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PSITTACOSIS IN THE UNITED STATES

Incidence, Scientific Aspects, and Administrative Control Measures 1

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I. CASES REPORTED IN 1933 AND 1934

Since the last meeting of the Permanent Committee of the International Office of Public Hygiene, we have seen a steady decrease in the number of human cases of psittacosis in the United States. Whereas during the year 1932 there were reported 76 cases with 7 deaths in this country, only 15 cases with 4 deaths were reported during the year 1933 and 2 cases with 1 death in 1934 up to March 1.2 Of the 76 cases reported in 1932, 41, or 53.9 percent, occurred in California. Of the 15 cases reported in 1933, 10, or 66.6 percent, occurred in California. All reported human cases occurring in the United States were traceable to California-bred birds. It can be said to the credit of the California health authorities, however, that all cases occurring outside the State of California during 1933 appear to have been contracted from birds that were shipped from California in violation of the regulations of the State Department of Public Health.

As a result of the occurrence of human cases in Minnesota and Connecticut in 1933, due to the illegal shipment of birds out of California, those States declared an absolute embargo against the importation of parrakeets. Oregon and the Territory of Hawaii had previously made similar regulations. On March 1, 1934, the State of Maine also declared an embargo against the importation of shell parrakeets.

Following the establishment of a Federal interstate quarantine in September 1932 against the unrestricted shipment of psittacine birds, together with the quarantine and isolation of all psittacine birds in California, the occurrence of human cases abruptly ceased for a time. It was hoped that the measures taken to prevent the spread of the disease had been successful. From October 1932 to February 1933 no cases of human psittacosis were reported anywhere in the United States. Suddenly, however, in the late winter and early spring of

¹ Report prepared for presentation to the Permanent Committee of the International Office of Public Hygiene at the meeting in Paris in May 1934.

² Editorial Note.—Since this article was written, an outbreak of psittacosis has been reported in Pittsburgh, Pa. The actual number of cases is not known, but investigation has revealed that 25 cases (including suspected cases) and 10 deaths occurred between Feb. 14 and Mar. 16, 1934. The outbreak originated in a store where birds were sold.

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1933, 5 cases occurred in Los Angeles County in rapid succession and from entirely unrelated sources. Of these 5 cases, 4 proved fatal. Two officers of the United States Public Health Service had the privilege of seeing 3 of the 4 fatal cases before death and all 4 at autopsy. The epidemiological, clinical, and pathological data for each of these 4 cases are summarized in the following:

The first case in this series was J. Mc., male, age 59, a stonemason, living in Los Angeles. He had one parrakeet which had been in his possession for about 3 years. On February 18 his landlady captured a stray parrakeet in her back yard and placed it in the cage with the old bird. Ten days later the recently acquired bird died and was destroyed. Fifteen days after the new bird had been acquired the older bird died and was shown (by the Hooper Foundation laboratory in San Francisco) to be infected with psittacosis.

On February 24, 6 days after the stray parrakeet was captured, the patient suddenly became very ill and rapidly grew worse. He was removed to the Los Angeles County Hospital on February 27, at which time his temperature was 103.4° F., pulse 98, respiration 22. These figures did not vary greatly until shortly before death, when pulse and respiration became more rapid and the temperature lower.

On admission a diagnosis of pneumonia was made, but repeated examinations of the chest failed to show any appreciable decrease in resonance. Râles could be heard over the right base posteriorly and the X-ray showed considerable pulmonary infiltration which, as the disease progressed, spread over the entire right lung and part of the left. When seen by us, the patient was for the most part rational but extremely dull and apathetic, making conversation difficult. Contrary to the general rule, this patient began to expectorate thick, tenacious sputum very early in the course of the disease. The virus of psittacosis was recovered from this sputum both by the Hooper Foundation laboratory in San Francisco and the United States Public Health Service laboratory in Pasadena, Calif. The patient died 27 days after the onset of illness.

At autopsy both lungs were found to be involved throughout by what appeared grossly to be a diffuse confluent bronchopneumonia, beginning in the hilar regions and extending out fan-like toward the periphery but not involving the pleura. Examination both macroscopically and microscopically showed the density to be much less than in that of ordinary pneumonia, thus accounting for the resonant percussion note during life.

White mice were inoculated with emulsions of fresh lung tissues but the virus was not recovered from this source. Histological examination of the lung, however, easily revealed the "elementary bodies" or "L.C.L." bodies of psittacosis.

Aside from its clinical features this case clearly shows the incubation period in the man to have been 6 days and in the bird 15 days. It also shows that old healthy birds may be susceptible to psittacosis, though we know young birds to be more so. The ultimate source of the infection could not be traced, but it is interesting to note that about this time many aviary owners whose birds were in quarantine were releasing them rather than have them destroyed. This practice was condemned by the local health authorities as being a publichealth menace, and it seems that in this case at least their fears were well founded.

The second case was that of M. P., male, age 41, a blacksmith and wrestler by trade. This case differed from the average from the standpoint of age and excellent physical development of patient, mode of infection, and rapid fatal termination.

This man had a small aviary of about 20 parrakeets which he had started 3 years before and had added no new stock. About March 10, he bought a parrot from a local dealer, which is said to have bitten him on the finger soon after bringing it home. The man became ill on March 19, 8 days after being bitten, having a sudden onset of fever, chills, headache, and great prostration. The illness was diagnosed as pneumonia; but the case was not hospitalized, and clinical records were not available. The patient grew rapidly worse and died 7 days after onset of the disease. Shortly before death a specimen of sputum was obtained, from which the virus of psittacosis was recovered. Both the parrot which was said to have bitten the patient and 9 of the 20 parrakeets were shown by laboratory examination to be infected with psittacosis.

At autopsy no evidence of a bite wound could be found. Both lungs showed massive consolidation of denser consistency than is usually seen in psittacosis. As the body had been embalmed, we were unable to perform animal inoculation tests with the fresh lung tissue, but "L.C.L." bodies were readily demonstrated in the lung on histological examination.

This case aptly illustrates two important factors: One is the speed with which psittacosis can spread through a flock of psittacine birds, and the other is the great increase in virulence when the virus is introduced directly into the blood stream by the bite of an infected bird. This man, who was comparatively young and in the finest of physical condition, would ordinarily be conceded an excellent chance of recovery; but he succumbed in less than half the usual time required in cases where the infection is contracted through the respiratory tract.

