403.7 365.1	451.6 307.9 326.6	118.0 129.4 116.9	258.7 277.9 181.7	397.1 400.6 341.5	360.4 275.9 256.6	308. 1 294. 9 262. 4	297.2 270.4 252.3	226.9 214.9 166.6	371.5 248.0 227.5	389. 1 422. 5 345. 5	452.0 396.9 366.8
118.8 120.3 142.4	101.5 93.3 93.9	163.2 116.2 117.9	44.7 37.3 50.5	62.3 88.3 68.1	94.3 83.0 78.6	162.3 146.3 129.8	121.0 116.1 118.9	107.1 90.4 94.5	111.7 99.2 89.5	94.8 85.5 85.3	133.4 135.8 122.4	126.3 122.6 101.9
47. 2 40. 1 34. 1	40.8 32.2 24.9	32.9 26.3 27.7	14.3 18.2 11.5	18.1 26.5 13.8	40.3 33.3 31.0	19.4 20.8 17.3	26.3 31.8 24.9	30.3 34.8 26.4	15.3 15.2 13.4	27.2 18.4 21.7	34.1 29.7 27.9	43.9 32.6 32.8
155.4 117.2 123.8	149.7 160.8 131.6	115.0 97.5 107.3	59.0 52.1 63.8	87.5 94.8 67.3	142.6 140.6 132.6	121.8 112.5 114.0	125.5 120.2 139.2	119.9 112.9 119.6	74.3 75.5 58.2	97.9 81.2 86.4	133. 0 149. 0 145. 1	148.7 138.1 129.8
40°5	65.0 69.6 69.6	54.5 56.1 58.4	88.88 89.89 84.80	16.6 19.3 21.9	47.3 48.9 46.1	30.4 44.2 72	14.4 19.0 20.5	23.3 24.2 27.9	88.0 89.0 89.0	69.6 61.0 72.6	25.2 33.5 33.1	97.2 80.9 81.4
1.5 (7) 1.5	2.1.3 2.6	1.1 1.1	8. .9		004	е. <mark>1</mark>	1.0	1.1 		1.7	3.5	555
3 <b>.</b> 5	(1) 1.3	141	EEE	1.6.88	4.0.4	-1-6	4.2.75	1.4	4.1	<u></u>	33. <sup>2</sup>	1.02
EEE	33.	eee	1.7 (1) 1.8	EEE	-99	3.2 .5	(1). 5	EEE	1.2		33	33
33.5 24.7 27.9	21.0 8.9	87.4 38.8 41.6	545 53	25.2 28.1 21.9	20.0 35.6 10.5	23.8 23.8 23.8 25.8	39.1 31.1 23.9	43.5 38.7 25.3	6860 8860 8870	100.4 45.2 59.0	22.4 32.5 23.6	24.3 23.0 16.1
3.1 3.1	1.9	2. 1.1.1 8.4	9.65 10 10 10	3. 8. E	1.3 1.6	1.9 3.1	1.2	1.1	3.8 3.8 3.8	535 53 57 57 57 57 57 57 57 57 57 57 57 57 57	1.4 8.6 1.4	1.2.
3.0 10.8	3.1 1.9 .6	100 707	0.04 4.00	848 849	.9.	2.1.0 2.1.08	40.	1.5 3.3	3.9 8.1.8 4.8	8.5 4.9 5 5	¢.4.8 8.1-80	4.1.4
33°		0.4.4	EE°.	4.00.00	4.0.5 7,004	0.4 0 0 4 0			1.5	С 	1.8 3.5	1.257
~~33 ~~33	<u>6</u> 03	277 877	<b>666</b>	1.6 0 .8 4 1.6	4 3 3 7.1	8.39 8.39	5 2.4 5 .5	5.28 5.20 5.20	8 1.1 2 5.2	8.1.8 .05	4 1. 9 3.8 3.8	527
<u>€6</u> +	<u>644</u>	10.80	-104	000	4.00	000		<del>404</del>	11.	20010 20110	1.12	~~~~
್ 4 ಲೆ	0,004	~ 67	-i-icirci	5 S S S S S S S S S S S S S S S S S S S	ಣೆ ಣೆ ಣೆ	ণ্ড ৰা ৰা	€-i <del>*</del> i	ं चं चं	644	729 74 6.6	ম্য বা বা	60 52 88 80 52 88
44 61 61	89 69 53 89 69 53	2883	86255 56255	8834	444	<b>464</b>	©44 843	4885 4885	2 (°) 45 52	400	86757 506757	8 2 2 2 
16.4 17.6 15.9	22.0 21.1 20.3	17.2 17.1 16.9	19 19 19	27.2 2.1 2 0.0	13.8 13.9 14.3	16.0 15.7 15.8	(e) 17.3 16.9	13.	ିଟ୍ଟର୍	21.22	16. 17.	17.18
13.6 13.7 13.2	14.8 14.3 13.7	17.0 13.6 14.3	7.0 6.9 7.2	9.6 10.4 9.4	12.4 12.6 11.5	12.8 12.8 11.6	10.9 10.9 10.5	11.1 10.8 10.8	10.9 11.0 9.4	15.5 12.3 12.3	12.8 14.0 12.9	15.7 14.4 13.4 ble.
Delaware: 1940. 1889. 1888. District of Columbia:	1940 1939 1938 Florida: *	1940 1839 1938 Hawaii	1940 1839 1838 1838 1838	1940 1839 1938 111inois	1940 1939 1938 1938	1940 1939 1938 1938	1940. 1939 1938 Kansar	1940 1939 1938 Kentucky:	1940 1939 1938 Louisiana: *	1940 1839 1838 Maine:	1940 1939 1938 Marvland:	1940

Provisional mortality from certain causes in the first 3 months of 1940, with comparative provisional data for the corresponding period in preceding years—Continued

	Automobile secidents (170s, b, c)	188	14 18 18 18 18 18 18 18 18 18 18 18 18 18	19.0 20.0	888 28 88 28	10 10 10 10 10 10 10	9.44 4.44 4.90	121 121 121 121 121 121 121 121 121 121
	All secidents, including sutomobile secidents (169-196)	2589 2589 2589	885	8:99 1 1 99	288 288	885	<b>333</b> 74	131.5 78.7 78.7 107.4 54.3
	Nephritis, all forms (130-132)	898 898 033	464 464	22 8.88 8.88	114.4 116.4 107.1	85.2 86.2 8.7 8	288 288	52.52 S
	Diarrhes and entertits under 2 years (119)	14 CU 14 CU 14 CU		66 10	8554 41~61	8999 899 899		<sup>ه</sup> 333
	Diseases of the digestive system (115-129)	288	තී ලි යි ම හ හ ම හ හ	45.1 45.7	58.2 59.2 57.3	88.1 88.6 8.8	584 498	81.2 80.4 81.7 81.7
	Pneumonis, sil forms (107–109)	1987 1987 1987	200 200 200 200 200 200 200 200 200 200	114.0	116.6 130.0 141.2	63.8 118.7 116.8	K83	131.5 166.4 166.0 166.0
ests)	Diseases of the heart	850.4 832.8 808.2	315.0 265.7 255.9	190.0	272.0 272.0 287.8	28.1	555 55 55 55 55 55 55 55 55 55 55 55 55	200.0 884.7 802.2 304.4
d land	Cerebral hemorrhage, embolism, and throm- bosis (83a, b)	101.8 97.6 8.4	105.7 102.8 90.5	91.9 83.3	102.4 98.6 91.9	888 848	128.0 285.6 78.5	86.1 86.6 71.6 96.1
Desth rate per 100,000 population (annual besis)	(18) surfillem setedald	20.25 20.25	2002 2002 2002	18.1 14.1	32.4 27.3	8 8 8 0 8 9 0 8 9	82.8 1.8	19.3 15.9 15.9
opalst	Cancer, all forms (45-55)	128.9 128.9 118.1	136.4 139.4	200 200 200	130.4 126.9 122.6	114.5 112.8 84.8	122.2 107.8 117.0	116.0 129.9 90.4 133.5
0,000 p	Tuberculosis, all forms (13-22)	2000 2000	8888 8888	48.2 40.5	44.8 48.9 51.8	<b>44.</b> 2 80.1 80.1	18.7 16.4 12.7	42.5 56.1 87.5 41.1
per 10	Cerebrospinal (meningo- coccus) meningitis (6)	0	404	3.0	1.03 .8	1.4 .7 1.5	0.00	°EE E
th rate	Acuto infectious enceph- slitts (lethargic) (37)	0 0	<b>0</b>	1.0	10.04	. 4E	е <b>-</b> е	EEE
Dee	Acute polioencephalitis (36) polioencephalitis (36)	333	.е.	1.0	- 1917		EEE	33"
	(EE) (9dqfr3) <b>san</b> snfhal	17.04 13.3	18.8 24.4 14.3	118.7 84.8	888 80 10	21.0 35.4 33.5	255.8 26.8 20.0	11.6 15.7 4.0 9.7
	Diphtheris (10)	011	4.00.00	64-1 200	91-6	1.57	1.90	68 <b>°</b>
	W hooping cough (9)	800 1111	2.84	5 5 5 6	. 2.1 7.2	4.8	8.8 8	EEE
	Scarlet fever (8)	1.00 1.00 1.00 1.00 1.00 1.00 1.00 1.00	10.02 10.12 10.02 10 10 10.02 10 10 10 10 10 10 10 10 10 10 10 10 10	<b>6</b> .	4864	5 3 0 2 2 2 5 2 2 5 2 2 5 2 5	1.8	° 333
	(35) Measles (35)	323 1-0 223 223	8.0 8 . 0 . 8	9.9	4-0	48. 	€-€ 	· 3°3
	Typhoid fever (1-2)	00 00 00	 			<u>600</u>	<u>∞∞∞</u>	EE4
Rate per 1,000 live births	Maternal mortality	න්න්න් 	<u>644</u>	23 23	4 00 00 22 22 22	884 440	6983 6983	<u>8888</u> 794 9
	tilariom tnaini latoT	4.01	1 10 00	23 EE	12.0	19.80	16.7	19.9
rths) per	Births (exclusive of stillbi	11.0	10.81		808		040	000001 4
bobnja-	All causes, rate per 1,000 tion (annual basis			21 1		939 		<u> 1111</u>
	State and period	9.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00	800 to 1	0 0				66869: 10 10 10 10 10 10 10 10 10 10 10 10 10
	~	Michigan: 1940. 1939.	1939 1938	Minor	1940 1938 1938	999	19 19	New Jersey 1939

