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## Laboratory Studies On the 1950 Outbreak of Influenza

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During the first 3 months of 1950, a number of outbreaks of influenza were reported in military and civilian populations throughout the world. In the United States during this period, 164,352 patients were diagnosed on clinical grounds as having influenza. The results of serological and isolation procedures performed in different laboratories established that the infecting agent in a number of these outbreaks was influenza $A$ virus, while, in a few instances, influenza B virus appeared to be of etiological significance (1).

The present report is concerned primarily with observations on four strains of virus which were isolated from military patients with an influenza-like disease during January 1950 in Georgia and Wyoming. These agents are closely related antigenically to each other but differ somewhat from the so-called A prime strains of influenza A.

## Materials and Methods

Clinical Specimens. These were limited to nasal and pharyngeal washings collected shortly after the onset of clinical influenza and to acute phase and convalescent phase sera collected from persons during the first several days and several weeks, respectively, after initial symptoms.

Isolation of Virus. Nasal or pharyngeal washings from patients suffering from a disease clinically diagnosed as influenza were mixed with antibiotics and inoculated into the allantoic and amniotic sacs of embryonated eggs; subsequent passages were made by the allantoic route. After two or three transfers, each strain yielded allantoic fluids which had hemagglutinating titers of $1: 320$ to $1: 1280$. These fluids were infective for eggs in dilutions of $10^{-7}$ or $10^{-8}$.

[^0]The four 1950 strains of virus employed in the present study (Francis Warren-1-50, Francis Warren-2-50, Atlanta Headquarters-1-50 and Atlanta Depot-1-50) were used as allantoic fluid from the second to fourth passage in eggs.

Strains of Virus Isolated Prior to 1950. The following strains of influenza virus were used in the present studies. In each instance, infected allantoic fluids from the indicated passage were used throughout.

PR8. Isolated in 1934 from a patient in Puerto Rico (2). After one passage in ferrets, the virus was maintained through 691 passages in mice and 3 in embryonated eggs before it was procured from the Influenza Strain Study Center. The infected allantoic fluid used in this study was from the sixth and seventh egg passage.

Weiss. Isolated in 1943 from a patient at Fort Custer, Mich. (8). Received by this laboratory from the Biologics Control Division of the National Institutes of Health after 79 passages in mice. Transferred to eggs and used in this investigation as sixth and seventh egg-passage material.

FM1. Isolated in 1947 from a patient at Fort Monmouth, N. J. (4). Obtained from the Influenza Strain Study Center after 7 passages in mice and 12 in eggs. Fourteenth egg-passage material was used.

Wilfong. Isolated in 1948 from a patient in Los Angeles by Capt. C. H. Kempe of the Sixth Army Area Medical Laboratory. Originally isolated in embryonated eggs and used as allantoic fluid from the 21st and 22d passages in chick embryos.

SF-1-49. Isolated in 1949 from a patient in San Francisco by Capt. D. L. Weiss at the Sixth Army Area Medical Laboratory. Isolated and maintained exclusively in eggs. Sixth passage material was used in this study.
$4 M L-1-48 / 49$. Isolated in the winter of 1948-49 from a patient in Germany by Dr. E. Sheris at the 4th Medical Laboratory, Heidelberg. Allantoic fluids from an early but undetermined number of egg passages were used.

Lee. Isolated in 1940 (5) from a patient in New York State. After 8 passages in ferrets, the virus was maintained through 310 passages in mice and 3 in eggs before it was obtained from the Influenza Strain Study Center. The material used in this study was from the sixth egg passage.

All of these strains are of the A type except Lee which is type B.
Preparation of Antisera. Rooster, ferret, and mouse antisera were prepared for use in the present studies. ${ }^{1}$ Roosters weighing from 5 to 8 pounds were injected both intravenously and intraperitoneally with 5.0 ml . amounts of virus-infected allantoic fluids having hemagglutinating titers of 1:320 or greater. Young adult ferrets were inoculated intranasally with approximately $10^{4} \mathrm{egg}$ infectious doses of virus. There were no signs of illness during the first week, and they were reinoculated with about $10^{6}$ egg infectious doses of the same virus, again without obvious illness. The roosters were bled 10 days and the ferrets 2 weeks following the last injection of virus. The sera were stored at $4^{\circ} \mathrm{C}$. until used. Sera obtained from the roosters and

[^1]ferrets prior to immunization did not contain demonstrable antibodies against influenza virus.

Groups of mice weighing 14 to 16 grams were injected intraperitoneally with 0.5 ml . amounts of serial fivefold dilutions ( $1: 5-1: 3125$ ) of influenza vaccine lot 22A. This vaccine was a commercial product containing PR8 and FM1 strains of A virus and Lee strain of B virus. The mice were bled 2 weeks later, and the sera were stored in the frozen state until used.
Hemagglutination Techniques. The hemagglutination and hemag-glutination-inhibition titrations were performed by the standard diagnostic procedure recommended by the Committee on Serological Procedure of the Armed Forces Epidemiological Board (6). Type " $O$ " human erythrocytes were used in these tests.

Titration of Virus Infectivity. Titrations to determine the infectivity of allantoic fluids were performed by inoculating groups of six embryonated eggs via the allantoic cavity with 0.1 ml . amounts of serial tenfold dilutions of material to be tested. The presence or absence of hemagglutinin in fluid from each egg following 2 days' incubation at $35^{\circ}$ C., as determined by the spot plate method (4), indicated whether or not infection had occurred. The $\mathrm{ID}_{50}$ were calculated by the method of Reed and Muench (7).

Neutralization Tests. Neutralization tests with rooster and ferret antisera were performed in the following manner: Serial fourfold dilutions of serum, inactivated at $56^{\circ} \mathrm{C}$. for 30 minutes, were mixed with equal volumes of virus suspensions diluted to contain approximately $1000 \mathrm{ID}_{50}$ in 0.1 ml . The serum-virus mixtures were held at $37^{\circ} \mathrm{C}$. for 30 minutes and then were inoculated in 0.1 ml . amounts into the allantoic cavities of embryonated eggs. After incubating for 2 days, the presence or absence of hemagglutinin in the eggs was determined as already described. The neutralizing titer was taken as that dilution of serum in which the majority of eggs were protected from infection.

Neutralization tests with mouse antisera were performed in eggs by the method described for rooster and ferret sera except that constant amounts of serum diluted $1: 5$ were employed in place of the serial dilutions of serum.

## Experimental

Evidence for Influenza A Infection in 1950. Table 1 presents the results of agglutination-inhibition tests using acute and convalescent sera from patients in the Georgia and Wyoming outbreaks and antigens prepared from a number of strains of influenza $A$ virus and one strain of influenza $B$ virus. It is evident from the tabular data that influenza $A$ and not influenza $B$ was the virus involved in these cases.

The mean titers of inhibitory antibody for PR8 and FM1 viruses in the acute phase sera were appreciably higher than for the 1950 strain. The mean titers for the convalescent sera, on the other hand, were of approximately the same magnitude. Therefore, even though the convalescent serum titers were essentially the same, the number of cases showing fourfold or greater rise in serum titer was different in the tests with the several antigens. Nine of the 12 sera showed fourfold or greater rise in titer in tests with AD-1-50 and 8 of the 12 with FM1 virus, while only 5 of these sera showed such a rise with PR8 virus.