The third case was W. F., male, age 73. This man raised parrakeets on his small ranch. He usually kept about 125 to 150 birds; and,

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as he attended them himself, it could not be learned whether or not he had recently acquired any new stock. It was learned, however, that a few weeks before taking sick he had found a parrakeet outside the cage and had placed it in with the others. After this patient had been taken sick, his daughter began taking care of the birds, and it was noticed that occasionally a bird would die. The owners sunposed this to be due to irregular feeding. However, as the man appeared to be ill with psittacosis, his aviary was visited by officials of the United States Public Health Service on March 31, 1933, and a parrakeet which had died on the previous day was secured and brought to the laboratory for examination. At autopsy this bird was grossly suspicious for psittacosis, having an enlarged liver with many areas of necrosis. The spleen was also enlarged to a diameter of 6 millimeters. A portion of the spleen and liver inoculated into white mice produced the typical lesions of psittacosis, and the liver of the bird on histological examination showed numerous virus bodies.

This patient was taken to the hospital on March 24, 1933, 3 days after the onset of his illness, and was extremely ill from the beginning. The characteristic disproportion between pulse and temperature rate was well illustrated. At time of admission to the hospital, the temperature was 102.6° F., rising to 104° F. the following day and remaining between that and 105° F. for the first 2 weeks of his illness. The pulse rate, however, on admission was only 60 and remained between 60 and 70 until the latter part of the second week of his illness, when it slowly rose to 120 and remained at that figure until death. When seen by us early in the second week of his illness the patient was comatose and had Cheyne-Stokes breathing. It appeared that death was but a few hours distant but the fact that he lived for nearly 2 weeks longer served again to emphasize the fact that it is impossible to make an accurate prognosis in this disease.

The illness of this patient was attended with considerable coughing throughout, with the production of a great deal of extremely tenacious sputum. We were unable to demonstrate the virus of psittacosis in the first specimen obtained, but were able to do so easily in the second specimen. It has frequently been demonstrated that virus may be present in one specimen and absent in another, although the physical nature of the two specimens may appear the same. This emphasizes the necessity of taking repeated specimens of sputum in all suspected cases.

Death occurred on April 13, 23 days from onset. At autopsy both lungs showed extensive involvement radiating out from the hilar region but not involving the pleura. The involved areas did not show the consistency of hepatization, but were of a rubbery nature, and a frothy purulent material could be expressed. An emulsion of the

fresh lung tissue readily produced the lesions of psittacosis in white mice. Sections of the lung examined histologically showed numerous "L.C.L." bodies. After death of this patient, all birds in his aviary were sacrificed by the owners and examination of these birds at the Hooper Foundation laboratory demonstrated that about 25 percent showed macroscopic evidence of psittacosis. Hence, in this case we have an unusually complete picture, in that virus of psittacosis was demonstrated, first, in the parrakeet, both by animal inoculation and direct histological examination; second, in the patient's sputum; third, in animal inoculation of fresh lung tissue emulsion, and fourth, by histological examination of the human lung and spleen.

The fourth and last fatal case in this series occurred in Pomona, Calif. (about 50 miles from Los Angeles), in May 1933.

A. C., a female, age 53 years, had been living with a family who had had one parrakeet for several months. A young male parrakeet was obtained to mate with the old female; and about 2 weeks after the young parrakeet had been acquired, the woman was taken sick. As psittacosis was suspected, both birds were turned over to the United States Public Health Service laboratory for examination. Autopsy showed that the older bird was healthy, but that the young bird was infected with psittacosis.

On admission to hospital, shortly after the onset, the patient complained of severe headache, chills, and pains in the back. There were also several nervous manifestations, consisting of parasthesia in the fingers and toes, and a nonproductive cough, described by the patient as a "nervous cough." She further stated, that she had a "fluttering sensation in her abdomen." Patient's temperature on admission was 103° F., and ranged between that and 104° F. until just before death, when it fell to normal. The pulse rate maintained a characteristic low level, ranging from 70 to 100 throughout, with the consistently relatively slow respiration rate of 20 per minute.

When seen by us on the seventh day after admission to the hospital, the patient was obviously extremely ill but conscious and rational. While apparently the outcome was likely to be fatal, her condition at that time, based on our previous experience, did not indicate that death would intervene for several days. However, the patient died within a few hours after having been seen by us.

At autopsy the lungs were strikingly similar to those observed in the three preceding cases. The color was a grayish-purple, and consolidation was found to be extensive in both lungs. On gross examination it was seen that the consolidation was central in type and did not extend to the pleura, there being a layer of crepitant tissue surrounding each consolidated area. On cut section, the consolidated area was grayish-red in color and exuded a mucopurulent material on slight pressure. It was further observed that April 4, 1994 456

the consolidated areas did not show the firm consistency of ordinary pneumonia, but that sections cut from consolidated areas floated on water.

The virus of psittacosis was obtained by inoculation of emulsions of both lung and spleen tissues into white mice. "L.C.L." bodies were also demonstrated in the lung by direct histological examination.

This case again emphasizes the considerable danger involved in acquiring immature parrakeets, especially from untested sources, and further demonstrates that the prognosis in human cases of psittacosis must be made with extreme caution.

II. SCIENTIFIC ASPECTS

Concerning the scientific developments in the studies on psittacosis, it must be admitted that fundamentally the disease remains almost as much of a mystery as it was at the time of our last meeting. it is caused by a filterable virus has of course been established since 1930. That the nature of the virus still remains a mystery is admitted by all investigators. The nature of the "L.C.L." bodies observed in both animal and human tissue infected with psittacosis is likewise unknown. Are they the virus or are they reactionary products? Are they bacterial or protozoan in nature? That they are the virus itself perhaps seems the more probable. Bedson has suggested that the virus goes through a fairly rapid developmental cycle at some stage of which it is virulent and at others avirulent or nearly so. Several observations of our own seem to indicate that this may be true. We have seen that it is difficult, if not impossible, at times to recover virus by filtration from material known to contain the virus. It is also obvious that the visible objects which we consider virus bodies are too large to pass through the pores of a Berkefeld N candle. For this reason it seems that at some time the virus must assume an ultra-microscopic form. That the virus in infected birds is extremely virulent over relatively short spaces of time has been seen on several occasions, when persons having contact with sick birds over a long period of time and others having only momentary exposure become sick almost simultaneously.