16.5 18.5	647 879 879	14.1 13.5 17.3	21.7 21.7	12.4 7.4 10.9	0 8 8 8 8	16.5 21.8 17.4	32.1 26.1 17.6	14. X 12. 8 16. 9	***	899 800 400	808 808 808	800
<b>48.0</b> 51.2	89.5 89.5 87.5 87.5	61 4 4 6 8 8 6 6 4 4 6	0.989	188 0-4	79.2	8.09 9.84 9.84	21.0	55.3 60.6 80.8 80.8 80.8 80.8 80.8 80.8 80.8	823	62.8 51.6 57.4	57.6 2 81.9 3	494 104
80.1	24.0	81.3	ල හ හ ල හ හ ල හ හ	844 844 41-4	80.8	57.4 61.3 66.6	132.7	21.5 20.5 20.5 20.5 20.5 20.5 20.5 20.5 20	888	00r	480	87.10
893 893	40.4	000 000	010 010	4000 000	0000 0000	846	440	444	804 804	000 000	600 800	1011 1011
0.00	<u></u>	787	010	00-1-00	P- 00 00	000	004	000		-08	000	00 00
. 7 8 57.	213	666	9999 9999	884 444	255	848 \$48	8984 8484		040 8233	884 884	6488 4888	848
0 80 80	1100 148. 148.	0 8 8 0 5 8 8 0	192		907 9518	8189 8189	190 8,8,8	<u>+ 0 4</u>	9 125.	12 12 12 12 12 12 12 12 12 12 12 12 12 1	82780 82480	8.94 19.98 19.98
7 343. 343.	75154 75155	8 4 8 8 8 8 8 8	6 158 174 166		801-90 802-80 802-80 80-90 80 80-90 80 80 80 80 80 80 80 80 80 80 80 80 80	405 12 12 12 12 12 12 12 12 12 12 12 12 12	1004 1208 1208	6 800. 6 864. 347.	0 216 170. 185.	44 1983 1983	245.00	340 339.71 339.71
91. 85.	843	225	88.88	878	82.21	<b>88</b> 8	118. 131. 107.	***	01 88 28	822	843	128.02
38.1 30.8	14.1 10.5 3.8	85.5 85.5 2.2	17.9 14.0 11.6	880 880 10	28.6 28.6	17.6 15.7 14.4	30.6 31.1 25.1	45.4 30.4	17.7 13.1 11.8	18.9 11.6 10.8	16.2 14.8 22.6	19.5 34.4 30.5
132.0	88.98 89.79 9.79	158.6 158.7 156.8	57.5 58.3 58.3	99. 6 87. 4 84. 2	142.5 134.2 127.6	75.4 75.4	142.6 135.7 131.3	122.7 121.9 118.1	8.44 8.74 8.73	883 120	88.8 88.8	114. 9 139. 7 121. 9
44. 7 45. 6	91.2 94.2	49. 1 52. 6 52. 3	888 440	18.1 22.3 20.6	<b>44</b> 46.7 48.8	45.5 42.1 54.1	23.2	41.8 41.1 42.1	80.0 80.0 80.0 80.0	76.1 71.8 74.4	18.5 21.8 21.0	38.0 37.5 45.2
1.0	6%-i	1.13	1.65	611 121	1.1.48	1 . 1 1	1.1	40.0	400	1.1	.0.4 8.627	31.0
<u></u>	EEE	0.001-	-1.02	31.1	604		1.1	400	<u> </u>	<u>616</u>	3.8 8.8	е <b>-</b> е
.1	0.1-0 1-0	EE		EEE	<u></u>	1.5	£	Э.:е	<u>80</u> 60 4	1.91	33 <sup>.</sup>	333
13.9 S.1	33.8 64.7 32.7	686 400	28.28	15.8 30.3 10.3	848 8748	48.9 43.7 34.6	20.1 14.8 16.5	31.8 15.0 20.4	65.5 65.8 65.8 4 65.8 8 8 8 8 8 8 8 8 8 8 8 8 8 8 8 8 8 8	60.9 66.79 46.97	24.7 14.0 21.0	25.7 38.6 24.2
1.3	4.8.9		<b>4</b> 46 201	800 800	1.6	4.03	4 <sup>C</sup> -	1.28	1.75	3550 3550	EEE	ε-ε
2.1	2008 2008	1.3	101-	1.7	5038 5138	1.2 9.8 9.8	3. 1.68	1.202	8.76	887 987	3.1 4.7	1000 1001
1.3	e∔e	-1000	41-0	1.28	101	1.6	1.6 1.6	2.0	1.1	040	80. 1 1 1	e-e
13	E-18	61 ID 61	10.08	6.9 1.1	5.3 5.3	6.0 1.1	<b>*</b> EE	1000	9.4.6 9.4.0	1.1	46°	ÊS3
<u></u>	80.08 81-181	-99	1.01	1.91	664	-00 -00	3. <u>1</u> .5	C- 00-73	1.10		EEE	-66
8 8 8 8 8	807 108	8884	89.08 19.14 19.14	1.00 Li 1.00 Li	8.4.8 0.09	844 811	80111 10100	800 800 800	r.98 440	0 1 0 6 2 2 0	0000 0000	3.50
43	823	844	858	384	134	284	848	888	82 288 87 288	228	488	37 23
12.9 12.9	42.0 35.3 32.6	14.1 14.6 14.6	21.3 22.4 22.4	19.2 19.4 18.0	15.8 14.4 16.1	16.6 17.0 18.5	16.0 15.1 15.7	13.6 15.7 16.3	18.8 17.4 17.2	15.2 14.2 14.2	24.5 24.5	17.0 14.5 14.8
11.5	13 19 19 19 19 19 19 19 19 19 19 19 19 19	11.4 12.9 12.3	10.4 10.3	1001	13.0 13.1 12.0	866 866	12 2 12 2 12 2 12 2	12.8 11.9 12.0	11.6 9.2 10.1	11.2 9.5 9.5	9.5 9.5	10.9 12.4 12.3 ble.
1839 1988 New Mexico:	1940. 1939. 1838. New York:	1940 1839 1938 North Carolina:	1940 1939 1938 North Dakota:	1940 1639 1938 Oblio:	1940. 1939. 1938. Oklahoma:	1940	1940 1939 1938 Pennsylvania: •	1940 1938 1938 South Carolina:	1940 1939 1938 Tennessee:	1940. 1939. 1938. U tah:	1940 1939 1938 Vermont:	1940

1351

July 26, 1940

Provisional mortality from certain causes in the first 3 months of 1940, with comparative provisional data for the corresponding period in preceding years—Continued