Table 1. Agglutination-inhibition tests with paired sera from patients with influenza

| Patients from the Georgia and $\mathbf{W}$ yoming outbreaks, January 1950 | Fold-increase in serum titer |  |  |  |
| :---: | :---: | :---: | :---: | :---: |
|  | Type A antigens |  |  | Type B antigen |
|  | PR8 (1934) | FM1 (1947) | $\underset{(1950)}{\text { AD-1-50 }}$ | Lee (1940) |
| 1 (AH-1-50) | 2 | 4 | 8 | 0 |
| 2 (FW-2-50) | 2 | 16 | 16 | 0 |
| 3 (AD-1-50) | 2 | 4 | 2 | 0 |
| 4 (FW-1-50) | 2 | 2 | 4 | 0 |
|  | 8 | 16 | 32 |  |
|  | 8 | 8 | 32 | 0 -2 |
| 7 | 8 | 8 | 32 64 | -2 |
| 8 | 16 | 32 | 64 32 | -2 |
| 10 | 0 | 2 | 8 | 0 |
| 11. | 0 | 0 | -2 | -2 |
| 12. | 2 | 0 | 0 | 2 |
| Geometric mean titer: |  |  |  |  |
| Acute phase serum... | 1:80 | 1:80 | 1:20 | 1:160 |
| Convalescent serum | 1:320 | 1:640 | $1: 320$ $16 \times$ | 1:160 |
| Number of Positive Cases | 5/12 | 8/12 | 9/12 |  |
| Total Cases |  |  |  |  |

Comparison of Influenza Virus Strains by the Agglutination-Inhibition Technique. The titration results of hemagglutination-inhibition tests using sera of roosters immunized against strains of influenza A virus isolated since 1934 and homologous and heterologous antigens are presented in table 2. These same data are summarized in interpretive form in table 3. It is evident from the tabular data that the strains of influenza A employed in these tests, and considered to be representative of the virus during the period from 1934 to the present, show progressive change in antigenic structure. It is of interest that the strains from 1947 and 1948 react in a manner which suggests that they are essentially identical. Of the two, however, the 1948 strain is perhaps more closely related to the viruses recovered in 1949 and 1950. Furthermore, the 1949 and 1950 strains appear to be identical to each other and to have practically all of the antigenic components of the strains from the 2 preceding years. However, they seem to possess
additional factors since antisera against the 1947 and 1948 strains give higher titers with their homologous antigens than with the 1950 antigens.

While the influence of numerous passages $(8,9)$ of the older strains in animals and eggs cannot be assessed, this factor would appear to be of minor importance in strains isolated since January 1947. Whatever the relationship of the current PR8 and Weiss viruses to the strains isolated from patients in 1934 and 1943, it is clear that these viruses have little relationship to the more recently isolated strains.

Table 2. Agglutination-inhibition tests using influenza antigens and rooster antisera

| Rooster antiserum against: | Serum titers obtained using antigens: |  |  |  |  |  |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | PR8 | Weiss | FM1 | Wilfong | $\underset{49}{\mathrm{SF}-1-}$ | $\begin{aligned} & \text { 4ML- } \\ & \hline 48 / 49 \end{aligned}$ | $\underset{50}{\mathrm{AD}-1-}$ | $\begin{gathered} \text { FW-1- } \\ 50 \end{gathered}$ | $\underset{50}{\mathrm{AH}-1-}$ | $\underset{50}{\mathrm{PW}-2-}$ |
| PR8 (1934).-.------ | * 800 | 50 | 50 | **0 | 0 | 0 | 0 | 0 | 0 | 0 |
| PR8 (1934)-------- | *800 | 100 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Weiss (1943) .------- | 200 | 800 | 100 | 50 | 0 | 0 | 0 | 0 | 0 | 0 |
| FM1 (1947) ---.---- | 50 | 100 | *800 | 400 | 100 | 100 | 100 | 100 | 100 | 100 |
| Wilfong (1948)...-.- | 50 | 100 | 800 | 400 | 200 | 400 | 200 | 200 | 200 | 200 |
| SF-1-49 | 0 | 0 | 400 | 400 | 400 | 400 | 400 | 400 | 200 | 200 |
| AD-1-50...---...-- | 0 | 0 | 800 | 400 | 400 | 400 | 400 | 400 | 200 | 200 |
| FW-1-50. | 0 | 0 | 800 | 400 | 800 | 800 | 800 | 800 | 400 | 400 |
| A $\mathrm{H}-1-50$. | 0 | 100 | 800 | 800 | 800 | 800 | 800 | 800 | 800 | 800 |

*These sera, having titers of $1: 1600$ or 1:3200, were initially diluted $1: 2$ or $1: 4$ in the tests to bring their titers to a level comparable with that of other sera used.
${ }^{* *}(0)=$ Titer less than 1:50, the lowest dilution tested.
Table 3. Interpretive summary of agglutination-inhibition tests showing relationships of influenza type $\boldsymbol{A}$ strains according to year of isolation
(Based on results of table 2)

| Rooster antisera against strains isolated in: | Antigens from strains isolated in: |  |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | 1934 | 1943 | 1947 | 1948 | 1949 | 1950 |
| 1934 | $\underline{+++}$ | + | $\pm$ | 0 | 0 | 0 |
| 1943 | ++ | + + + + | + | $\pm$ | 0 | 0 |
| 1947 | $\pm$ | + | $\underline{++++}$ | +++ | + | $+$ |
| 1948 | $\pm$ | + | ++++ | $+++$ | +++ | ++ |
| 1949 | 0 | 0 | ++++ | ++++ | $\underline{+++}$ | $++++$ |
| 1950 | 0 | 0 | ++++ | + + + + | $\underline{+++}$ | $\underline{+++}$ |

The results of tests with ferret antisera and various viral antigens (table 4) are essentially intermediate between those obtained with rooster and with human antisera. However, they tend to support the differences noted when chicken sera were employed. The somewhat broader range of serological activity shown by ferret sera was perhaps to be expected since the susceptible ferrets received infectious material on two occasions, whereas, the resistant chickens received only a single inoculation.

Protective Effect of Sera from Immune and Vaccinated Animals. The results of neutralization tests with rooster and ferret sera, sum-
marized in table 5, showed an appreciable difference between FM1 virus and the current strains of influenza. The degree of difference exhibited in neutralization tests with rooster antiserum was slightly greater than that demonstrated by the hemagglutination-inhibition test. The two types of tests brought out differences of a similar degree when ferret antisera were employed.

Since such a marked antigenic difference existed between the FM1 and 1950 viruses, it was of interest to determine whether animals injected with standard polyvalent influenza vaccine (which contains PR8, FM1, and Lee antigens) would develop protective antibodies against the 1950 strains. The results presented in table 6 indicate that moderate protection was afforded against the homologous strains of virus but there was no protection against the current strains.