The technique of diagnosis of this disease has improved little if any since Krumweide, Rivers, and Berry, and other workers, demonstrated that white mice were susceptible to the disease and made excellent diagnostic animals. This procedure has been used exclusively by us and, except for the time required, has been entirely satisfactory and reliable. We have found sputum to be the only ante mortem material of value in making inoculation tests. If repeated specimens are taken, we rarely fail to demonstrate the virus if the case is psittacosis. We have found the patient's blood to be of little or no

value as inoculating material for diagnostic tests and have discontinued its use for this purpose. While formerly we considered a histologic examination of both bird and mouse tissue necessary before a diagnosis of psittacosis could be made, we now use this procedure largely as a confirmatory step. We have found that the relative ease with which "L.C.L." bodies can be demonstrated in fresh impression smears of the mouse spleen, using a modification of Castaneda's stain, together with the characteristic appearance of the liver, makes practical diagnosis possible within a few minutes after the mouse is autopsied. In this way the diagnosis of a human case can usually be confirmed in 5 to 10 days. However, negative results do not necessarily prove that the case is not psittacosis.

Rapid diagnosis can frequently be made in the suspected birds by direct examination. It has been found that, in general, parrakeets having spleens less than 4 millimeters in diameter are unlikely to be infected with psittacosis, and those having spleens over 4 millimeters are likely to be infected. This is by no means a hard and fast rule, however, and is used only as a convenience in assorting spleens for animal inoculation. Spleens under 4 millimeters may contain the virus and spleens may be enlarged to more than 4 millimeters by some other disease. Fresh impression smears of the bird spleen stained with a modification of Castaneda's stain frequently show the presence of "L.C.L." bodies and establish an immediate diagnosis. The failure to demonstrate "L.C.L." bodies in the bird spleen cannot be considered as an indication that psittacosis is not present, however, and animal inoculation must be made in all cases.

It is believed that a correct bedside diagnosis is more frequently arrived at than was the case 2 years ago. There is no doubt that a great many cases of psittacosis were missed in the first few years after the disease became known in the United States. Extensive publicity by the lay press, together with considerable information given out through medical literature, has brought the disease to the attention of practically every physician and health officer in the country. It is now probable that instead of cases being missed, some are erroneously diagnosed psittacosis. Whether or not that be true, the number of reported cases has steadily and materially decreased.

There have been no new developments in the treatment of psittacosis. It has not been possible to demonstrate protective antibodies for psittacosis virus in human convalescents, or in artifically immunized animals. For this reason the routine use of human convalescent serum has for the most part been discontinued. This would seem to discredit the popular belief that one attack of psittacosis conveys permanent immunity to the individual. However, we have no knowledge of a second attack of psittacosis occurring in human beings.

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With our limited knowledge of psittacosis, our most effective weapon in dealing with the disease is in control of the birds themselves. A recent important observation in this respect is that young birds are more susceptible to psittacosis and, therefore, a more potent source of danger. With this fact established, we have required that all psittacine birds be at least 8 months of age before being shipped in interstate commerce. It is believed that this regulation alone has been of vital importance in reducing the incidence of the disease.

III. ADMINISTRATION OF PSITTACOSIS CONTROL

At the time of the meeting in April and May 1932, the United States Government had already instituted measures to prevent the importation of psittacosis. At the time of the 1929–30 epidemic, it appeared that most human cases had been contracted from parrots and that these birds were the principal vectors. Consequently, the first control measure instituted by the United States was the placing of an embargo, by Executive order, on the importation of parrots. This embargo was made effective January 24, 1930, and was absolute except under certain conditions prescribed by the Secretary of the Treasury, which allowed the importation of parrots only after 15 days' quarantine detention and careful inspection by an official of the United States Biological Survey to determine the freedom of the birds from psittacosis.

By 1932 it had been determined that parrots were not the only vectors of psittacosis, or indeed the most important, that role having been assumed by the shell parrakeet (Melopsittacus undulatus), apparently due to its greater frequency in commerce, and that all, or nearly all, psittacine birds were actual or potential vectors of psittacosis. Consequently, on October 6, 1932, the original Executive order of January 24, 1930, which had placed limitations on the importation of parrots only, was further amended and extended to include all the psittacidae, naming specifically all birds commonly known as parrots, Amazons, Mexican double heads, African gravs, cockatoos, macaws, lories, parrakeets, love birds, and all similar birds. At the same time it was provided further that the importation of all psittacine birds should be in accordance with strict sanitary measures. which were essentially those recommended in the report of the Commission on Psittacosis and approved by the Permanent Committee of the Office International d'Hygiène Publique at its meeting of May 4, 1932.

The amendment to the Federal Quarantine Regulations of October 6, 1932, further provided that the ports of entry for importation of psittacine birds into the United States shall be limited to such ports only as have quarantine detention facilities, and that each shipment

shall be detained at such stations under observation for a period of 15 days. If any death or serious illness occurs in the birds during the 15-day period they are not released, and the detention period is extended over another period of 15 days from the date of last illness or death. If psittacosis is discovered in any shipment of psittacidae, as proved by laboratory examination, the shipment is disposed of in such manner as the Surgeon General of the Public Health Service may deem necessary. In practice, such shipments usually are deported or destroyed.

It has been established that both in naturally and artificially infected birds, the incubation period may be many weeks and that the disease may remain latent for a very extended period of time. For this reason it might appear that the 15-day detention period would be inadequate, and this view has been held by some investigators. Practically, however, the results seem to have been satisfactory, as no human cases of psittacosis are known to have occurred from contact with recently imported birds since October 1932. Since psittacine birds are extremely sensitive to changes of location and climate, any incipient disease is almost certain to become apparent during the 15-day detention period, and thus automatically increases the length of time in which the birds are held under observation.

During the years 1931 and 1932, sporadic cases of psittacosis due to exposure to infected parrakeets occurred in a great many different States. Investigation pointed to California as the origin of the birds in nearly every case. The California State health officials, believing that psittacosis was being introduced into the State from outside sources, adopted a resolution on February 13, 1932, prohibiting the importation, into the State of California, of all birds of the psittacine family for a period not to exceed 6 months. As human cases continued to occur after the State embargo became effective, further study of the problem became necessary.

Early in 1932 the California State Department of Health, with the assistance of the United States Public Health Service, began an extensive epidemiological study of the psittacosis problem. It soon became apparent that parrakeet breeding was an industry of major proportions in California. Eleven hundred and forty aviaries with upward of 100,000 parrakeets were inspected and registered. Seventy-six and nine tenths percent of these were located in seven southern California counties, 54.9 percent being in Los Angeles County alone. The remaining 23.1 percent was distributed over 30 northern counties. The great majority of these aviaries was of the small "back-yard" variety and, hence, not readily amenable to regulation.

During 1932, in the course of investigations of several human cases, it was determined by laboratory tests that psittacosis had become endemic in California aviaries. Further laboratory tests revealed that the number of infected aviaries was surprisingly high, being

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reported by Dr. K. F. Meyer of the Hooper Foundation laboratory, where most of the tests were performed, as being close to 60 percent.