	Automobile accidents (1708, b, c)	22.00 21.22 21.21 21.20 21.20 21.20 21.20 21.20 21.20 21.20 21.20 21.20 21.20 21.20 21.20 21.20 21.20 20 20 20 20 20 20 20 20 20 20 20 20 2
	All accidents, including automobile accidents (169–195)	88.2 74.8 56.7 74.8 56.7 74.8 71.3 69.1 5 71.3 69.3 88.6 5 64.8 88.6 64.7 71.3 89.1 8 88.6 6 47.3 8 88.6 8 88.6 8 88.6 8 88.8 8 88.8 8 88.8 8 88.8 8 88.8 8 88.8 8 88.8 8 87.3 8 85.0 8
	Nephritis, 811 torms (130–132)	118.2 86.7 86.7 86.5 86.5 86.5 86.5 86.5 86.5 86.5 86.5
	Diarrhea and enteritis under 2 years (119)	111.2         284.4         120.8         40.1         4.0         1           112.2         285.2         112.6         40.0         3.6         9           94.1         245.2         112.6         40.0         3.6         9           94.1         245.2         112.6         40.0         3.6         9           94.1         245.2         112.6         40.0         3.6         9           78.1         114.6         79.6         30.7         5.1         7           78.1         168.2         110.6         44.7         5.1         7           78.1         100.2         307.1         84.6         4.7         5.1           111.0         344.1         80.0         (*)         3.2         9           100.2         307.1         84.6         7         7.9         4.9           100.2         307.1         84.6         7         7.9         4.9           100.2         307.1         84.6         7         7.9         8.6           100.2         307.4         84.6         7         7.9         8.6           100.2         316.4         84.8         5.1 <td< td=""></td<>
	Diseases of the digestive system (115-129)	1285 000 1284 1285 000 1284 1285 000 1284 1285 000 1284 1285 000 1284 1285 000 1284 1285 000 1285 000 1285 1285 000 1285 000 1285 000 1285 1285 0000 1285 0000 1285 000000000000000000000000000000000000
	Pneumonis, all forms (107-109)	120.8 111.4 111.6 111.6 90.0 80.0 84.5 84.5 84.5 84.5 84.5 84.5 84.5 84.5
asis)	Diseases of the heart (90–95)	284. 4 252. 9 245. 2 245. 2 245. 2 173. 4 173. 4 173. 4 173. 4 375. 9 375. 9 249. 4 210. 3 210. 3 210. 3
Desth rate per 100,000 population (annual basis)	Cerebral hemorrhage, embolism, and throm- bosis (83a, b)	
ion (an	Diabetes mellitus (61)	23.22 29.22 29.22 29.23 20.23
opulati	Cancer, all forms (45–55)	$ \begin{array}{c ccccccccccccccccccccccccccccccccccc$
0,000 p	Tuberculosis, all forms (13-22)	56 56 50 50 50 50 50 50 50 50 50 50 50 50 50
per 10	Cerebrospinal (meningo- coccus) meningitis (6)	1.3% 91-1% 92-00 
th rate	Acute infectious enceph- alitis (lethargic) (37)	0.0 0.411 0.00 4.00 1.00 0.411 0.00 4.00 1.00
Dea	Acute poliomyelitis (36) polioencephalitis (36)	<b>3</b> 31. <b>1</b> . <b>3</b> . <b>1</b> . <b>1</b> . <b>1</b> . <b>3</b> . <b>1</b> . <b>1</b> . <b>1</b> . <b>3</b> . <b>1</b>
	(SS) (9qqirg) szn9uftal	63. 27.9 27.4 27.9 2
. · · ·	Diphtheris (10)	Ning Ni⊣n 00ml
	(9) dguce guiqoodW	7.3     7.8     4.6     0.6     0.7     0.4     4.9       7.5     7.3     5.6     1.2     5.3     5.6     1.2     3.7       7.4     5.8     1.2     5.8     1.2     5.8     7.7       7.4     5.8     1.2     6.2     2.1     3.8       7.0     4.8     7.3     5.6     1.2     3.8       7.4     6.8     1.7     14.6     1.7     10.7       7.0     42     3.8     7.1     1.7     1.8     2.6       7.0     42     3.8     7.1     1.7     1.8     2.6       7.0     42     3.8     7.1     1.4     1.6     2.6       7.1     2.5     3.1     1.1     1.6     2.6       7.1     5.8     7.1     1.7     7.8     2.6       7.1     5.8     1.7     1.7     1.6     2.6       7.1     5.8     1.7     1.7     1.6     2.6       7.1     5.8     1.7     1.7     1.6     2.6       7.1     5.8     1.7     1.7     1.6     2.6       7.1     7.1     7.7     7.7     2.7     2.6       8.9     7.1     7.7 </td
	Scarlet fever (8)	448 811 800 448 811 800
	Measles (35)	7 33 33 33 9 14.6 31.1 14.6
	Typhoid fever (1-2).	00000000000000000000000000000000000000
Rate per 1,000 live births	Maternal mortality	400 044 000 000
	Total infant mortality	855± 4555 8883 3378 855± 4555
l basis) tins) per	Births (exclusive of stilling sunns) noitslupped 000,1	17. 17. 17. 19. 19. 19. 19. 19. 19. 19. 19. 19. 19
	All causes, rate per 1,000 tion (annual basis	9384 1112 1112 1112 1112 1112 1112 1112 11
	State and period	Virginia: 1940 - 112 4 1940 - 111 5 1839 - 111 5 1839 - 111 5 1839 - 111 5 1840 - 111 5 1841 - 111 5 1841 - 111 5 1841 - 111 5 1843 - 111 5 1843 - 111 5 1848 - 1

Includes all States with data for the 3-month period of 140, 1939, and 1938. The District of Columbia is included as a staten from the April 1399 and April 1940, 77,305,000.
 These data are taken from the April 1399 and April 1940 Statistical Bulletins published by the Metropolitan Life Insurance Co. All figures are provisional and are subject to correction, since they are based on provisional estimates of lives exposed to risk. Data do not include all diseases reported to the Public Health Service.
 Excludes pricarditis, acute endocarditis, and eaute mycoarditis.
 Excludes collisions between automobiles and trains or street cars.
 Excludes collisions between automobiles and trains or street cars.
 Datamary and Pebruary only.
 Datamary and Pebruary only.
 Datamary and Pebruary only.
 Datamary and Pebruary only.

# **COMFORT DURING HOT WEATHER**<sup>1</sup>

During the summer months many people are concerned about safeguarding health and increasing personal comfort and efficiency. The observance of the following procedures will do much to lessen the discomfort ordinarily experienced during the hot season. Feed.

The influence of a warm climate on the amount of food required by an individual is commonly exaggerated. The temperature of the body is adjusted not so much by increasing or diminishing the amount of heat we produce, as by regulating the amount of heat lost. It is therefore desirable during hot weather to increase the intake of fluids which will promote sweating, a mechanism by which the skin is cooled. Fresh fruits and vegetables are excellent sources of fluid and in addition contain food elements much needed by the body during hot weather. As a general consideration fried foods and rich pastries should be curtailed as foods of these types tend to increase heat production.

# Drink.

Attention has already been called to the necessity of drinking adequate amounts of water (6 to 8 glasses a day) to induce sweating. Fruit juices are excellent hot weather drinks, being palatable and effective in quenching thirst. Avoid large amounts of sweetening and the excessive use of alcohol.

When on motor trips drink only from wells and springs approved by the health department. In many States, the State health department has signs posted denoting safe water supplies. When in doubt it is advisable to inquire of local authorities.

When sweating is profuse a large amount of sodium chloride is lost. When excessive, the loss of fluid and of chlorides from the blood may lead to heat cramps and to heat exhaustion. It is believed that these conditions may be prevented by the drinking of an occasional glass of water to which a small amount of table salt has been added. Three or four grains of salt to a pint of water should be sufficient.

## Clothing.

The weight, texture, and color of the clothing have a great influence on the loss of heat through the evaporation of moisture from the skin. A safe and comfortable body temperature is maintained by free evaporation of sweat from the surface of the body. To aid in such evaporation, the clothing should be loose and of such character as to permit the easy passage of air. Materials such as cotton or linen aid most in avoiding the burning effect of the hot sun. It should be remembered

<sup>&</sup>lt;sup>1</sup> This material is available in leaflet form and may be obtained by addressing the Surgeon General, U. S. Public Health Service, Washington, D. C.

that dark colors absorb the sun's rays and are, therefore, warm in hot weather. White clothes reflect the rays of the sun and are cool in hot weather.

# Exercise.

Light exercise adapted to your own strength and condition of health is preferable. All forms of active physical exercise immediately before or after meals should be avoided. Swimming is one of the best sports for the hot weather since it does not cause overheating of the body.

Refrain from all strenuous exercise during the hottest part of the day. Sleep.

A comfortable night's rest during the severe heat waves of the summer will make the next day's heat seem less oppressive and maintain good health. The use of an oscillating electric fan which keeps air in motion without harmful direct drafts will help to insure a good night's sleep.

It is particularly beneficial to observe regular hours of sleep during the summer.

## Bathing.

Frequent bathing helps to keep the body cool and refreshed. The shower bath is recommended as it does not have the sedative and weakening effects of the long tub bath.

# Exposure to Sun.

It is best to begin with brief exposure each day until the skin becomes lightly tanned, after which the body may be exposed to the rays of the sun for longer periods. Persons with sensitive skins should be especially careful as overexposure to direct sun rays may cause severe burns.

To be comfortable during hot weather, live sensibly, form regular habits of living, get plenty of rest, and above all try to acquire a cheerful and philosophical outlook on life.

# **COURT DECISION ON PUBLIC HEALTH**

License for sale of soft drinks.—(Minnesota Supreme Court; State v. Comer, 290 N.W. 434; decided February 2, 1940.) The defendant was found guilty in the trial court of selling soft drinks without first procuring a license from the division of hotel inspection of the Minnesota Department of Health.

The facts were that the defendant, who operated a gasoline filling station, sold at his station, without having a license to do so, soft drinks which were consumed by the purchaser directly from the original bottle, no drinking glasses or other conveniences being provided. In addition to the soft drinks there were retailed at the station more than 40 different articles for automobile and household use.

The State statutes required the procuring of a license from the division of hotel inspection by a person operating a place of refreshment, which included a place where drinks were sold or served at retail. The statutes provided, however, that a general merchandise store or grocery store retailing or serving soft drinks should not be deemed a place of refreshment if such soft drinks were sold and delivered to the public in an original container and the purchaser thereof consumed the contents directly from the original container.