Table 4. Agglutination-inhibition tests using influenza antigens and ferret antisera

| Ferret antiserum against: | Serum titers obtained using antigens: |  |  |  |  |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | PR8 | Weiss | FM1 | Wilfong | $\underset{49}{\mathrm{SF}-1-}$ | $\begin{gathered} \text { 48L/49 } \end{gathered}$ | $\underset{50}{\mathrm{AD}_{2}-1-}$ | $\underset{50}{\text { FW-1- }}$ | $\underset{50}{\mathrm{AH}-1-}$ |
| PR8 (1934) ----------------- | 640 | 640 | 80 | * 0 | 0 | 0 | 0 | 0 | 0 |
| Weiss (1943) ---.-.-.-.-.-.-.-.--- | 640 | 640 | 160 | 40 | 10 | 10 | 10 | 10 | 10 |
| FM1 (1947) | 80 | 20 | 640 | 160 | 160 | 160 | 160 | 160 | 160 |
| AD-1-50 | 10 | 10 | 640 | 320 | 320 | 640 | 640 | 640 | 640 |
| FW-1-50 | 20 | 0 | 640 | 640 | 640 | 640 | 640 | 640 | 640 |
| AH-1-50.. | 20 | 0 | 640 | 640 | 640 | 640 | 640 | 640 | 640 |

$*(0)=$ Titer less than 1:10, the lowest dilution tested.
Table 5. Egg neutralization tests with FMI antisera and 1950 strains of virus

| Virus strain | Dilution of serum neutralizing 500 ID $_{50}$ of virus |  |
| :---: | :---: | :---: |
|  | Rooster serum | Ferret serum |
| FM1 | 1:6400 | 1:1600 |
| AD-1-50 | 1:100 | 1:400 |
| FW-1-50 AH- | 1:100 | 1:400 |

Table 6. Assay of standard (NIH) influenza vaccine using homologous and heterologous strains of challenge virus

| Dilution of vaccine injected into donor mice | Results of neutralization tests in eggs with challenge virus |  |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | PR8 | FM1 | AD-1-50 | FW-1-50 | AH-1-50 | Lee |
| 1:5. | *0/7 | 0/7 | 5/6 | 6/6 | 7/7 | 0/7 |
| 1:25 | $2 / 7$ | 1/7 | 7/7 | $7 / 7$ | 7/7 | 3/7 |
| 1:125 | 4/6 | $7 / 7$ | 7/7 | $7 / 7$ | 7/7 | 5/5 |
| 1:625 | 7/7 | 7/7 | 7/7 | 7/7 | 7/7 | 7/7 |
| Dilution of virus used | 10-4.0 | 10-5.0 | 10-5.0 | 10-6.0 | 10-4.0 | $10^{-1.0}$ |
| Control virus titration ID ${ }_{\text {s0 }}$ | $10^{-7.8}$ | $10^{-9.0}$ | 10-\%. | 10-\%.8 | $10^{-8.0}$ | $10^{-7.7}$ |

*Numerator $=$ Number of eggs infected. Denominator $=$ Total number of eggs.

## Discussion

The data clearly indicate that considerable antigenic difference exists between the current strains of influenza virus and the PR8 and FM1 strains recovered during epidemics in previous years.

It would appear that progressive and continuous antigenic change has been occurring under natural conditions in some strains of influenza A virus which have infected man during recent years. Even though the rate of progress of this change during the last 4 years would appear to have been relatively slow, it is nevertheless appreciable. Indeed, these laboratory studies have shown that the differences are sufficiently great to warrant serious consideration of the elimination of older strains of virus from the vaccines and diagnostic antigens and the substitution of more recently isolated agents in their place. In any case, a periodic revision of the components in diagnostic and immunizing influenza materials seems indicated in order that the strains used may more closely resemble the current strains infecting man.

## Summary

The results of hemagglutination-inhibition and protective tests indicated that a number of strains of influenza A virus recovered in 1950 differed antigenically from those isolated in previous years.

The changing antigenic structure of strains of influenza virus affecting man appears to progress slowly but steadily. Over a number of years, the divergence is sufficient so that the earlier isolated strains elicit few protective antibodies against the newly isolated ones.

## REFERENCES

(1) National Office of Vital Statistics, Public Health Service: Communicable disease summaries and laboratory supplements for the weeks ending February 4 and March 25, 1950. Release of February 9 and March 30 (1950).
(2) Francis, T., Jr.: Transmission of influenza by a filterable virus. Science 80: 457-459 (1934).
(3) Salk, J. E., Menke, W. J., and Francis, T., Jr.: Identification of influenza virus type A in current outbreak of respiratory disease. J.A.M.A. 124: 93 (1944).
(4) Rasmussen, A. F., Jr., Stokes, J. C., and Smadel, J. E.: The army experience with influenza, 1946-1947. II. Laboratory aspects. Am. J. Hyg. 47: 142-149 (1948).
(5) Francis, T., Jr.: A new type of virus from epidemic influenza. Science 92: 405-408 (1940).
(6) Committee on Serological Procedure, Armed Forces Epidemiological Board: An agglutination-inhibition test proposed as a standard of reference in influenza diagnostic studies. In press.
(7) Reed, L. J. and Muench, H.: A simple method of estimating 50 percent endpoints. Am. J. Hyg. 27: 493-497 (1938).
(8) Hirst, G. K.: Studies on the mechanism of adaptation of influenza virus to mice. J. Exper. Med. 86: 357-366 (1947).
(9) Hirst, $\mathcal{G}$. $\dot{K}$.: Comparisons of influenza virus strains from three epidemics. J. Exper. Med. 86: 367-381 (1947).

# Salmonella in Hen Eggs 

By Mary June Carter, M. S., Marcus P. Powell, M. S., C. E., and Irving H. Borts, M. D.

The purpose of this study was to determine the presence of Salmonella in fresh hen eggs by periodical sampling of the raw eggs being prepared for consumption in a local institution. No attempt was made to distinguish between shell contamination and internal contamination of the egg. The technique of breaking the eggs to obtain cultures was the same as that used by the cook in the ordinary course of kitchen duties.

## Review of Literature

The presence of Salmonella organisms has been reported in hen, turkey, duck, goose, and pigeon eggs. However, less information is available in the literature concerning the occurrence of Salmonella in hen eggs or their products.

Edwards and Bruner (1), Chase and Wright (2), Buxton and Gordon (3), Scott (4), and Savage (5) have reported on the incidence of Salmonella in chickens.

Tanner (6), Rettger et al. (7), Haines and Moran (8), Solowey et al. (9), and Schneider and his co-workers (10) have demonstrated different modes of infection, including an infected ovary and penetration of the shell by the organism.

Crowe (11), Mitchell and his co-workers (12), Brown, Combs, and Wright (18), Greenblatt et al. (14), Stewart and Slack (15), Verder and Sutton (16) have described numerous vehicles of transmission of the organism. Watt (17) has described an outbreak of 28 cases of food poisoning due to Salmonella montevideo on board a merchant vessel. The organism was probably in the salad mayonnaise made from raw hen eggs. This species was isolated from the shells of 2 cases of unused eggs obtained from the ship. These eggs, which were taken on board ship 2 months previously, were traced to a carload originating in southern Iowa. Three egg-grading plants had contributed to this shipment. At ihe time of Watt's report, more than 5,000 eggs from 850 farms had been cultured in an attempt to determine the extent and location of infection among poultry flocks.