Attempts by the California State Department of Public Health to limit the spread of the disease by regulation of breeding and marketing activities were met by such a storm of protest from the aviary owners as seriously to handicap their efforts. Since cooperation of some of the parrakeet breeders and dealers could not be obtained, and human cases of psittacosis traceable to California-bred birds continued to occur in widely separated areas of the United States, it became necessary for the United States Government to impose limitations on the interstate shipment of these birds. Consequently, on September 28. 1932, a provision was added to the Interstate Quarantine Regulations prohibiting the interstate transportation by common carrier of any bird or birds of the parrot family unless such shipments were accompanied by a certificate of the State health authorities stating that such birds were, to the best of their knowledge and belief, from sources free from psittacosis. As it was difficult to determine that aviaries were free from psittacosis, certificates of health were not freely given and the interstate shipment of parrakeets almost ceased. That these measures were of considerable protective value is seen in the greatly reduced number of cases occurring outside the State of California.

Diligent efforts to control the spread of psittacosis within the State have been made by the California authorities since early in 1932. In March of that year, all aviaries found to contain infected birds were placed in quarantine for an indefinite period. Those found to be free from psittacosis were permitted to sell their birds. In addition, all persons or firms engaged in the breeding or commerce in psittacine birds were required to register with the State Department of Public Health. Definite instructions were given as to the keeping of records of all transactions in such birds, as well as a record of all cases of sickness and death. Regulations further gave instructions in the sanitary housing, care, and shipment of such birds. Itinerant bird vendors, who had previously been responsible for several human cases of psittacosis, were required to obtain a permit from the health officials before offering any psittacine birds for sale.

While known infected psittacine birds had been under quarantine since March, it was felt that psittacosis was still being disseminated from unquarantined sources; and so, on October 6, 1932, in addition to the quarantine of infected birds, all psittacine birds in the State of California were placed in isolation on the premises where located and not moved therefrom except by written permission of the local health officers. This regulation was modified in December 1932 to permit local health officers to issue certificates of health for the interstate shipment of all psittacine birds other than parrakeets, provided such birds had not been in contact with parrakeets for a period of 90 days.

By the first of the year 1933, much of the confusion and uncertainty that had attended the earlier attempts to regulate the bird-breeding industry in California had been overcome and a practical working procedure decided upon. Breeders were required to maintain at least 3 pens separated by a distance of at least 5 feet. The first pen was maintained for breeding purposes only; the second pen for maturing the birds to the age of 7 months. At the age of 7 months the birds were given a leg band on which was stamped the registration number and code number of the owner, and placed in the third or isolation pen for a period of 30 days. At the end of the 30-day period, the birds were inspected by a health officer and a certificate of health given for their release. At the same time the owner was required to sign an agreement to the effect that if any case or cases of human psittacosis were traced to his aviary, and laboratory examination of 10 percent of his birds proved the presence of psittacosis, such aviary was to be destroyed and his certificate of registration revoked.

That these measures to prevent the spread of psittacosis were attended with considerable success is seen in the greatly reduced incidence of the disease during the year 1933. On several occasions, however, certificates were fraudulently altered and young and sickly birds were shipped out of the State, resulting in human cases of psittacosis. It then became apparent that more stringent methods of control would have to be instituted, and on December 20, 1933, the United States Interstate Quarantine Regulations were amended to require that no birds of the psittacine family could be shipped in interstate commerce unless such birds be at least 8 months old and be accompanied by a certificate of health signed by the State health officer stating that to the best of his knowledge and belief they are from a source free from psittacosis, such certificates to be granted after the usual inspection supplemented by such laboratory tests as the certifying authority may deem necessary. In the future it is intended that certificates shall be granted only to birds from laboratory-tested aviaries, and that aviaries found to be infected shall be voluntarily destroyed or placed in permanent quarantine.

In February 1934 an improved type of health certificate was adopted by the California State Department of Public Health, which describes in detail the shipment for which issued. This certificate is executed in quadruplicate and sworn to by the shipper. One copy is sent to the central State health office, 1 to the health officer at point of destination, 1 becomes the property of the common carrier, and 1 is retained by the issuing office. This improved certificate not only prevents the shipment of unauthorized birds but serves to advise the health officer at point of destination of the arrival of such birds. Having thus been informed of the arriving shipment, all local health officers may refuse its admission to their jurisdiction if deemed advisable.

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In conclusion, it must be said that although efforts to suppress the spread of psittacosis in the United States have met with constant opposition and have been attended with many technical difficulties, the results obtained have been quite gratifying. Whether the decreased incidence of the disease has been entirely due to international efforts at control, or whether some other factor is involved, it is impossible to say. In any case, the fight is not over. We know little more of the intrinsic nature of the disease now than was known 2 years ago.

To what extent the disease may spread under favorable conditions is likewise unknown. Certainly few diseases can claim a more diversified list of susceptible species. It has been proved that a considerable number of the smaller nonpsittacine species of birds are susceptible. We have recently demonstrated that chickens are readily susceptible experimentally both by inoculation and by feeding of infected material. That the disease might become an economic as well as a public-health problem seems entirely possible.

EFFECT OF ALUM-PRECIPITATED RAGWEED POLLEN EXTRACT ON GUINEA PIGS

By W. T. HARRISON, Surgeon, United States Public Health Service

It has been shown that guinea pigs may be readily sensitized by the injection of extracts prepared from various plant pollens. These animals react in the usual manner, presenting characteristic symptoms of anaphylactic shock when later injected intravenously with extracts of the pollen to which they have been sensitized. Sublethal doses will bring about desensitization so that the animal will, for the usual period, fail to react to the full shocking dose.

The precipitation of crude toxoid by the addition of potassium alum yields a product which is a much more effective immunizing agent, both in man and in animals, than the crude toxoid from which the precipitate was obtained. This increased efficiency has been generally attributed to the very slow absorption of precipitated toxoid with resulting continued stimulation of the immunity mechanism over a comparatively long period, since induration at the site of injection may be detected for as long as 6 to 8 weeks.

An attempt has been made to apply this principle to ragweed pollen extracts in the hope that slow absorption of the precipitated extract would permit the injection of larger amounts in fewer doses and at greater intervals. Precipitated extracts have been prepared and attempts have been made to desensitize guinea pigs previously sensitized by injection with an aqueous extract of giant ragweed pollen.