The supreme court, in passing on the matter on appeal, agreed with the defendant's contention that he did not conduct a place of refreshment within the meaning of the statutes and, consequently, did not have to have a license. The court took the view that "the present exempting provision is broad enough to include within the category of 'general merchandise store' a filling station retailing more than 40 different articles and dealing in commodities for both automobile and home use." "The term 'general merchandise store,'" said the court, "must be understood in a broad rather than a restricted sense so that constitutionality can be preserved."

DEATHS DURING WEEK ENDED JULY 13, 1940

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

_	Week ended July 13, 1940	Correspond- ing week, 1939
Data from 88 large cities of the United States:         Total deaths.         Average for 3 prior years.         Total deaths, first 28 weeks of year.         Deaths under 1 year of age.         Average for 3 prior years.         Deaths under 1 year of age, first 28 weeks of year.         Deaths under 1 year of age, first 28 weeks of year.         Deaths under 1 year of age, first 28 weeks of year.         Deaths under 1 year of age, first 28 weeks of year.         Death form industrial insurance companies:         Policies in force.         Number of death claims.         Death claims per 1,000 policies in force, annual rate.         Death claims per 1,000 policies, first 28 weeks of year, annual rate.	7, 927 8, 126 246, 419 448 525 14, 117 65, 102, 755 11, 048 8, 9 10, 1	7,670 244,231 471 14,497 67,044,842 11,529 9.0 10.9

# **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**

#### **REPORTS FROM STATES FOR WEEK ENDED JULY 20, 1940**

#### Summary

For the week ended July 20, 1940, decreases were noted in the incidence of diphtheria, influenza, measles, scarlet fever, smallpox, whooping cough, and Rocky Mountain spotted fever as compared with the preceding week. Slight increases were noted in the incidence of meningitis, poliomyelitis, typhoid fever, and endemic typhus fever. Of the nine communicable diseases appearing in the following table, only measles was higher than the seasonal expectancy, based on the 1935–39 median.

The trend of poliomyelitis continued upward, conforming closely to the seasonal pattern, but with an appreciably lower incidence than the corresponding weekly median. The current incidence of 119 cases compares favorably with the report of 137 cases for the corresponding week last year.

A total of 345 cases of typhoid fever was reported as compared with 238 for the preceding week and with a 1935–39 median figure of 548 cases. The principal increases over the preceding week were noted in the South Atlantic and the South Central States.

A total of 33 cases of meningitis was scattered among 20 States. While this was a slight increase over the 22 cases reported for the preceding week, the accumulated total for the first 29 weeks of the current year is 1,025 cases as compared with 1,259 for the first 29 weeks of 1939. If the present trend continues, 1940 will prove to be the lowest year on record since weekly reports of meningitis cases have been available.

# Telegraphic morbidity reports from State health officers for the week ended July 20, 1940, and comparison with corresponding week of 1939 and 5-year median

In these tables a zero indicates a definite report, while leaders imply that, although none were reported, cases may have occurred.

	D	iphthe	ria		Influenz	<b>:8</b>		Measle	8	M	eningi	tis. ccus
Division and State	Week	ended	Me	Week	ended		Week	ended			ended	Me-
	July 20, 1940	July 22, 1939	dian, 1935- 39	July 20, 1940	July 22, 1939	Me- dian, 1935–39	July 20, 1940	July 22, 1939	Me- dian, 1935-39	July 20, 1940	July 22, 1939	dian, 1935– 39
NEW ENG.												
Maine New Hampshire Vermont Massachusetts Rhode Island Connecticut	2 0 7 0 7 0 0	1 0 5 0 1	1 0 5 0 2		  1	  1	93 2 12 605 41 36	25 2 29 207 29 49	25 2 23 167 19 41	1 0 0 1 0	0 0 0 1 0	0 0 1 0 0
MID. ATL.												
New York New Jersey Pennsylvania	5 6 3	11 1 10	16 5 23	1	13 3	13 2 	486 273 201	491 15 61	660 171 277	2 1 2	1 2 6	4 1 4
E. NO. CEN.												
Ohio Indiana <sup>3</sup> Illinois <sup>4</sup> Michigan <sup>3</sup> Wisconsin	8 4 14 3 2	13 2 19 5 2	13 7 19 10 2	4 3 4 6	2 8 2 2	3 8 6 	21 4 123 241 390	7 8 15 73 124	127 16 58 115 124	1 2 0 2 0	0 1 2 2 0	2 1 2 1 0
W. NO. CEN.												
Minnesota Iowa Missouri <sup>3</sup> North Dakota <sup>3</sup> Nebraska Kansas	1 0 4 0 1 0 6	0 1 5 0 1 2	5 6 5 0 1 2	1 2 	1   1	 11  8	23 53 11 0 3 3 41	17 64 1 2 8 2 21	25 18 10 2 1 7 17	1 1 0 0 0 0	0 0 0 0 0 1	0 1 1 0 0 0 1
80. ATL.												
Delaware <sup>3</sup> Maryland <sup>3 3 4</sup> Dist. of Columbia Virginia <sup>3</sup> West Virginia <sup>3</sup> North Carolina <sup>4</sup> South Carolina <sup>4</sup> Georgia <sup>3</sup> 4 Florida <sup>4</sup>	0 1 2 13 3 1 3 1 0	0 1 10 9 11 3 8 4	0 5 8 3 11 3 8 4	2 17 1 46 9	1 20 10 4 110 25 4	2 10 58	0 5 1 45 6 51 3 9 5	4 10 14 47 3 32 3 6 7	4 25 14 47 11 32 3 0 7	0 0 5 0 1 0 0	0 0 0 1 0 1 0	0 2 0 2 2 2 1 1 0
E. 80 CEN.												
Kentucky Tennessee <sup>1</sup> Alabama <sup>4</sup> Mississippi <sup>3</sup>	8 1 5 1	2 2 7 7	2 5 11 8	4 14	20 7	14 7	24 25 76	4 3 26 0	50 19 6	2 0 4 0	1 2 2 1	2 2 2 0
<b>W. 80. CEN.</b>												
Arkansas Louisiana <sup>4</sup> Oklahoma Texas <sup>4</sup>	8 2 8 12	3 7 1 10	3 9 4 23	1 4 2 56	10 14 4 81	3 13 6 81	5 3 6 118	8 36 5 54	6 4 7 54	0 0 1 1	1 0 0 1	0 1 0 1
MOUNTAIN												-
Montana Idaho Wyoming <sup>3</sup> Colorado New Mexico Arizona Utah <sup>3</sup>	2 0 10 0 6 0	0 0 13 2 0 0	0 0 4 1 1 0	1 1 3 2 25	8 6 13	10	7 8 2 29 21 14 37	42 2 4 9 1 6 18	33 9 5 20 4 7 23	2 0 0 0 1 0	0 0 0 0 0 0	0 1 0 1 0 0 0

Telegraphic morbidity reports from State health officers for the week ended July 20, 1940, and comparison with corresponding week of 1939 and 5-year median— Continued

	D	iphthe	ria		Influen	4		Measle	8	M me	feningi ningoco	tis, ocus
Division and State	Week	ended	Me-	Week	ended	Me-	Week	ended	Me-	Weel	k ended	Me-
	July 20, 1940	July 22, 1939	dian, 1935– 39	July 20, 1940	July 22, 1939	dian, 1935–39	July 20, 1940	July 22, 1939	dian, 1935–39	July 20, 1940	July 22, 1939	dian, 1935– 39
PACIFIC												
Washington			1 2		6	7	22 38	209 36			0	0
California	15		23	11	Ť	<u>1i</u>	91	315		ľ	ľ	2
Total	114	213	301	220	318	238	3, 308	2, 154	2, 801	33	27	50
29 weeks	8, 194	10, 879	13, 097	167, 533	150, 548	140, 981	220, 675	344, 403	344, 403	1, 025	1, 259	3, 860
	Pol	iomyel	litis	Sc	arlet fev	ver	· 8	Smallpo	r	Typh typ	oid and bhoid fe	l para- ver
Division and State	Week	ended	Me-	Week	ended	Me-	Week	ended	Me-	Week	ended	Me-
	July 20, 1940	July 22, 1939	dian, 1935– 39	July 20, 1940	July 22, 1939	dian, 1935–39	July 20, 1940	July 22, 1939	dian, 1935–39	July 20, 1940	July 22, 1939	dian, 1935 39
NEW ENG.												
Maine New Hampshire Vermont Massachusetts Rhode Island Connecticut	0 0 1 0 1	0 0 1 0 1	0 0 3 0 1	5 3 4 29 2 12	2 1 2 31 8 14	4 1 3 53 8 7	0 0 0 0 0	0 0 0 0	000000000000000000000000000000000000000	0 0 1 0 2	4 0 2 3 0	2 1 0 2 0 1
MID. ATL.	-	-				· ·	Ĩ	Ĩ	Ŭ		Ů	-
New York New Jersey Pennsylvania	4 0 1	7 1 5	7 1 1	134 55 101	75 24 94	84 24 114	0 0	0 0 0	0 9 0	14 5 15	10 3 15	11 5 16
E. NO. CEN.												
Ohio Indiana <sup>3</sup> Illinois <sup>4</sup> Michigan <sup>3</sup> Wisconsin	1 1 1 8 5	2 0 6 17 0	2 1 2 4 0	80 30 100 61 38	40 26 63 76 30	40 22 102 82 56	0 2 1 0 2	4 4 3 1	1 4 10 1 3	3 7 15 3 0	9 11 11 6 1	9 9 18 6 1
W. NO. CEN.						1						
Minnesota Iowa Missouri <sup>a</sup> North Dakota South Dakota <sup>a</sup> Nebraska Kansas	0 6 1 0 2 8	1 0 2 1 0 1 0	0 1 2 0 0 1 0	17 12 21 4 5 3 20	19 9 10 3 17 3 18	29 19 13 7 8 4 18	7 3 1 3 3 0 0	0 8 3 4 3 2 0	4 9 3 4 3 2 4	0 2 12 0 0 0 6	1 4 22 0 1 0 2	1 3 21 1 0 9
80. ATL.												
Delaware <sup>2</sup>	0 1 0 2 4 1 0 1 0	0 0 1 2 8 12 5 8	0 0 2 0 3 1 2 0	0 9 4 12 18 11 2 8 0	2 10 0 15 13 20 0 13 3	1 10 3 13 11 11 2 5 3	0 0 0 0 0 0 0 0	000000000000000000000000000000000000000	0 0 0 0 1 0 0	1 3 0 4 3 12 15 24 4	1 6 3 10 12 25 30 20 5	1 11 2 18 12 25 23 43 5