[^2]
## Experimental Procedure

This study was made on fresh eggs which were purchased in 30dozen lots for use in a local institution. The routine procedure of the institution in using eggs was to immerse 1 dozen eggs at a time in hot water ( $120^{\circ}$ to $122^{\circ} \mathrm{F}$.) for 2 minutes. The water was then drained, and the eggs were broken into a mixing bowl and beaten with a dover-type egg beater. This procedure was followed in the experiment by the senior author. ${ }^{1}$ A $30-\mathrm{ml}$. sample of the egg melange was pipetted into a covered beaker. Then, 0.5 ml . of blended egg mixture was pipetted into 10 ml . of tetrathionate broth (Difco) and incubated at $37^{\circ} \mathrm{C}$. for 7 days. In preliminary studies, a 2- to 4-day incubation period was found inadequate; hence, a 7 -day incubation period was adopted. At the end of the incubation period, a loopful of the inoculated tetrathionate broth was plated to eosin methylene blue agar and to bismuth sulfite agar and incubated at $37^{\circ} \mathrm{C}$. for 48 hours.

Suspicious colonies were picked and stained by Gram's method.
Typical colonies from eosin methylene blue agar or bismuth sulfite agar were transferred to Kligler's iron agar and incubated 48 hours. A loopful of growth from Kligler's was transferred to the following: 2 meat infusion agar slants for stock cultures; dextrose, mannite, maltose, lactose, xylose, sucrose, dulcite, and salicin for fermentation tests; tryptose broth with lead acetate paper for $\mathrm{H}_{2} \mathrm{~S}$ production; tryptose broth for motility; tryptone broth for Kovac's indole test; gelatin plate plus ammonium sulfate solution for gelatin liquefaction; litmus milk; and buffered-peptone solution for acetyl methyl carbinol test.

## Results

In this study all cultures diagnosed as positive were forwarded for confirmation and typing to the Salmonella Center, Agricultural Experiment Station of Kentucky, Lexington.

Of the 186 samples representing 247 dozen, or 2,964 eggs, numbers $6,25,26,27,153$, and 157 were identified as Salmonella (see table). Culture number 6 was found to be Salmonella paratyphi B, a gramnegative, hydrogen sulfide-producing organism. It produced acid and gas in dextrose, mannite, maltose, xylose, and dulcite but did not ferment lactose or sucrose, nor produce indole. The culture S. paratyphi $B$ recovered from the eggs belonged to the tartrate negative or human type. In his letter confirming the identification of this organism, Dr. P. R. Edwards commented as follows: "Relatively few human-type cultures of $S$. paratyphi $B$ are found in animals. We have only two or three from chickens. I believe the isolation of this organism from eggs is a most significant thing."

Culture numbers 25, 26, 27, 153, and 157 were Salmonella pullorum.

[^3]|  | $\begin{gathered} \text { ® } \\ \text { ⿷匚⿳ } \end{gathered}$ |  |  | $\begin{aligned} & \infty \\ & \text { 品 } \\ & \hline \end{aligned}$ |  |  |  |  | $\begin{aligned} & \mathscr{0} \\ & \text { OU } \\ & \text { O} \\ & \text { H } \end{aligned}$ | $\begin{aligned} & \mathscr{8} \\ & \frac{0}{\lambda} \\ & \text { A } \end{aligned}$ |  | S ¢ ¢ | 莵 | 怘 |  | Litmus milk |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| 25 | A | 二 | $\stackrel{\mathbf{A G}}{\mathbf{A G}}$ | ＋＋ | $+$ | $+$ | $\pm$ | $\pm$ | 二 | $\pm$ | － |  |  | － |  | （＊）${ }^{\text {Slightly acid without coag－}}$ ulation． |
| 26 | B | － | AG | $+$ | ＋ | ＋ | ＋ | － | － | $+$ | － | － | － | － | － | Slightly acid． |
| 27 | B | － | AG | $+$ | ＋ | $+$ | $+$ | － | － | $+$ | － | － | － | － | － | Do． |
| 153 157 | A | － | AG | $+$ | $+$ | $+$ | $+$ | － | － | ＋ | － | － | － | － | － | Do． |
| 157 | A | － | 1 G | ＋ | ＋ | ＋ | ＋ | － | － | ＋ | － | － |  |  |  | Do． |

＊Stock culture died before these tests were made．
They produced acid and gas in dextrose，mannite，and xylose but did not ferment maltose，lactose，sucrose，dulcite，or salicin．They did not produce indole or acetyl methyl carbinol，liquefy gelatin，or coagu－ late milk．

The eggs used in this series of experiments were handled exactly as the cook would have handled them up to the taking of the samples． The senior author was examined and proved not to be a Salmonella carrier．Hence positive cultures could have originated only within the eggs or on the shells．The recovery of a few positive cultures opens epidemiologic possibilities，whether the organisms were within the eggs or were on the shells．If the latter were true，the routine pro－ cedure followed by the cook failed in these instances to damage the contaminating organisms．

In any event，the use of uncooked hen eggs，e．g．，in eggnogs and mayonnaise，is not without a certain amount of hazard from patho－ genic Salmonella．

## Conclusions

1．One hundred eighty－six samples representing 247 dozen，or 2,964 eggs，taken from 56 cases（ 30 dozen per case）were examined． Positive cultures were obtained from 6 samples（or 3.2 percent of the samples）．Each positive sample came from a mixture of 1 dozen eggs．

2．Salmonella paratyphi $B$ was recovered from 1 sample and Sal－ monella pullorum from the remaining 5.

3．Under the procedure，the infection could come only from the contents or from the shells of the eggs．

4．Although hen eggs，as a source of Salmonella infection，have been incriminated in only a few instances，the possibility of the transmission of this organism by serving uncooked eggs should be recognized． The finding in this study that 3.2 percent of the lots tested contained Salmonella emphasizes that the potential hazard of infection from uncooked eggs is greater than heretofore has been recognized．