The aqueous extracts were prepared by extracting the dry pollen for 7 days with a solution containing 2.5 grams of sodium chloride, 2.7 grams of sodium bicarbonate, and 5 cc of phenol per liter, and then filtering. To prepare the precipitate, potassium aluminum sulphate 1.1 percent was added to the aqueous extract and the golden-yellow precipitate washed with the extractive fluid and made up to original volume. The precipitate tended to settle out on standing, but the supernatant liquid remained clear and colorless.

Guinea pigs were sensitized by injecting intraperitoneally 1 cc of a 2-percent aqueous extract. After 4 weeks, 3 of these pigs received subcutaneously 1 cc of precipitated extract and 3 were reserved as controls. After an additional 10 days all animals were injected intravenously with 2 cc of a 4-percent aqueous extract. The 3 control pigs showed slight to moderate symptoms, rubbing of nose, coughing, roughing of hair of back and neck, followed by prompt recovery, while of the 3 pigs which received a "desensitizing" dose of 1 cc precipitated extract all showed immediate severe symptoms, 1 died from typical anaphylaxis, the others recovered slowly. In these pigs the indurated nodule at the site of the injection of the precipitated extract 10 days previous to the shocking dose was still very noticeable, showing that all of the injected material had not been absorbed.

A 1-percent aqueous extract was prepared in the usual way, and one half was precipitated by the addition of alum and made up to original volume. A series of guinea pigs was injected subcutaneously with 1 cc, half of them receiving the aqueous extract and half the precipitated extract. After 6 weeks, 5 of each group were injected intravenously with 1 cc of a 2-percent aqueous extract. Of the pigs sensitized with the aqueous extract, 3 showed no symptoms and 2 showed mild symptoms. Of those sensitized with the alum-precipitated extract, 3 showed severe symptoms, 1 dying in 3 minutes of typical anaphylaxis, and 2 showed moderate symptoms. These pigs were sick after the reaction had subsided, recovered slowly, and could be separated readily by a disinterested observer from those sensitized with the aqueous extract.

An effort was next made to determine the amount of potassium alum that could be added to an aqueous pollen extract without interfering with its desensitizing action. Since earlier experiences had shown that alum-precipitated extract is a much better sensitizing agent than aqueous extract of the same strength, a series of pigs was sensitized by subcutaneous injection with 1 cc of a 1-percent alum-precipitated extract. Alum (0.1, 0.2, 0.3, 0.4, 0.6, and 0.8 percent) was added to a 2-percent aqueous extract, and 1 cc of each alum dilution was injected subcutaneously in each of two pigs of the sensitized series 25 days after the sensitizing dose. After an additional 5 days all pigs received intravenously a shocking dose of 1 cc of a 2-percent

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aqueous extract. Pigs receiving 0.1, 0.2, and 0.3 percent of alum extract were completely desensitized, and one of the pigs receiving 0.4-percent alum extract showed slight symptoms, but those receiving 0.6-percent and 0.8-percent alum extracts were very sensitive, showing immediate severe symptoms. Two pigs which had received only the original sensitizing dose of precipitated extract showed severe symptoms following the shocking dose.

The desensitizing value of these alum extracts seemed to be in inverse proportion to the amount of alum added and the amount of induration following the desensitizing dose. Six tenths and 0.8 percent alum caused an indurated nodule which was still present 5 days after injection.

CONCLUSIONS

- 1. Alum-precipitated ragweed pollen extract is a very effective sensitizing agent in guinea pigs. This solid form is much more effective than the same amount of extract in aqueous solution. Guinea pigs with the precipitated extract in the abdominal wall for 10 days were still very sensitive to a shocking dose given intravenously.
- 2. It is probable that the slow absorption of precipitated pollen extract more closely approaches the natural method by which humans become sensitive to plant pollens.
- 3. Addition of alum, in concentration as high as 0.3 percent, to ragweed pollen extract does not interfere with its desensitizing properties. It is possible that this small amount might slow absorption sufficiently to permit injection of larger doses in hypersensitive persons.

COURT DECISIONS ON PUBLIC HEALTH

City held liable for damages resulting from operation of sewage disposal plant.—(Texas Court of Civil Appeals; City of Tyler v. House et ux., 64 S.W.(2d) 1007; decided Oct. 26, 1933.) An action was brought against a city for damages alleged to have resulted from the operation of the city's sewage disposal plant. The jury's findings established that the plaintiffs, who owned a farm in the vicinity of the disposal plant, had been caused material discomfort and annoyance in the occupation and enjoyment of their home and premises and that the rental value of their farm had been materially reduced. The trial court rendered judgment for the plaintiffs upon the jury's verdict, and the city appealed.

Some of the points decided by the court of civil appeals were as follows: (a) It could be safely stated as the law in Texas that a city was liable in damages to neighboring property owners when it constructed and operated on its premises a sewage disposal plant which polluted the air and produced such discomfort and annoyance as to

impair the comfortable enjoyment of such neighboring property by persons of ordinary sensibilities, and that this was true irrespective of any question of negligence on the part of the city in the construction and operation of its plant; (b) likewise, a city would be held liable when it permitted the filth and waste from its sewage plant to escape into a stream and be thereby carried and spread upon the lands of another to his injury; and (c) a temporary injury to land was measured by the reduced rental value of the property since the time complained of.

The trial court's judgment was affirmed.

Compensation granted under workmen's compensation act for death from tularaemia.—(Georgia Court of Appeals, Division No. 1; Great Atlantic & Pacific Tea Co. v. Wilson, Wilson v. Great Atlantic & Pacific Tea Co., 171 S.E. 827; decided Nov. 11, 1933.) A claim under the workmen's compensation act was brought by a widow for the death of her husband from tularaemia. The deceased had been employed as the manager of the meat department in a retail store. An award in favor of the claimant was made by the director of the department of industrial relations and was affirmed by the superior court. On appeal to the court of appeals the action taken below was affirmed, the appellate court summing the matter up as follows:

In conclusion, we are of the opinion, after reading many authorities and after a careful scrutiny of the evidence, that the commissioner [director] was authorized to find that the deceased was injured by cutting his hand while in the course of his employment, that the disease of tularaemia was contracted from rabbits handled in his place of work, and that such disease was the natural and unavoidable result of the accident and was the contributing cause of his death. * * *

DEATHS DURING WEEK ENDED MAR. 17, 1934

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

	Week ended Mar. 17, 1934	Corresponding week, 1933
Data from 86 large cities of the United States: Total deaths Deaths per 1,000 population, annual basis Deaths under 1 year of age per 1,000 estimated live births Deaths under 1 year of age per 1,000 estimated live births Deaths per 1,000 population, annual basis, first 11 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 11 weeks of year, annual rate	9, 012 12. 6 625 58 12. 7 67, 590, 873 16, 012 12. 4 11. 1	8, 676 12.1 647 1 55 12.4 68, 819, 116 13, 721 10.4 11.2

¹ Data for 81 cities.