•

Telegraphic morbidity reports	from State health officers	for the week ended July 20,
1940, and comparison with	corresponding week of	1939 and 5-year median-
Continued		

	Po	liomye	litis	8	carlet fe	ver	'	Smallpo	I	Typl tyj	noid an phoid f	d para- ever
Division and State	Week	ended	Me-	Week	ended	Me-	Week	ended	Me-	Week	ended	Me
	July 20, 1940	July 22, 1939	dian, 1935- 39	July 20, 1940	July 22, 1939	dian, 1935–39	July 20, 1940	July 22, 1939	dian, 1935-39	July 20. 1940	July 22. 1939	dian, 1935– 39
E. 50. CEN.										'		
Kentucky Tennessee <sup>1</sup> Alabama <sup>4</sup> Mississippi <sup>1</sup>		1 1 0 1	2 3 1 8	18 10 6 5	10 12 8 0	12 11 8 6	0 0 1 0	0 0 0	000000000000000000000000000000000000000	11 11 8 6	80 28 6 9	89 28 15 14
W. 80. CEN.												
Arkansas. Louisiana <sup>4</sup> Oklahoma. Texas <sup>4</sup>	0 7 2 8	1 1 0 7	1 3 0 2	2 1 5 17	0 6 1 15	2 4 11 23	0 0 0	0 0 2 1	0 0 0 1	53 28 9 43	26 43 24 52	26 25 27 52
MOUNTAIN												
Montana Idaho Wyoming <sup>1</sup> Oolorado New Mexico Arizona Utah <sup>1</sup>	4 1 0 1 0 1	0 0 2 1 0 0	0 0 0 1 0	6 2 1 9 3 4 6	11 0 0 17 7 4 10	5 8 17 7 8 10	0 0 3 0 1 0	0 2 2 0 1 0	4 2 1 1 0 0 0	0 3 0 2 2 5 2	0 0 2 5 3 2 0	1 0 1 4 3 2 0
PACIFIC												•
Washington Oregon California	18 2 15	0 0 51	0 0 21	14 13 38	10 4 68	11 10 7 <del>3</del>	0 1 1	0 2 0	0 2 1	1 4 6	2 2 7	2 8 7
Total	119	137	137	960	814	1,002	29	46	85	345	464	548
29 weeks	1, 064	1, 157	1, 157	116,252	113,489	161,216	1, 872	8, 500	7, 693	8, 444	5, 065	5, 496

.

Telegraphic morbidity repo	ris from	State heal	th officers	for the we	ek ended July 20,
1940, and comparison	with corr	esponding	week of	1939 and	5-year median-
Continued			•		•

	Whoop	ng cough		Whoopi	ng cough	
Division and State	Week	ended	Division and State	Week	ended	
	July 20, 1940	July 22, 1939		July 20, 1940	July 22, 1939	
NEW ENG.			80. ATL-continued			
Maine	32	6			1	
New Hampshire	0	5	South Carolina 4	11	2	
Vermont	8	30	Georgia <sup>3 4</sup> Florida <sup>4</sup>	40	8	
Massachusetts	132	124	Florida 4	8	i š	
Rhode Island	5	15			-	
Connecticut	45	51	<b>E. 80. CEN.</b>			
MID. ATL.			Kentucky	83	4	
			Tennessee 1	48	6	
New York	205	356	Alabama 4	21	7	
New Jersey	79	291	Mississippi 1			
Pennsylvania	899	618	W. 80. CEN.			
E. NO. CEN.						
1			Arkansas	47	1	
Dhio	387	103	Louisiana 4		2	
ndiana 1	80	189	Oklahoma	19	:	
llinois 4	126	363	Texas 4	253	8	
Michigan <sup>*</sup>	235	269				
Wisconsin	83	262	MOUNTAIN			
W. NO. CEN.	1		Montana	8	(	
finnesota			Idaho	3	1	
linnesota		22	Wyoming *	9	(	
owa	27	33	Colorado	15	2	
fissouri 1	69	49	New Mexico	38	19	
Jorth Dakota	10	7	Arizona	13	1	
outh Dakota 1	10	2	Utah 3	87	4	
lebraska	7 53	87 15	PACIFIC			
					-	
80. ATL.			Washington	45	2	
elaware !	12	. 3	Oregon California	19	2	
forviand \$14	163	57		276	13	
faryland <sup>2 2 4</sup> Dist. of Col	103	37	Total	3.426	1 00	
irginia <sup>2</sup>	36	107	1.0641	3, 420	4, 06	
Vest Virginia <sup>3</sup> forth Carolina <sup>4</sup>	51	26	29 weeks	93. 427	112 40	
	146	239	40 WUVAJ	90, 44/	113, 405	

New York City only.
 Rocky Mountain spotted fever, week ended July 20, 1940, 15 cases, as follows: Indiana, 2; Missouri, 1; South Dakota, 1; Delaware, 1; Maryland, 4; Virginia, 3; Georgia, 1; Tennessee, 1; Wyoming, 1.
 Period ended earlier than Saturday.
 Typhus fever, week ended July 20, 1940, 51 cases, as follows: Illinois, 1; Maryland, 1; North Carolina, 3; Bouth Carolina, 1; Georgia, 17; Florida, 7; Alabama, 8; Louisiana, 1; Texas, 12.

# VENEREAL DISEASES

# New Cases Reported for May 1940<sup>1</sup>

## Reports from States

				i	Syphil	ls				Gono	rrhea		vene- seases
		Early		L	ate	Cong	enital	Allsy	ohilis <sup>3</sup>		-ndo		-ndo
	Primary and secondary	Early latent 3	Rate per 10,000 population	Includes late- latent	Rate per 10,000 population	Number	Rate per 10,000 population	Number	Rate per 10,000 population	Number	Rate per 10,000 popu- lation	Number	Rate per 10,000 popu- lation
Alabama Alaska 4	231	338		406				1, 851	6. 32		1.12		0. 02
Alaska 4 Arizona Arkansas California Colorado Connecticut Delaware Dist. of Columbia	88 11 6	233 	2.22 .63 .82 .14	43 433 1, 505 169 92 6	2.09 2.41 1.57 .53	92 22 12	. 20	2 135	2.32 5.64 3.41 2.59 .94 5.97 10.69	87 229 1, 634 120 128 39 268	.89 1.10 2.61 1.11 .73 1.48 4.21	12 34 	. 06
Florida Georgia Hawaii Idaho Illinois Indiana	165  18 106 105	806 1 387 90	2.59 .15 .36 .62 .56	• 17 1, 425 270	. 59 . 34 1. 80 . 77	6 4 107 38	. 44 . 15 . 08 . 14 . 11	1, 834 1, 615 48 44 2, 025 724	10, 79 5. 19 1. 19 . 88 2. 56 2. 07	143 108 57 5 1, 360 147	.84 .35 1.41 .10 1.72 .42	8  19	.03 .03 .02
Kansas. Kentucky. Louisiana Maine 4 Maryland.	36 48 83 362 103	36 24	. 36	103 72 311  210	. 40 . 39 1. 05 	11	.04 .06 .05 .01	198 239 644 697 919	.77 1.28 2.18 3.25 5.45	129 91 344 87 	.50 .49 1.16 .41	1	.01 .01 .03 .19
Massachusetts Michigan <sup>4</sup> Minnesota Mississippi Missouri	74  302 179	20 830 370	. 17 . 10 5. 45 1. 34	417 182 749 279	. 94 . 68 3. 67 . 67	27	.06 .04 .49 .06	518 222 4, 835 878	1. 17 . 83 23. 70 2. 18	313 168 2, 392 159	.71 .63 11.73 .40		. 02
Montana Nebraska Newada New Hampshire New Jersey New Mexico	16 38  90 33	4	. 29 . 31 . 39 . 53 1. 02	17 60 13 10 484 110	.31 .44 1.27 .20 1.11 2.61	7 1 1 61 23	.05 .10 .02 .14 .55	36 109 18 22 826 192	. 66 . 80 1. 76 . 43 1. 89 4. 55	19 66 15 10 229 49	. 35 . 48 1. 47 . 20 . 52 1. 16	1 	. 01
New York North Carolina North Dakota Obio Oklahoma	352 271 4 208 149 36	332 956 6 229 203	. 53 3. 48 . 14 . 65 1. 33 . 71	2, 611 1, 008 5 892 350 91	2.01 2.86 .07 1.32 1.36 .88	23 160 93 1 96 33 6	. 12 . 20 . 01 . 14 . 13 . 06	3, 744 2, 328 25 1, 425 952 172	2.88 6.59 .35 2.11 3.70 1.66	1, 445 458 23 97 238 125	1. 11 1. 30 . 22 . 14 . 93 1. 20	81 84 	. 02 . 10
Oregon Pennsylvania 4 Rhode Island South Carolina South Dakota Tennessee	7 645 85 223	10 448 61 382	. 25 5. 78 2. 11 2. 07	96 686 37 591	1.41 3.63 .53 2.02	8 29 9 61	.04 .15 .13 .21	117 1, 847 196 1, 260	1. 72 9. 76 2. 83 4. 31	27 40 16 321	.40 .21 .23 1.10	1	.01
Texas Utah Virginia Washington West Virginia Wisconsin Wirgonia	900	485 10 8 262 22 111	1.31 .44 .26 2.04 .34 1.98	798 46 4 738 101 139	1.28 .88 .10 2.69 .60 .73	92 5 1 48 4 24	.15 .10 .03 .17 .02 .13	2,068 75 15 1,467 192 800	3. 32 1. 44 . 39 5. 35 1. 15 4. 20	865 19 15 219 193 258	1.39 .36 .39 .80 1.15 1.36	4	.08
West Virginia Wisconsin Wyoming Puerto Rico 4 Virgin Islands 4	205 8 11	26 3	1.90 .12 .59	139 122 18	. 41 . 76	5 4	. 02 . 17	161 40	1.69	67 16	.23 .68	1 2	. 003
Virgin Islands 4 Total	5, 681	7, 497	1. 16	17, 515	1. 54	1, 487	. 13	40, 044	8. 49	 13, 401	1. 17	336	. 04