## REFERENCES

(1) Edwards, P. R. and Bruner, D. W.: Incidence of Salmonella types in fowls. In the U. S. Proc. 7th World Poultry Congress, 1939, p. 271.
(2) Chase, F. E. and Wright, M. L.: The occurrence and distribution of Salmonella types in fowls. Canad. J. Research F 24 : 77 (1946).
(s) Buxton, A. and Gordon, R. F.: Epidemiology and control of Salmonella thompson infection of fowls. J. Hyg. 45: 265-281 (1947).
(4) Scott, W. M.: Food poisoning due to eggs. Brit. Med. J. 2: 56-58 (1930).
(5) Savage, W. G.: Some problems of Salmonella food poisoning. J. Prev. Med. 6: 425-451 (1932).
(6) Tanner, F. W.: Microbiology of Foods. Ed. 2, 1944, pp. 943-945.
(7) Rettger, L. F., Hull, T. G., and Sturges, W. S.: Feeding experiments with Bacterium pullorum. The toxicity of infected eggs. J. Exper. Med. 23: 475-489 (1916).
(8) Haines, R. B. and Moran, T.: Porosity of and bacterial invasion through the shell of the hen's egg. J. Hyg. 40: 453-461 (1940).
(9) Solowey, M., Spaulding, E. H., and Goresline, H.: An investigation of the source and mode of entry of Salmonella organisms in spray-dried whole egg powder. Food Research 11: 380-390 (1946).
(10) Schneider, M. D.: Isolation of Salmonella tennessee from frozen whole and powdered egg. Bull. U. S. Army Med. Dept. 4: 477 (1945).
(11) Crowe, M.: Localized outbreak of Salmonella poisoning apparently transmitted by hen's egg. J. Hyg. 44: 342-345 (1946).
(12) Mitchell, R. B., Garlock, F. C., and Broh-Kahn, R. H.: An outbreak of gastroenteritis presumably caused by Salmonella pullorum. J. Infect. Dis. 78: 57 (1946).
(18) Brown, E. G., Combs, G. R., and Wright, E.: Food-borne infection with Salmonella aertrycke. J. A. M. A. 114: 642-644 (1940).
(14) Greenblatt, A. R., Delay, P. D., Breslow, L., and Greenblatt, I. J.: Salmonella epidemic from commercially prepared sandwiches. Bull. U. S. Army Med. Dept. 5: 345-348 (1946).
(15) Stewart, J. K. and Slack, J. M.: Salmonella food poisoning. Bull. U. S. Army Med. Dept. No. 88, 3: 120-122 (1945).
(16) Verder, E. and Sutton, G.: Salmonella food poisoning. J. Infect. Dis. 53: 262 (1933).
(17) Watt, J.: Outbreak of Salmonella infection in man from infected chicken eggs. Pub. Health Rep. 60: 835-839 (1945).
(18) Felsenfeld, O.: Salmonella problem: practical laboratory application of recent advances. Am. J. Clin. Path. 15: 584-608 (1945).

## Prevalence of Poliomyelitis in 1949

By C. C. Dauer, M. D.*

Prevalence in the United States. In 1949 the incidence of poliomyelitis cases was higher than in any previous year if only the number of reported cases is considered. However, the case rate per 100,000 population was lower than in 1916 when 44 States reported 29,061 cases as shown in table 1. In spite of the descriptions that 1949 was the "worst polio year" in the history of the country, there are certain data which suggest a less gloomy picture.

Table 1. Number of poliomyelitis cases and deaths reported, and morbidity and mortality rates per 100,000 population for certain years in the United States

| Year | Total cases reported | Number States reporting | Case rates per 100,000 population | $\begin{aligned} & \text { Number } \\ & \text { deaths } \\ & \text { registered } \end{aligned}$ | Number States reporting | Death rates per 100,000 population |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| 1916 | 29, 061 | 44 | 30. 2 | 7, 130 | 26 | 10. 6 |
| 1925 | 5, 926 | 44 | 5. 6 | 1,492 | 40 | 1. 5 |
| 1927 | 10, 533 | 48 | 8. 9 | 2, 013 | 41 | 1. 9 |
| 1931 | 15, 790 | 43 | 14. 6 | 2, 096 | 47 | 1.8 |
| 1945 | 13, 619 | 48 | 10. 3 | 1,186 | 48 | . 9 |
| 1946 | 25, 191 | 48 | 18.0 | 1,845 | 48 | 1. 3 |
| 1947 | 10, 734 | 48 | 7. 4 | 580 | 48 | 4 |
| 1948 | 27, 680 | 48 | 18. 9 | 1,895 | 48 | 1. 3 |
| 1949 | ${ }^{1} 42,174$ | 48 | 28. 3 |  |  | 21.6 |

${ }^{1}$ Provisional.
2 Rate based on a 10-percent sample of deaths by National Office of Vital Statistics.
The death rate from poliomyelitis in 1949 has been estimated by the National Office of Vital Statistics to have been about 1.6 per 100,000 population. As shown in table 1, the death rate was higher in 1916, 1927 and 1931, and almost as high in 1925 as it was in 1949; however, the numbers of cases reported were much lower than the number reported in 1949. This would appear to indicate that either the disease was less severe in 1949, or that many more nonparalytic cases were being included in the total number of cases reported, or that some other infectious diseases possibly were being called poliomyelitis.

Prevalence in States. The incidence in the various States in 1949 is shown in table 2, and for comparison the morbidity rates for the previous 5 years are included. This tabulation shows that only the six States, Maine, Massachusetts, Connecticut, Michigan, Arkansas and Oklahoma, had significantly higher morbidity rates in 1949 than

[^4]Table 2. Number of poliomyelitis cases reported in 1949; and morbidity rates per 100,000 population by States, 1944-49