PREVALENCE OF DISEASE

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

UNITED STATES

CURRENT WEEKLY STATE REPORTS

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

Reports for Weeks Ended Mar. 24, 1934, and Mar. 25, 1933

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended Mar. 24, 1934, and Mar. 25, 1933

	Diph	theria	Infl	lenza	Mea	sles	Mening meni	ococcus ngitis
Division and State	Week ended Mar. 24, 1934	Week ended Mar. 25, 1933	Week ended Mar. 24, 1934	Week ended Mar. 25, 1933	Week ended Mar. 24, 1934	Week ended Mar. 25, 1933	Week ended Mar. 24, 1934	Week ended Mar. 25, 1933
New England States: Maine	15 15 1 8	1 17 2 6	1	2 1 5 1 19	54 255 17 2, 177 7 26	44 375 240	0 0 0 2 0	0 0 0 1 0
New York. New Jersey. Pennsylvania East North Central States:	52 30 59	76 22 73	1 19 24	1 36 9	1, 411 483 2, 449	3, 903 1, 716 1, 176	8 3 2	3 1 6
Ohlo	25 15 32 18 10	40 24 48 18 7	29 46 46 6 41	10 90 32 12 64	904 1, 525 1, 908 141 1, 363	639 112 398 823 390	3 3 14 0 2	0 10 29 3 0
Minnesota Iowa Missouri Missouri North Dakota South Dakota Nebraska Kansas South Atlantic States:	4 11 48 9 5 5 7	27 11 30 9 4 13 5	1 12 244 5 10 4	2 22 1 2 3	287 291 881 113 571 225 263	1, 326 5 250 21 3 27 309	2 3 4 1 0 1 3	2 0 1 4 0 0
Delaware Maryland District of Columbia Virginia West Virginia North Carolina South Carolina Georgia Florida	2 8 9 27 8 18 8 10 7	1 8 3 13 14 17 7 8 5	39 39 47 586	24 1 12 64 751 319 10	221 1, 055 711 1, 290 92 3, 384 546 1, 995 243	7 12 5 480 276 509 171 64 57	0 1 0 6 3 0 0	0 0 1 2 0 0 0 1

See footnotes at end of table.

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Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended Mar. 24, 1934, and Mar. 25, 1933—Continued

	Diph	theria	Influ	ienza	Mea	sles	Mening	gococcus ingitis
Division and State	Week ended Mar. 24, 1934	Week ended Mar. 25, 1933	Week ended Mar. 24, 1934	Week ended Mar. 25, 1933	Week ended Mar. 24, 1934	Week ended Mar. 25, 1933	Week ended Mar. 24, 1934	Week ended Mar. 25, 1933
East South Central States: Kentucky	. 8 . 13 14 . 5	6 9 14 5	49 99 118	53 105 121	636 1, 157 705	130 53 15	1 2 1 0	0 2 2 4
Arkansss. Louisiana Oklahoma Teras Mountain States:	7 27 18 109	9 17 9 132	42 18 94 422	48 33 56 147	681 408 563 1, 461	152 31 77 1, 180	0 0 4 6	3 1 2 1
Montana (Idaho (Wyoming (Colorado New Mexico Arizona	1	4 1 14 3	3 21	5 31	62 179 50 299 42 61	57 32 4 11 10 33	0 0 0 1 1	1 0 0 0 2 0
Utah [†] Pacific States: Washington Oregon ⁴ California	43	3 9 55	25 54 45	3 42 50	542 196 142 1, 158	37 64 1, 378	0 1 0 2	0 1 0 7
Total	713	799	2, 193	2, 190	33, 230	16, 604	80	92
	Polion	yelitis	Scarlet	fever	Smal	lpox	Typhoi	d fever
Division and State	Week ended Mar. 24, 1934	Week ended Mar. 25, 1933	Week ended Mar. 24, 1934	Week ended Mar. 25, 1933	Week ended Mar. 24, 1934	Week ended Mar. 25, 1933	Week ended Mar. 24, 1934	Week ended Mar. 25, 1933
New England States: Maine New Hampshire Vermont Massacusetts Rhode Island Connecticut Middle Atlantic States: New York New Jersey Pennsylvania East North Central States: Ohio Indiana Illinois Michigan Wisconsin West North Central States: Minnesota Iowa Iowa Minnesota Iowa South Dakota North Dakota Nebraska Kansas South Atlantic States: Delaware Maryland District of Columbia Vermise	0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0	0 0 0 0 0 0 0 1 0 0 0 1 0 0 0 0	8 15 9 302 61 81 947 220 674 712 924 713 265 84 84 123 38 18 18 38 92	8 25 26 456 31 147 1, 110 354 1, 069 635 175 535 603 154 109 54 78 15 19 42 65	0 1 0 0 0 0 0 0 0 0 0 0 1 1 6 3 3 7 2 4 7 3 0 4 7 3 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0	0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0	6 0 1 1 0 2 6 4 6 1 1 1 2 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0	100044000001116688322551
Virginia West Virginin North Carolina South Carolina Georgia Florida	0 0 0 0 0	0 1 0 0 0	47 87 40 1 14 3	63 31 51 3 7	0 0 0 2 1	0 1 1 0 3 0	0 2 6 1 6 8 6	1 0 5 8 2 5 3

See footnotes at end of table.