# 1362

				1	Syphil	5				Gond	orrhea		r vene- iseases
		Early		L	ate	Cong	enital	All sy	philis		-ndo		-ndo
	Primary and secondary	Early latent	Rate per 10,000 population	Includes late- latent	Rate por 10,000 population	Number	Rate per 10,000 population	Number	Rate per 10,000 population	Number	Rate per 10,000 popu- lation	Number	Rate per 10,000 popu- lation
Akron Atlanta Baltimore Birmingham Boston Buffalo Chicago	11 85 89 29 12 79	20 250 18 40  2 198	1. 13 8. 33 1. 23 4. 38 . 36 . 23 . 76	106 172 61	3. 53	 8 20	. 10 . 68 . 09 . 13	356 574 418 178 121	2.95 11.86 6.87 14.20 2.24 2.01 3.35	23 62 187 62 101 48 947	0.84 2.06 2.24 2.11 1.27 .80 2.58	 30 3  19	. 10
Cincinnati 4 Cleveland Columbus Dallas Dayton Denver Detroit	31 23 48 10 39 27	36 33 49 10 	.71 1.79 3.19 .90 .57 2.51	181 74 156 46 	1.92 2.36 5.13 2.07 1.28 2.87	14 2 2 2 12 10	. 15 . 06 . 07 . 09 . 07 . 07	132 255 68 104 347	2.77 4.21 8.39 3.07 3.45 1.91 9.54	102 15 153 27 69 267 157	1.08 .48 5.03 1.22 2.29 1.47 4.38	9 14 2 21 21	.46 .09 .12
Houston Indianapolis. Jersey City Kansas City 4 Lou SAngeles. Louisville. Memphis 4	15 3  15	4 4 129 2	. 49 . 22 . 85 . 50	17 17 591 100	. 44 . 52 3. 88 2. 95	1	.03 .12 .16 .09	134 32 745 196	3. 48 . 99 4. 90 5. 78	31 4 449 68	. 80 . 12 2. 95 2. 01		
Milwaukee Minneapolis Newark New Orleans 4	1 1 14	7 26 6	. 13 . 54 . 44 . 79	62 44 179 1, 748	.98 .88 3.94 2.33	4 7 91	.08 .15 .12	277	1. 11 1. 50 6. 10 	7 48 71 1.060	. 11 . 96 1. 56 		
New York Oakland Omaha Philadelphia <sup>4</sup> Pittsburgh	262 5 	332 12 	. 79 . 38 . 22	1, 748 70 7	2. 33 2. 24 . 31	8 8 1	. 12 . 26 . 04	2,707 90 13 	3. 01 2. 87 . 58 4. 88	1,000 51 13 26	1. 41 1. 63 . 58	01 	.01
Portland Providence Rochester St. Louis St. Paul	9 5 1 33	18 4 1 167	. 84 . 35 . 06 2. 37	52 69 13 324	1.62 2.66 .38 3.84	3 2 1 13 	.09 .08 .03 .15	82 80 16 537 26	2.56 3.08 .47 6.37 .90	53 20 31 147 22	1.65 .77 .91 1.74 .77	9	.11
San Antonio 4 San Francisco Beattle Syracuse Toledo Washington, D. C	47 13 2 8	20 1 4	. 68 . 85 . 13 . 39	151 92 61 45	2. 19 2. 38 2. 71 1. 45	4 1 5 1	. 06 . 03 . 22 . 03	202 129 69 58 680	2.93 3.33 3.06 1.86 10.69	211 140 6 17 268	3.06 3.62 .27 .55 4.21	6 3 4	. 09 . 08 . 06
Total	917	1, 520	. 93	5, 925	2. 25	314	. 12	11, 027	3. 91	4, 963	1. 76	158	. 08

## Reports from cities of \$00,000 population or over

Figures preliminary and subject to correction.
Includes "not stated" diagnosis.
Duration of infection under 4 years.
No report for current month.
Includes early latent of less than 1 year's duration.
Includes early latent, late, and late latent.

## WEEKLY REPORTS FROM CITIES

## City reports for week ended July 6, 1940

This table summarizes the reports received weekly from a selected list of 140 citics for the purpose of showing a cross section of the current urban incidence of the communicable diseases listed in the table.

State and city	Diph- theria	Inf	uenza	Mea-	Pneu- monia	Scar- let	Small-	Tuber- culosis	Ty- phoid	Whoop- ing	Deaths, all
State and city	cases	Cases	Deaths	cases	deaths	fever cases	pox cases	deaths	fever cases	cough cases	causes
Data for 90 cities: 5-year average Current week <sup>1</sup> .	106 28	29 21	14 9	1, 840 2, 015	322 197	589 472	9 0	363 292	52 42	1, <b>296</b> 871	
Maine: Portland			0	15	1	0	0	2	1	4	26
New Hampshire:					1.1				0	0	12
Concord Manchester	0		0	0	0	0	0	0	0	Ó	16
Nashua	0		0	0	0	0	0	0	0	0	5
Vermont: Barre											
Burlington	0		0	0	0	0	0	0	0	0	95
Rutland Massachusetts:	U			, v	-				-		· ·
Boston	0		0	118 78	5	21 0	0	6	1	29 10	199
Fall River Springfield	ŏ		ŏ	12	Ō	ő	0	0	Ó	1 1	26 33
Worcester	0		0	174	8	0	0	8	0	3	42
Rhode Island: Pawtucket	0		0	0	0	1	0	0	0	0	13
Providence	0		0	48	2	2	0	1	0	8	44
Connecticut: Bridgeport	0		0	2	0	1	0	0	0	0	19
Hartford	0		0	0	0	22	0	1 2	0	02	34 32
New Haven	0		, v	1	-	-	v	<b>^</b>	U	<b>1</b>	<u>م</u> د
New York:	0		0	3	8	15	0	4	0	6	130
Buffalo New York	9 9	4	ŏ	298	31	92	Ö	65	3	98	1,257
Rochester	0		0	3	2	0 2	0	1	0	3	68 41
Syracuse New Jersey:	0		0	1	0	Z	0	1			
Camden	0		0	2	2	5	0	1	0	0	32
Newark Trenton	0	1	0	185 0	0	8	0	5 2	0.	18 0	86 31
Pennsylvania:	-							17	7	~	380
Philadelphia Pittsburgh	1	1	2 1	150 2	10 9	28 2	0	5	ó	23 13	137
Reading	Ő		Ō	2	Ō	0	0	1	0	21 1	16
Scranton	0			1		0	0		0	1	
Ohio:									1	13	93
Cincinnati Cleveland	1		0	4	2	5	0	2	1	19	
Columbus	0		0	1	0	8	0	1	0	3	56
Toledo Indiana:	0		0	2	4	6	0	2	0	16	57
Anderson	2		0	0	0	0	0	0	0	0	3
Fort Wayne Indianapolis	0		0	2200	1	0	0	04	0	0 5	23 90
Muncie	Ō		0	õ	2	0	0	Ó	8	1	11
South Bend	0		Ó	0	0	02	0	0	Ŏ	0 1	8 22
Illinois:	-			-							
Alton	0 5	i	0	0 169	0 11	ө 115	0	1 23	0	0 38	8 555
Chicago Elgin	0		2	1	1	0	0	0	ě	0	22
Moline	0		0 0	5	0 1	0 1	0	0	0	0	6 28
Springfield Michigan:			-	-	-			-			
Detroit	1		0	239 1	18 1	33 3	0	19 1	1	83 1	226 24
Flint Grand Rapids	ŏ		ŏ	6	ō	5	ŏ	ô	Ô	17	29
Wisconsin:	0		0	12	0	0	0	0	0	0	5
Kenosha Madison	0		Ó	29	0	0	0	0	0	3	7
Milwaukee Racine	Ő		0	253 6	4	8	0	8	0	9 1	103 16

<sup>1</sup> Figures for Barre, Cleveland, and Shreveport estimated; reports not received.