| States by division | $\begin{gathered} \text { Cases re- } \\ \text { ported } \\ 1949 \end{gathered}$ | Morbidity rates |  |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  |  | 1949 | 1948 | 1947 | 1946 | 1945 | 1944 |
| New England: |  |  |  |  |  |  |  |
| Maine- | 447 | 49.2 | 4.4 | 4.8 | 5.1 | 11.3 | 2.7 |
| New Hampshire. | 225 | 41.4 | 4.4 | 6.5 | 41.5 | 7.5 | 15.0 |
| Vermont.---.-- | 153 | 41.5 | 6.9 | 10.9 | 23.2 | 19.3 | 13.3 |
| Massachusetts | 1,805 | 38.3 | 3.7 | 7.3 | 9.2 | 12.6 | 10.6 |
| Rhode Island. | 193 | 26.0 | 1.1 | 18.9 | 11.9 | 1.9 | 1.8 |
|  |  |  |  |  |  |  |  |
|  |  |  |  |  |  |  |  |  |
| New York.-... | 5, 241 | 36.4 | 9.7 | 8.4 | 10.8 | 14.4 | 48.9 |
| New Jersey-- | 1,518 | 31.2 | 16.9 | 6.6 | 6.1 | 22.6 | 13.5 |
|  |  |  |  |  |  |  |  |
| Ohio-...-.-.-.-..- | 1,803 | 22.6 | 15.0 | 18.8 | 10.4 | 6.7 | 17.1 |
| Indiana. | 1,150 | 28.8 | 9.8 | 6.7 | 13.2 | 5.9 | 9.9 |
| Illinois | 2,842 | 33.6 | 12.7 | 10.3 | 33.1 | 14.3 | 7.4 |
| Michigan | 2, 914 | 45.9 | 12.4 | 10.4 | 19.9 | 3.9 | 16.4 |
| West North Central: |  |  |  |  |  |  |  |
|  |  |  |  |  |  |  |  |  |
| Minnesota | 1,889 | 63.5 | 47.0 | 8.7 | 127.4 | 11.5 | 22.1 |
| Iowa--- | 1,221 | 46.2 | 48.0 | 6.7 | 28.0 | 19.1 | 9.0 |
| Missouri. | 1,326 | 33.7 | 8.1 | 3.4 | 35.6 | 8.4 | 5.3 |
| North Dakota | 451 | 74.5 | 13.2 | 13.4 | 88.6 | 3.2 | 9.9 |
| South Dakota. | 408 | 62.9 | 1428 | 4.6 | 68.4 | 3.8 | 1.5 |
| Nebraska. | 681 | 53.0 | 55.1 | 15.4 | 53.9 | 10.0 | 5.5 |
| Kansas. | 741 | 38.1 | 16.1 | 4.7 | 61.4 | 7.4 | 6.9 |
| South Atlantic: |  |  |  |  |  |  |  |
| Delaware- | 46 | 14.8 | 43.1 | 39.2 | 11.1 | 10.1 | 33.9 |
| Maryland | 265 | 12.2 | 8.0 | 4.9 | 6.1 | 6.0 | 25.6 |
| District of Columbia | ${ }^{1} 106$ | ${ }^{1} 12.3$ | 15. 1 | 2.7 | 4.0 | 14.8 | 21.5 |
| Virginia-- | 336 | 10.8 | 18.8 | 5.8 | 4.6 | 10.9 | 21.3 |
| West Virginia | 348 | 17.9 | 9.3 | 7.8 | 3.3 | 3.8 | 128 |
| North Carolina | 247 | 6.4 | 67.4 | 8.1 | 4.4 | 4.5 | 26.7 |
| South Carolins | 110 | 5.5 | 19.0 | 3.4 | 1.1 | 9.9 | 3.1 |
| Georgia | 229 | 7.2 | 7.2 | 2.5 | 5.1 | 4.0 | 3.5 |
| Florida- | 281 | 11.3 | 16.3 | 4.7 | 23.9 | 6.0 | 5.0 |
| East South Central: |  |  |  |  |  |  |  |
| Tennessee. | 541 | 16.7 | 11.9 | 5.5 | 6.3 | 15.2 | 4.7 |
| Alabama | 253 | 8.7 | 7.2 | 1.7 | 13.4 | 5.4 | 3.8 |
| Mississippi | 358 | 16.8 | 7.7 | 2.9 | 16.4 | 3.8 | 6.4 |
| West South Central: |  |  |  |  |  |  |  |
| Arkansas.- | 994 | 50.6 | 7.6 | 4.3 | 22.9 | 3.9 | 2.5 |
| Louisiana. | 228 | 8.7 | 6. 5 | 2.1 | 15.5 | 5.5 | 6.8 |
| Oklahoma | 1, 313 | 57.0 | 15.3 | 24 | 21.3 | 9.8 | 2.7 |
|  |  |  |  |  |  |  |  |
|  |  |  |  |  |  |  |  |  |
| Montana. | 98 | 18.8 | 12.9 | 5.1 | 28.1 | 17.9 | 8.3 |
| Idaho - | 510 | 86.1 | 23.0 | 72.1 | 9.6 | 4.8 | 3.2 |
| W yoming | 120 | 42.3 | 29.8 | 6.6 | 49.4 | 9.7 | 4.2 |
| Colorado. | 669 | 55.1 | 10.7 | 5.8 | 80.3 | 13.0 | 6.0 |
| New Mexico | 196 | 33.3 | 13.8 | 7.6 | 31.0 | 4.7 | 4.7 |
| Arizona | 180 | 24.2 | 26.0 | 5.5 | 23.3 | 4.0 | 6. 0 |
| Utah | 299 | 43.8 | 32.3 | 4.4 | 24.0 | 41.3 | 4.2 |
|  |  |  |  |  |  |  |  |
|  |  |  |  |  |  |  |  |  |
| Oregon.-- | 327 | 18.8 | 13.4 | 7.2 | 12.8 | 5.7 | 20.4 |
| California | 2, 779 | 28.1 | 57.7 | 8.9 | 24.8 | 10.3 | 6.2 |

${ }^{1}$ Final figure.
had been reported during the previous 5 years, i. e., a rate which was more than 20 points higher. In the New England area, there had been no widespread outbreak of the disease for a number of years, but the morbidity rates were considerably lower than those of certain States in the Midwest and Rocky Mountain regions where outbreaks had occurred as recently as 1946 or 1948.

Prevalence in Counties. The distribution of poliomyelitis by coun-

ties in 1949 is shown on the map. While the disease was epidemic over a large area in the central part of the country, there was a distinct tendency toward grouping of counties with relatively high morbidity rates and some intervening groups with few or no cases. Two smaller areas, one in the extreme northeast section and the other in the northern Rocky Mountain region also had relatively high rates, especially in the latter group.

There were 24 counties with populations of 10,000 or more (1943 estimate) which had rates in excess of 200 per 100,000 population, most of them located in the North Central States. In addition to these, 36 counties with populations under 10,000 had rates in the same range. In the latter group, several had populations so small that excessively high rates could have been a matter of chance occurrence in one or two places. The incidence in one of the 24 counties is worth reviewing. Tom Green County in the west central part of Texas began to report cases in July 1948 and continued to do so throughout each of the subsequent fall and winter months; the peak in incidence was reached in June 1949 when 103 cases were reported. A steady although decreasing number of cases was reported throughout the remainder of 1949 , and reports continued into the first 3 months of 1950. Thus in this population of nearly 40,000 persons, two-thirds of whom live in San Angelo, a total of 430 cases of poliomyelitis were reported from June 1948 through March 1950. The rate in Tom Green County in 1949 was slightly more than 800 per 100,000 population, but over a period of 22 months 1 percent of the population was reported to have had the disease. Korns (1) reported an incidence rate of over 800 for the first 9 months in a village in upper New York State, but in this instance the cases were reported principally in August and September 1949.

Of the 24 counties with relatively high rates, none had been entirely free of the disease for 5 consecutive years just prior to 1949. Eight had reported cases during each of the preceding 5 years, and an equal number had a morbidity rate of 100 or more during 1 of these 5 years. None of the 24 counties with rates of 200 or more had a population in excess of 100,000 which is consistent with the view that severe epidemics tend to occur more frequently in communities which are predominantly rural. However, Gilliam (2) reported only a slightly greater probability of epidemics in counties with the smaller populations located in northern States.

Prevalence of Other Central Nervous System Virus Infections. There are definite indications that infections due to viral agents other than the poliomyelitis virus are being erroneously diagnosed and reported as nonparalytic poliomyelitis. Kilham (3) reported on a series of 17 cases occurring in Massachusetts in 1948, in which there was a diagnosis of nonparalytic poliomyelitis. Six of the 17 appeared to have
mumps meningoencephalitis as shown by serological tests and recovery of mumps virus from the spinal fluid in 3 of the 6 . He also commented upon the greater frequency of mumps meningoencephalitis without parotitis during the summer months. Without appropriate serological tests or recovery of mumps virus, a distinction cannot be made between this infection and nonparalytic poliomyelitis because clinical symptoms are quite similar.

Another virus infection resembling nonparalytic poliomyelitis in man was first reported in 1948 by Dalldorf and Sickles (4) from upper New York State. Subsequent reports by other investigators have appeared which suggest a fairly wide distribution of the virus, now commonly called the Coxsackie virus, or rather a group of viruses since there are at least two distinct strains. The infection appears to be present mainly in the summer time and like poliomyelitis, the agent can be recovered from the throat and feces of infected persons. Persons from whom the virus has been recovered have had clinical symptoms which are similar to those seen in nonparalytic poliomyelitis, and these viruses have been reported from three persons in Nassau County, New York, who had extensive paralytic poliomyelitis. Most essential features of the epidemiology of Coxsackie virus infections are still to be worked out. However, presence of the virus can be determined more easily than in poliomyelitis because suckling mice injected with infectious material show a typical pathological picture, namely, a degeneration of muscle fibers.