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended Mar. 24, 1934, and Mar. 25, 1933—Continued

	Polion	nyelitis	Scarle	t fever	8ma	llpox	Турьо	id fever
Division and State	Week ended Mar. 24, 1934	Week ended Mar. 25, 1933	Week ended Mar. 24, 1934	Week ended Mar. 25, 1933	Week ended Mar. 24, 1934	Week ended Mar. 25, 1933	Week ended Mar. 24, 1934	Week ended Mar. 25, 1933
East South Central States:								
Kentucky	0	0	33	64	0	0	1	
Tennessee.	Ŏ	l i	34	. 41	l š	Ž	1 2	8 2 10
Alabama	Ŏ	i ī	5	13	Ŏ	14	3	ž
Mississippi ³	Ŏ	Ō	4	3	2	0	4	10
West South Central States:		1		1	_	_		
Arkansas	0		6	1 8	0	15	3	3
Louisiana	Ŏ	Ιŏ	30	11	i	ō	14	7
Oklahoma •	ě	l ŏ	16	15	i i	7	2	i
Texas 3	ž	l i	73	37	27	Ř	12	12
Mountain States:	_	_		•		•		
Montana 4	0	n	11	10	0	0	0	5
Idaho 4	2	Ŏ	1	7	13	6	ĭ	ĭ
Wyoming 4	ō	Ĭ	8	8	Ō	ň	Õ	î
Colorado.	. ŏ	ň	20 20	11	Ă	ň	ŏ	î
New Mexico	ĭ	ĭ	19	17	ō	ň	ŏ	i
Arizona	ō	ō	25	23	ŏ	ŏ	š	ñ
Utah 2	ň	ŏ		-6	ŏ	ň	ត់	ň
Pacific States:	•	•	•	•		•	•	·
Washington	1	0	68	61		8	2	0
Oregon 4	â	ŏ	30	29	š	2	2	. 3
California	7	8	216	176	ä	48	7	8
Total	19	16	6, 430	6, 549	144	231	147	163

SUMMARY OF MONTHLY REPORTS FROM STATES

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

State	Menin- gococ- cus menin- gitis	Diph- theria	Influ- enza	Ma- laria	Mea- sles	Pel- lagra	Polio- mye- litis	Scarlet fever	Small- pox	Ty- phoid fever
February 1934										
Alabama	4	105	856	55	1, 903	11	1	96	2	11
ArizonaCalifornia	.3	16	122		153	1	1	103	3	4
Georgia	11 5	170 83	177 846	6 51	6, 334	9 21	24	1,000	14	29 30 3 16 5
Idaho	1	လို	020	91	7, 283 453	41	0	38 48	0 29	30
Illinois	31	136	174	7	2,416		1	2, 423	15	18
Iowa	4	32	49		481		2	299	22	10
Louisiana	ī	91	54	45	489	6	ī	111	12	35
Minnesota	3 2	96	9		807		ī	275	28	6
Montana	2	14	169		78		Õ	79	Õ	Š
North Carolina	5	103	290		11, 164	13	2	213	i	3
Oregon		12	204		250		1	178	18	3
Rhode Island		15			22		0	66	0	0
South Dakota	2	4	43		1, 984		1	57	2	1
Tennessee	.7	52	598	40	3, 254	4	1	203	4	18
Texas	16	589 11	2, 874	788		56	8	607		95 8
Washington	•	11	70		932		2	245	16	8

New York City only.
 Week ended earlier than Saturday.
 Typhus fever, week ended Mar. 24, 1934, 12 cases, as follows: North Carolina, 1; Georgia, 5; Texas, 6.
 Rocky Mountain spotted fever, week ended Mar. 24, 1934, 5 cases, as follows: Montana, 1; Idaho, 1; Wyoming, 1; Oregon, 2.
 Exclusive of Oklahoma City and Tulsa.

February 1934		Lead poisoning:	Cases 3	Septic sore throat—Con.	Cases
Actinomycosis:	Cases	Leprosy:		Tennessee	
Illinois	1	California	1	Washington	
Anthrax:	_	Washington	1	Tetanus:	
California	1	Lethargic encephalitis:		Alabama	. 4
Botulism:	3	AlabamaCalifornia	5 4	California	1 2
Montana Chicken pox:	٠	Georgia	i	Illinois Tennessee	
Alabama	223	Illinois	4	Trachoma:	•
Arizona	172	Iowa	4	Arizona	31
California	2, 861	Louisiana	2	California	. 7
Georgia	227 30	Minnesota	1	Georgia	
Idaho Illinois		Oregon	1	Illinois	
Iowa	337	Washington	3	Oregon.	
Louisiana	100	Milk sickness:	_	Tennessee	
Minnesota	676	Illinois	1	Trichinosis:	
Montana	127 811	Mumps:	56	California	
North Carolina Oregon	205	Alabama	19	Illinois	3 6
Rhode Island	184	California		Tularaemia:	U
South Dakota	81	Georgia	229	Alabama	3
Tennessee	220	Idaho	6	Arizona	1
Washington	473	Illinois		Georgia	.8
Conjunctivitis:	3	Iowa Louisiana	228	Illinois Louisiana	10
Georgia Dysentery:	0	Montana	2	Minnesota	9
Alabama (amoebic)	4	Oregon.	15	Montana	5 2 1
Arizona	3	Rhode Island	4	North Carolina	4
California (amoebic)	31	South Dakota	94	Tennessee	4
California (bacillary)	11	Tennessee	293	Typhus fever:	20
Georgia (amoebic) Georgia (bacillary)	1 9	Washington Ophthalmia neonatorum:	480	Alabama	30 29
Illinois (amoebic)	50	California	1	Illinois	1
Illinois (bacillary)	4	Illinois	2	Louisiana	1
Illinois (carriers)	232	Tennessee	6	North Carolina	3
Louisiana	.5	Paratyphoid fever:	2	Undulant fever:	•
Minnesota (amoebic)	17 1	Georgia Louisiana	1	Alabama Arizona	2 2
Montana (amoebic) South Dakota (amoe-	•	Oregon	i	California	12
bic)	1	Tennessee	ī	Georgia	3
Tennessee	15	Texas	8	Illinois	4
Washington (amoebic)	1	Puerperal septicemia:		Iowa	5
Washington (bacillary)	6	Illinois Oregon	11	Louisiana Minnesota	2 4
Food poisoning: California	5	South Dakota	i	Montana	ī
German measles:	·	Washington	î	North Carolina	1 3
Alabama	257	Rabies in animals:		Oregon	2
Arizona	261	Alabama	72	Washington	3
California	392 73	California Illinois	78 19	Vincent's infection: Illinois	30
IllinoisIowa	637	Louisiana	9	Iowa	ĩ
Montana	6	Washington	10	Oregon	9
North Carolina	21	Rabies in man:	1	Tennessee	12
Rhode Island	2	Idaho	1	Whooping cough:	404
Tennessee	70 4	Illinois	2	AlabamaArizona	481 132
Washington	. =	fever:	- 1	California	
Granuloma, coccidioidal: California	4	Montana	4	Georgia	260
Hookworm disease:	-	Oregon	1	Idaho	10
California	1	Scabies:	_ [Illinois	
Georgia	384	Montana	3	Iowa	116
Louisiana	55	Oregon Tennessee	21	Louisiana Minnesota	28 173
Impetigo contagiosa:		Washington	8	Montana	45
Arizona	10	Septic sore throat:	-	North Carolina	1, 165
Illinois	.4	Arizona	3	Oregon	131
Montana Oregon	14 55	California	26	Rhode Island	84
Tennessee	1	Georgia	20 47	South Dakota	20 98
Jaundice, epidemic:	-	Illinois Montana	3	Tennessee Washington	647
California	1	North Carolina	7	Yaws:	
Montana	7	Oregon	15	California	1