# City reports for week ended July 6, 1940-Continued

	Diph-	Inf	luenza	Mea-	Pneu-	Scar-	Small-	Tuber-	Ty-	Whoop-	Deaths,
State and city	theria cases	Cases	Deaths	ales cases	monia deaths	fever cases	pox cases	culosis deaths	fever cases	cough cases	all causes
Minnesota:											
Duluth	0		0	4	0	1	0	1	0	ļ	22 70
Minneapolis	<b>B</b>		e e e e e e e e e e e e e e e e e e e	0		8	0	1 2	0	4	70 60
St. Paul Iowa:	v ا			0	•	9	0		0	1.8	00
Cedar Rapids	0			1		0	0		0	0	
Davenport	0			1		0	0		0	0	
Des Moines	0		0	8	0	1	8	0	0	8	42
Sioux City Waterloo	0			0		1	0		0	2	
Missouri:	U			•		U			U	•	
Kansas City	0		0	4	4	2	0	2	1	1	82
St. Joseph	0		Ó	0	1	0	0	Ō	0	1	25 173
St. Louis	0		0	1	5	7	0	6	1	14	173
North Dakota:	0		0	0	0	0	0		0		2
Fargo Grand Forks	ŏ		v	ŏ	•	ŏ	ŏ	0	ŏ	0	
Minot	ŏ			ŏ		ŏ	ŏ		ŏ	ŏ	
South Dakota:				•		•	•		•		
Aberdeen	0			1		0	0		0	8	
Sioux Falls	0		0	0	0	1	0	0	0	0	7
Nebraska:	0		1.1.1	0		0	0		0	1	
Lincoln Omaha	ŏ		0	3	2	1	ŏ	2	ŏ	3	
Kansas:	v		v	J	<b>^</b>	- 1	v	•	•	J	
Lawrence	0		1	0	0	0	0	0	0	0	4
Topeka	0		0	12	6	1	0	0	0	0	17
Wichita	0		0	3	2	0	0	0	0	3	19
Delaware:											
Wilmington	0		0	0	0	0	0	1	2	1	28
Maryland: Baltimore	0	1	1	1	4	6	0	10	0	111	174
Cumberland	ŏ	- 1	ō	ō	ō	ŏ	ŏ	10	ŏ	111	1/3
Frederick	ŏ		ŏ	ŏ	i i	ŏ	ŏ	ŏ	ŏ	ŏ	
Dist. of Col.:	-		-	-		-					
Washington	1		0	2	1	11	0	7	0	3	123
Virginia: I			0		0			0		_	
Lynchburg Norfolk	0		ŏ	0	1	0	0	1	02	5 1	6 15
Richmond	ŏ		i	10 0	i	2	ŏ	3	2	ō	47
Rosnoke	ŏ		ō	13	ō	ō	ŏ	ŏ	ō	ž	10
West Virginia:								1			
West Virginia: Charleston	0		0	0	2	0	0	1	2	. 0	38
Huntington	0			0		2	0	ō-	0	0	
Wheeling North Carolina:	0		0	0	0	0	0		- 1	1	18
Gastonia	0	1	1	0		1	0		0	0	
Raleigh	ŏ		0	ŏ	0	ôl	ŏ	0	ŏ	3	15
wilmington	0		Ó	Ó	1	0	Ó	Ó	Ó	0	13
Winston-Salem.	0		0	0	1	0	0	2	0	0	21
South Carolina:				_				.	0		~ ~ ~
Charleston	0	2	0	0	4	0	0	1	ŏ	0	24
Florence Greenville	ŏ		ŏ	00	3	ŏ	ŏ	ŏ	ŏ	ŏ	9 23
Georgia:	Ŭ,		° I	۳	•	۳I	v I	v I	۳I	, v	~
Atlanta	0		0	7	1	1	0	6	0	10	64
Brunswick	0		0	0	0	0	0	0	0	0	2
Savannah	0	1		0	0	0	0	1	0	0	25
Florida: Miami	0		0	0	1	0	0	1	0	0	35
Tampa	ŏ		ŏ	4	ō	ŏ	ŏ	i	ĭ	2	28 28
Kentucky:	1								1		
Ashland	0		0	0	1	0	0	0	0	0	10
Asbland Covington	<b>0</b> į.		Ő	ŏ	1	ŏ	ŏ	ŏ	0	ŏ	
Lexington	0		0	21	1	0	0	1	0	7	14
Louisville	0		0	5	1	2	Ó	2	Ō	29	50
Cennessee:	0	1	0								
Knoxville Memphis	ŏ	- 1	ő	05	8	20	0	28	8	0	32 50
Nashville	ŏ		ŏ	2	ő	ŏ	ŏ	ő	ŏ	- 12	50 89
Alabama:	-			- 1		- 1				- 1	
Birmingham	0			1	1	8	0	5	1	5	67
3											
Mobile	0  - 0  -		Ó	40	0	2	8	2	8	0	20

	Diph- theria	Inf	uenza	Mea-	Pneu-	Scar- let		Tuber-	Ty- phoid	Whoop-	Dearns,
State and city	cases	Cases	Deaths	sles cases	monía deaths	fever cases	pox cases	deaths	former	cough cases	all causes
Arkansas:											
Fort Smith Little Rock Louisiana:	0		0	0	4	0	0	2	0	24	
New Orleans Shreveport	1		0	1	10	2	0	6	6	8	135
Oklahoma: Oklahoma City. Tulsa	0		0	0	22	3	0	0	0	1 9	46 12
Texas: Dallas	2		0	15	1	0	0	2	2	17	75
Fort Worth Galveston Houston	0		000000000000000000000000000000000000000	19 0 7	1 1 7	1 0 1	000000000000000000000000000000000000000		003	18 0 10	37 11 69
San Antonio	ŏ		ŏ	Ó	ó	Ō	ŏ	12	3	7	72
Montana: Billings	0		0	0	1	2	o		0	0	8
Great Falls Helena	0		0	2	1	0 1	0	0	0	-0 0	8 6 1
Missoula Idaho:	0		0	0	0	0	0	0	0	1	1 -
Boise Colorado: Color a do	0		0	1	.0	0	0	0	0	2	6
Springs Denver	03		0	0 5	1	1	0	02	0	0	10 74
Pueblo	ŏ		ŏ	ŏ	Ô	ŏ	ŏ	õ	ŏ	ŏ	5
Albuquerque Utah:	0		0	0	. 0	0	0	0	0	3	7
Salt Lake City_	0		0	46	0	2	0	0	0	60	33
Washington: Seattle	1		0	18	2	4	0	2	0	15	63
Spokane Tacoma	0		0	2 0	1 0	2 0	0	0	10	3 0	31 27
Oregon: Portland Salem	0		0	6 1	0	1	0	2	0	7	74
California: Los Angeles	1	3	1	5	3	6	0	11	0	59	255
Sacramento San Francisco	1 2 0	 1	0 0	0 0	3 2 0	1 6	0 0	11 3 10	000	59 7 17	255 25 142
	1	Menir	ngitis.	Deli					Meni	ngitis.	Delie
State and city	I		ococcus	Polio- mye-		State a	and city	.		ococcus	Polio- mye-

## City reports for week ended July 6, 1940-Continued

		Polio- mye-	State and city		Polio- mye- litis	
Cases	Deaths	Cases		Cases	Deaths	cases
0	0	ľ	Virginia: Norfolk.	0	0	1
0	0	2	Huntington	0	0	1
0 1	0 1	1 0	Greenville Oklahoma:	0	1	0
1	0	1	Texas:			1
1	0	0	Washington: Seattle	0	0	4
0	0	1	Tacoma California:	0	0	2
	mening Cases 0 0 1 1 1	0 0 0 0 1 1 1 0 1 0 0 0	meningococcus         Prono- inye- litis           Cases         Deaths           0         0           0         0           0         0           1         1           0         0           1         0           1         0           0         0           1         0           0         0	meningococcus         Prono- inve- litis         State and city           Cases         Deaths         Cases         State and city           0         0         1         Norfolk	meningococcus         Pono- mye- litis         State and city         mening           Cases         Deaths         cases         Cases         Cases         Cases           0         0         1         Norfolk	meningococcus         rono- inye- litis         State and city         meningococcus           Cases         Deaths         Cases         Cases         Cases         Deaths           0         0         1         Norfolk

Encephalitis, epidemic or lethargic.—Cases: New York, 1; Chicago, 1; Billings, 1; Missoula, 1. Pellagra.—Cases: Savannah, 1; Birmingham, 1; Los Angeles, 1. Typhus ferer.—Cases: Wilmington, N. C., 1; Charleston, S. C., 1; Savannah, 1; Miami, 1; New Orleans, 1. Deaths: Wheeling, 1; Miami, 1.