The extent to which these virus infections, erroneously diagnosed as nonparalytic poliomyelitis contribute to the total number of poliomyelitis cases reported is a matter of conjecture. Since mumps is endemic in many communities and epidemic only at intervals of 7 or 8 years, it is probable that this infection contributes only a limited number of erroneously diagnosed cases, depending on time and place. Not enough is known of the epidemiology of the Coxsackie group of virus infections to warrant an estimate of the number it contributes. The fact that the virus has been isolated from persons in the New England States, New York State, Ohio, North Carolina, Texas (5), and the metropolitan area of Washington, D. C., including Montgomery County in Maryland and nearby Virginia (6), and Alabama, Colorado, Delaware, Florida, Georgia, Louisiana, Oklahoma, South Dakota and Tennessee (7) suggests that it is widespread and accounts for a significant number of erroneously diagnosed cases of nonparalytic poliomyelitis.

The presence of these virus infections clinically indistinguishable from nonparalytic poliomyelitis makes it apparent that as the proportion of nonparalytic cases increases, the need for caution in interpreting morbidity data becomes more imperative. The contention of Leake and others is more pertinent than ever, namely, that com-
parisons of poliomyelitis morbidity in different places and at different times be made by using only paralytic cases.

## Summary

The incidence of poliomyelitis in 1949 was high in the United States as indicated by the number of reported cases, but the provisional death rate was relatively low. There is reason to believe that an unknown number of cases reported as nonparalytic poliomyelitis may have been instances of other viral infections.

## REFERENCES

(1) Korns, R. F.: Health in New York State. Health News 26: 6 (1949).
(2) Gilliam, A. G., et al.: Poliomyelitis epidemic recurrence in the counties of the United States. Pub. Health Rep. 64: 1584 (1949).
(3) Kilham, L., et al.: Nonparalytic poliomyelitis and mumps meningoencephalitis. J. A. M. A. 140: 934 (1949).
(4) Dalldorf, G., and Sickles, G. M.: An unidentified, filterable agent isolated from the feces of children with paralysis. Science 108: 61 (1948).
(5) Melnick, J. L., et al.: A virus isolated from patients diagnosed as nonparalytic poliomyelitis or aseptic meningitis. Soc. Exper. Biol. \& Med. 71: 344 (1949).
(6) Armstrong, C.: Personal communication.
(7) Howitt, B. F.: Recovery of Coxsackie group of virus from human sources. Proc. Soc. Exper. Biol. \& Med. 73: 443 (1950).

# INCIDENCE 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 MAY 27, 1950

Reported cases of influenza continued to decline but remained above the 5 -year (1945-49) median. A total of 1,259 cases was reported for the current week as compared with 1,917 for the previous week, and 871 for the corresponding week in 1949 . The 5 -year median was 871 .

The cumulative cases of influenza for the first 21 weeks of the year were 239,478 , which may be compared with the corresponding totals of 70,721 for 1949, and 295,843 for 1947, the highest on record for the past 5 years. The corresponding 5 -year median was 133,138 .

A decrease from the previous week was noted in the incidence of whooping cough, from 3,018 reported cases to 2,852 for the current week. The 5 -year (1945-49) median was 1,914 . The cumulative total for the first 21 weeks of the year was 55,847 as compared with the corresponding total of 21,430 for the same period last year; and 43,471 for the 5 -year median.

The total number of reported cases of acute poliomyelitis was 103 for the current week, as compared with 94 last week, and 102 for the week ended May 13. The corresponding figure last year was 168; and the 5 -year (1945-49) median, 77. For the current week Texas reported 39 cases; and California reported a decrease from 16 to 9 for the preceding week.

Reported cases of typhoid and paratyphoid fever increased from 47 for the previous week to 75 this week. The 5 -year (1945-49) median was 65. On a cumulative basis for the first 21 weeks of the year there were 1,030 cases reported as compared with 988 for the corresponding period last year, and 1,080 for the 5 -year median. Tularemia increased from 16 cases last week to 18 this week, but remained below the 5 -year median of 20 .

Decreases compared with the preceding week were indicated for the following diseases: Diphtheria ( 72 to 67), measles ( 15,846 to 15,066 ), meningococcal meningitis ( 68 to 62 ), and scarlet fever ( 1,280 to 1,139 ).

Two cases of anthrax were reported, one each in Connecticut and New Jersey. No cases of smallpox were reported in the United States.

Commumicable Disease Charts All reporting States, November 1949 through May 27, 1950 ACUTE POLLOMYELITIS
 DIPHTHERIA


The upper and lower broken lines represent the highest and lowest figures recorded for the corresponding weeks in the 5 preceding years. The solid line is the median figure for the 5 preceding years. All three lines thave been smoothed by a 3 -week moving average. The dots represent numbers of cases reported for the weeks of 1949-50.



## FOREIGN REPORTS

## CANADA

Provinces-Notifiable diseases-Week ended May 13, 1950.Cases of certain notifiable diseases were reported by the Dominion Bureau of Statistics of Canada as follows:

| Disease | New-foundland | Prince Edward Island | Nova Scotia | New <br> Bruns- <br> wick | $\begin{aligned} & \text { Que- } \\ & \text { bec } \end{aligned}$ | Ontario | Manitoba | Sas-katchewan | $\begin{aligned} & \text { Alber- } \\ & \text { ta } \end{aligned}$ | British Co$\operatorname{lum}_{\text {bia }}$ | Total |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| Brucellosis. |  |  |  |  | 2 | 5 |  |  |  |  |  |
| Chickenpox |  |  | 9 | 1 | 157 | 263 | 11 | 18 | 53 | 111 | 623 |
| Diphtheria |  |  |  |  | 1 | 1 |  |  |  |  |  |
| Dysentery, bacillary - |  |  |  |  |  | 1 |  |  |  |  |  |
| Encephalitis, infectious |  |  |  |  |  | 2 |  | 1 |  |  |  |
| German measles. |  |  | 40 |  | 22 | 1. 104 | 2 | 109 | 143 | 366 | 1,786 |
| Influenza |  |  | 11 |  |  |  | 3 |  |  | 25 | 42 |
| Measles. |  |  | 1 | 1 | 682 | 564 | 31 | 31 | 45 | 193 | 1. 548 |
| Meningitis, menin- gococcal.............. |  |  |  | 1 | 1 | 2 |  |  |  |  |  |
| Mumps |  |  | 54 |  | 187 | 456 | 3 | 85 | 93 | 240 | 1,118 |
| Poliomyelitis....-.....- |  |  |  |  |  | 1 |  |  |  | 1 |  |
| Scarlet fever- | 3 |  | 2 |  | 67 | 31 | 4 | 8 | 55 | 30 | 200 |
| Tuberculosis (all forms) | 4 |  | 3 | 20 | 80 | 19 | 14 | 7 | 9 | 40 | 196 |
| Typhoid and para- typhoid fever |  |  | 1 |  | 9 | 2 |  |  |  | 2 | 14 |
| Venereal diseases: |  |  |  |  |  |  |  |  |  |  |  |
|  | 17 |  | 13 7 | 6 | 47 66 | 48 26 | 14 3 | 10 | 29 | 54 8 | 134 |
| Whooping cough. | 1 |  | 13 | 1 | 65 | 90 |  |  | 3 | 57 | 230 |