# **FOREIGN REPORTS**

# CANADA

Provinces—Communicable diseases—Week ended June 15, 1940.— During the week ended June 15, 1940, cases of certain communicable diseases were reported by the Department of Pensions and National Health of Canada as follows:

Disease	Prince Edward Island	Nova Scotia	New Bruns- wick	Que- bec	On- tario	Mani- toba	Sas- katch- ewan	Alber- ta	British Colum- bia	Total
Cerebrospinal meningitis. Chickenpox Diphtheria Dysentery.		8	14	1 79 26 11	1 463 1	62 2	110	22	1 52	8 785 81 11
Influenza Lethargic encephalitis Measles	1	17	2	72	10 1 131	103	159		6 71	16 1 559
Mumps Pneumonia Poliomyelitis	1	5		17	212 11	1	14		6 1	256 19 4
Scarlet fever Tuberculosis Typhoid and para-	1	23	6 10	73 95	110 44	8 4	5 2	10 4	6	220 163
typhoid fever Whooping cough		1	2 1	15 204	6 132	45	41	2 9	55	25 488

#### EGYPT

Infectious diseases—Fourth quarter 1939.—During the fourth quarter of 1939, the following numbers of cases of infectious diseases and deaths from the same causes were reported in Egypt:

Disease	Cases	Deaths	Disease	Cases	Deaths
Anthrax. Carebrospinal meningitis. Chickenpox. Diphtheria. Dysentery. Eryaipelas. Leprosy. Lethargic encephalitis. Malaria. Measles.	4 45 102 821 665 871 1, 849 115 6, 672 1, 471	2 25 2 377 108 96 37 18 27 31 275	Mumpe.         Poliomyelitis.         Puerperal septicemia.         Rabies.         Scarlet fever.         Tuberculosis (all forms).         Typhoid fever.         Typhois fever.         Undulant fever.         Whooping cough.	<b>333</b> 108 10 22 127 1,460 1,188 128 7 494	5 1 65 9 9 67 771 249 26 1 13

Vital statistics—Fourth quarter 1939.—Following are vital statistics for the fourth quarter of 1939 for all places in Egypt having a health bureau:

Live births per 1,000 population	64,887     Deaths per 1,000 population	9, 200
	(1966)	

#### (1366)

#### SWITZERLAND

Communicable diseases-April 1940.-During the month of April 1940, cases of certain communicable diseases were reported in Switzerland as follows:

Disease	Cases	Disease	Cases
Cerebrospinal meningitis Chickenpox Diphtheria and croup German measies Influen.za Measles Mumps Paratyphold fever	103 75 51 197 199 1, 316 93 7	Poliomyelitis Scarlet fever Trachoma Tuberculosis Typhoid fever Undulant fever Whooping cough	8 369 1 319 5 10 176

## WORLD DISTRIBUTION OF CHOLERA, PLAGUE, SMALLPOX, TYPHUS FEVER, AND YELLOW FEVER

From medical officers of the Public Health Service, American consuls, International Officer of Public Health, Pan American Sanitary Bureau, health section of the League of Nations and other sources. The reports contained in the following tables must not be considered as complete or final as regards either the list of countries included or the figures for the particular countries for which reports are given.

#### CHOLERA

[C indicates cases; D, deaths]

NOTE .- Since many of the figures in the following tables are from weekly reports, the accumulated totals are for approximate dates.

	January-	May	June 1940-week ended-					
Place	April, 1940	1940	1	8	15	22	29	
ASIA China: Shanghai C India C	16, 534		12			1		
Bassein 0 Calcutta 0 Cawnpore 0 Chittagong 0	12 808 10	130 307 1 4	5 67 1	65 1		95 		
MadrasC MoulmeinC Porto NovoC	1 1 31		10			1		
Rangoon	34 436 235							

#### 1 Imported.

#### PLAGUE

AFRICA Belgian Congo	3	9		 	 
Uganda	72 355 472	6 1 51	1	 	 
Rhodesia, Northern C Senegal: Dakar	1 \$1			 	 <b>-</b>
Thies O Union of South Africa O	12	1		 	 

<sup>1</sup> Includes 5 cases of pneumonic plague. <sup>9</sup> A report dated May 11, 1940, stated that there was an epidemic of bubonic plague in southern Morocco, where several hundred cases had been unofficially reported.

Imported.

#### PLAGUE-Continued

#### [C indicates cases; D, deaths]

	January-	May		June 19	ne 1940-week ended-			
Place	April, 1940	1940	1	8	15	22	29	
ASIA								
China.4							1	
Dutch East Indies: Java and Madura. C	187							
India C	12,099							
Bassein	16	1						
CochinC				·				
Plague-infected rats	8			.				
RangeonC Indochina (French)C	3							
Theiland:	•							
Bangkok	8			1				
Bisnulok Province								
Dhonpuri Province	3							
Javanad Province	3							
Kamphaeng Bajr Province C	29							
Kanchanapuri Province	12							
Koan Kaen Province	5							
Nagara Svarga Province	30			1				
Noangkhay Province	4							
Sukhodaya Province C	22							
EUROPE								
Portugal: Azores Islands C	2							
NORTH AMERICA								
United States. (See issue of July 19, p. 1321.)								
SOUTH AMERICA						ł		
Argentina:		÷	1 T					
Salta Province	26							
Santiago del Estero Province C Tucuman Province C	8							
Brazil:	•							
Alagoas State C	5			(				
Pernambuco State	ĭ							
Peru:	•							
Cajamarca Department	9							
Lambayeque Department	. Š							
Libertad Department	42							
Lima Department	24							
Piura Department Ö	6							
OCEANIA						ł		
Iawaii Territory: Plague-infected rats	12	1		1		2		
Tawan Territory. Lague-mecoco 1818	14					2		

<sup>4</sup> Information dated July 7 states that up to July 6, 17 cases of plague had been reported near Tungliao, Hsingan Province, China.

#### SMALLPOX

AFRICA					1
Algeria C Angola C	1 35			 	 
Belgian Congo C	1, 356	353		 	 
British East Africa C Dahomey O	12 17			 	 
French Guines C	13			 	 
GibraltarC Ivory CoastC	11	3		 	 
Nigeria	1, 481	187		 	 
Niger Territory O Nyasaland C	359 34	235 12		 	 
Portuguese East Africa	1			 	 
Senegal C	103	36 28	····	 	 
Sierra Leone	10 288	95		 	 
Budan (French)		95 1		 	 
Union of South Africa C	46			 	 

1 Imported.

#### SMALLPOX-Continued

### [C indicates cases; D, deaths]

Place	January-	May	June 1940-week ended-					
	April, 1940	May 1940	1	8	15	22	29	
AFIA China	255 512 533 4 90, 394 δ 24 805 148 113	111 	 	4		 		
Japan O Straits Settlements O Sumatra O Thailand C Great Britain C Greece	458 1 1 5 2 19	2 41  7						
Portugal C Spain C Turkey C NORTH AMERICA Guatemala	101 280 139	9 34	1 1	4	5	1 		
Mexico       C         SOUTH AMERICA       O         Bolivia       O         Brazil       C         Colombia       C         Ecuador       C         Venezuela (alastrim)       C	49 24 1 970 101	4  1 27	2					

<sup>2</sup> For the period May 3 to June 4.

#### **TYPHUS FEVER**

				-			· · · · · ·
Africa Algeria O	952	557		· .			
Belgian Congo C	1, 210						
British East Africa C Egypt C	2, 521	596	101				
Eritrea	40						
Morocco C Tunisia C	216 247	58 268					
Union of South Africa O	103	2	1				
ASIA							
ChinaC ChosenC	773	505					
India O	3						
Indochina (French) C Iran O	2 233						
IraqČ	72	14	2				
Japan O Palestine C	34	9	1	6			
Straits SettlementsC Trans-JordanC	13	1					
	10	-					
EUROPE BulgariaC	57	28	9				
Germany C	82 14	17					
Greece C Hungary C	52	10 17	3				
Irish Free State C Lithuania C	31		1	1		1	
Rumania C	977	115	33	12	27		
Spain C Turkey O	3 503	6	2				
YugoslaviaÖ	221	9					

# **TYPHUS FEVER**—Continued

#### [C indicates cases; D, deaths]

Place	January-	May	June 1940-week ended					
1,1804	April, 1940 May 1940		1	8	15	22	29	
NORTH AMBRICA Guatemala	179 159 3	82 2						
Bolivia	165 54 2 4	1						
OCEANIA C Hawaii Territory	9 12	1	1				2	

#### YELLOW FEVER

		1		,	
AFRICA					
Cameroon: NkongsambaC	11			 	 
French Equatorial Africa: Fort Ar- chambault	11			 	 
Gold CoastC Ivory CoastC Nimeria	1			 	 
Ibadan				 1	 
Oshogbe C		11		 	 
SOUTH AMERICA Brazil:					
Espirito Santo State D Rio de Janeiro State D	<sup>228</sup>			 	 
Colombia: Antioquia Department—San Luis D	2			 	 
Caldas Department— La Pradera	1				 
Samana D Victoria D	1			 	 
	-			 	 

<sup>1</sup> Suspected. <sup>9</sup> Jungle type.