## NEW ZEALAND

Notifiable diseases-4 weeks ended March 25, 1950, and 5 weeks ended April 29, 1950.-Certain notifiable diseases were reported in New Zealand as follows:

| Disease | $4 \text { weeks ended Mar. 25, }$ |  | $5 \text { weeks ended Apr. 29, }$ |  |
| :---: | :---: | :---: | :---: | :---: |
|  | Cases | Deaths | Cases | Deaths |
| Actinomycosis. |  |  | 1 |  |
| Brucellosis..-- | 1 |  | 3 |  |
| Diphtheria | 7 |  | 11 |  |
| Dysentery* |  |  |  |  |
| Amebic.- | $\stackrel{2}{26}$ | 1 | 3 43 |  |
| Encephalitis, lethargic. |  |  | 3 | 1 |
| Erysipelas .-.........- | 8 |  | 9 | .-....... |
| Food poisoning | 258 |  | 62 |  |
| Hookworm disease. | 1 | - |  |  |
| Malaria | 1 |  |  |  |
| Meningitis, meningococcal | 11 |  | 15 | 3 |
| Poliomyelitis... | 22 |  | 10 |  |
| Puerperal fever... | 2 |  | 2 |  |
| Scarlet fever..... | 88 3 |  | 145 5 |  |
| Trachoma |  |  | 1 |  |
| Tuberculosis (all forms) | 150 | 46 | 219 | 53 |
| Typhoid fever... | 11 |  | 17 | . 3 |

## JAPAN

Notifiable diseases-4 weeks ended March 25, 1950, and accumulated totals for the year to date.-Certain notifiable diseases were reported in Japan as follows:

| Disease | $4 \text { weeks ended Mar. 25, }$ |  | Total reported for th year to date |  |
| :---: | :---: | :---: | :---: | :---: |
|  | Cases | Deaths | Cases | Deaths |
| Diarrhea, infectious. |  |  | 10 |  |
| Diphtheria. | 1,120 | 134 | 3, 581 | 395 |
| Dysentery, unspecified | 406 | 91 | 1,024 | 251 |
| Filariasis | 4, 5 |  | ${ }^{26}$ |  |
| Leprosy.. | 4, 56 |  | 15, 2116 |  |
| Malaria | 50 | 5 | 138 | 16 |
| Measles | 5,835 |  | 13,201 |  |
| Meningitis, meningococcal | 99 | 19 | 274 | 61 |
| Paratyphoid fever | 61 | 3 | 201 | 8 |
| Pneumonia. | 20, 251 |  | 59,321 |  |
| Poliomyelitis. | 117 |  | 364 |  |
| Puerperal infection | 69 |  | 211 |  |
| Scarlet fever. | 277 | 1 | 968 | 5 |
| Schistosomiasis | 35 |  | 72 |  |
| Smallpox--- | 1 |  | 3 |  |
| Tetanus.- | 121 |  | 325 |  |
| Trachoma. | 10, 197 |  | 26,360 |  |
| Tuberculosis.- | 31, 726 |  | 88, 569 |  |
| Typhoid fever | 205 176 | 28 | 702 | 110 |
| Typhus fever- | 176 | 14 | 671 | 43 |
| Venereal diseases: Gonorrhea | 12,789 |  | 36,907 |  |
| Syphilis... | 11,076 |  | 30, 172 |  |
| Whooping cough | 9, 605 |  | 31, 188 |  |

## reports of cholera, plague, smallpox, typhus fever, and Yellow fever received during the current week


#### Abstract

Note.-The following reports include only items of unusual incidence or of special interest and the occurrence of these diseases, except yellow fever, in localities which had not recently reported cases. All reports of yellow fever are published currently.

A table showing the accumulated figures for these diseases for the year to date is published in the Public Health Reports for the last Friday in each month.


## Cholera

Pakistan-During the week ended May 6, 1950, 5 fatal cases of cholera were reported in Chittagong, and 9 cases (all fatal) were reported during the week ended May 13. Four cases, with one death, were reported in Dacca during the week ended May 6.

## Plague

Indochina (French).-Plague has been reported in French Indochina as follows: Week ended May 6, 1950, 5 cases (3 deaths) in the Giadinh area, Cochinchina; week ended May 13, 5 cases in the port of Phanthiet, Annam.

Indonesia-Java.-In Jogjakarta, 9 fatal cases of plague were reported during the week ended April 29, 1950, and 11 fatal cases during the week ended May 6.

## Smallpox

Republic of Korea.-During the month of February 1950, 453 cases of smallpox, with 83 deaths, were reported in Korea, and 506 cases ( 78 deaths) were reported during March.

## Typhus Fever

Republic of Korea.-Typhus fever has been reported in Korea as follows: For the month of February 1950, 299 cases, 21 deaths; for March 1950, 428 cases, 36 deaths. During April, 49 cases were reported in the city of Seoul.

## Yellow Fever

Peru-On February 16, 1950, one fatal case of yellow fever was reported in Juanjui, San Martin Department.

DEATHS DURING WEEK ENDED MAY 27, 1950

|  | Week ended <br> May 27, 1950 | Corresponding week, 1949 |
| :---: | :---: | :---: |
| Data for 94 large cities of the United States: |  |  |
| Total deaths. | 9,133 | 9,008 |
| Median for 3 prior years | 9,008 |  |
| Total deaths, first 21 weeks of year | 204, 564 | 202,313 |
| Deaths under 1 year of age | 616 | 661 |
| Median for 3 prior years. | ${ }_{6}^{675}$ |  |
| Deaths under 1 year of age, first 21 weeks of year | 13,065 | 13, 731 |
| Data from industrial insurance companies: |  |  |
| $\xrightarrow{\text { Policies in force }}$ Number of death claims | $69,796,561$ 13,122 | $70,393,900$ 13,287 |
| Death claims per 1,000 policies in force, annual rate | 9.8 | 9.8 |
| Death claims per 1,000 policies, first 21 weeks of year, annual rate...- | 10.0 | 9.7 |


[^0]:    *From the Army Medical Department Research and Graduate School, Washington, D. C. The technical assistance of S.F.C. F. Hawk, S.F.C. W. O. Blair and P.F.C. D. Thorlton is gratefully acknowledged.
    Information contained in this paper was reported to the Commission on Immunization of the Armed Forces Epidemiological Board at its annual meeting, March 17, 1950.

[^1]:    1 We wish to thank Dr. J. A. Morris of this department for preparing the ferret antisera and Dr. B. Eddy of the Biologics Control Division, National Institutes of Health, for the mouse antisera.

[^2]:    The studies upon which this report is based were made under the auspices of the Department of Hygiene and Preventive Medicine, State University of Iowa, Iowa City.

[^3]:    ${ }^{1}$ The senior author is not a Salmonella carrier.

[^4]:    *Director, Bureau of Preventable Diseases, District of Columbia Department of Health.