WEEKLY REPORTS FROM CITIES

City reports for week ended Mar. 17, 1954

[This table summarises the reports received regularly from a selected list of 121 cities for the purpose of showing a cross section of the current urban incidence of the communicable diseases listed in the table. Weekly reports are received from about 700 cities, from which the data are tabulated and filed for reference]

	Diph-	Infl	nenza	Mea-	Pneu-	Scar- let	Small-	Tuber-	Ty- phoid	Whooping	Deaths,
State and city	theria cases	Cases	Deaths	ales cases	monia deaths	fever cases	pox cases	culosis deaths	fever cases	cases	all causes
Maine: Portland	0		0	1	4	4		0	1	40	36
New Hampshire:				ŀ	1		l			ŀ	
Concord Manchester	0		0	32 9	1	0	0	0	0	3 0	14 11
Nashua	ĭ		Ō	i	Ō	2	0	Ö	Ö	9	
Vermont:	0		0	0	0	1		1	0	0	1
Burlington Massachusetts:	0		0	0	0	2	0	0	0	5	8
Boston	8		o l	850	33	47	9	7	1	87	243
Fall River	0		0	1 3	1 4	9		1	0	3 13	35 37
Springfield	ŏ		ĭ	. 20	8	10	ŏ	i	ŏ	10	42
Rhode Island: Pawtucket	0		0	0	0	2		0	0	0	0
Providence	ŏ		Ŏ	Ŏ	4	0	ě	2	0	0	67
Connecticut: Bridgeport	0	2	2	8	1	18	0	1	0	4	30
Hartiora	1 0		8	0	4	9	0	0	0	0	25 46
New Haven	•		ľ	0	"	•	ľ	1	•	•	70
New York: Buffalo	8		o	210	21	27	0	6	0	28	150
New York	24	29	15	82	182	340	0	81	4	106	1,668
Rochester Syracuse	1		1 0	1 3	9 3	50 7	0	2 1	0	42	65 57
New Jersey:		,			6	5	0	2	0	0	35
Camden Newark	3	1 4	0	124 5	6	27	Ò	ī	Ō	45	91
Trenton	0		0]	84	8	9	0	3	0	5	45
Pennsylvania: Philadelphia	5	10	5	1, 441	60	117	0	25	0	66	548
Pittsburgh Reading	5 1	4	1 0	107	32 6	32 7	0	4	0	37 13	168 33
Scranton	Ô		ŏ	î j	ŏ	7	ŏ	Ô	ŏ	3	
Obio:			1					1			
Cincinnati	3 9	;;-	4	69 56	11 25	36 1 6 3	0	7 15	8	15 152	159 223
Cleveland Columbus	4	45 2	2 2 1	3	5	32	Õ	0	Ō	28	101
Toledo Indiana:	3	1	1	111	8	30	0	4	0	89	94
Fort Wayne	5		0	5	0	16	0	0	0	2	21
Indianapolis South Bend	1		2 0	261 1	25 1	12 8	0	3	0	69 1	16
Terre Haute	ě		ŏ	i i	3	ĭ	ě	ō	ě	ô	23
Elinois: Chicago	2	6	4	128	67	287	0	44	0	211	752
Cicero	Ō	ll	0	0	1 1	0	0	1	Ö	0	6 36
Springfield Michigan:	4	2	0	284	5	_	0	1	١٠	- 1	
Detroit	8	6	4 0	36 16	44 10	201 109	0	23	1 0	134	296 31
Flint	ő		i	8	10	36	ŏ	ŏ	ŏ	9	81
Wisconsin: Kenosha		1	0	0	9	21	o	0	0	2	5
Madison	2		1	5		7	Ō		0	32 i	20
Milwaukee Racine	2		0	4	12 1	150	0 12	7	0	132 12	115 18
Superior	ŏ		ŏ	Ô	2	ő	-0	ŏ	ŏ	ĩ	18 6
Minnesota:								l	l	l	
Duluth	ō		0	0	1 8	7 27	0	1 2	0	0 37	21 99 73
Minneapolis St. Paul	5 0		ő	3	10	7	ŏ	4	ŏ	15	73
lowa: Des Moines	0	7		0		23	0		0	0	39
Sioux City	1			15		0	0		Ō	1	
Waterloo Missouri:	0			0		0	0		0	7	-
Kansas City	2		o	10	25	18	o l	7	o l	18	106 51
St. Joseph St. Louis	29		1 1	14 221	8 9	8 22	0 2	3 8	0	75	236
pt. Louis		'l		441	• •	اعم	- 1	0 '	0,	10 1	~

City reports for week ended Mar. 17, 1934—Continued

State and city Cases Cas	all causes
Fargo	
Crand Forks	
South Dakota: Aberdeen	8
Nebraska:	
Comaha	
Topeka	61
Wilmington	19 26
Maryland: 3 7 3 519 27 33 0 17 2 186 Cumberland 0 0 1 1 1 0 0 0 5 Frederick 3 0 8 0 7 0 <td>41</td>	41
Cumberland 0 0 1 1 1 0 0 0 5 Frederick 3 0 8 0 7 0 47 Virginia: 0 1 0 1 0<	071
Frederick	251 16
Washington 8 2 2 606 20 14 0 20 0 47 Urginia: 1 0 1 0 1 0 0 0 5 Norfolk 1 0 198 9 2 0 2 0 10 Richmond 2 3 20 1 7 5 0 5 0 3 Roanoke 3 0 0 2 3 0<	3
Virginia: 1 0 1 0 1 0 0 0 5 Norfolk 11 0 198 9 2 0 2 0 10 Richmond 2 3 201 7 5 0 5 0 3 Roanoke 3 0 0 2 3 0	205
Norfolk	9
Richmond.	39
West Virginia: 0 0 0 1 1 0 0 0 3 Charleston 0 0 0 0 15 0 8 North Carolina: 0 0 0 0 0 0 0 0 0 0 3 0	62 21
Charleston	21
Wheeling 0 0 1 5 24 0 0 8 North Carolina: 0 0 1 5 24 0 0 0 8 Raleigh 0 0 0 16 1 0 0 2 0 8 Wilmington 0 0 0 2 1 1 0 1 0 3 Winston-Salem 0 1 1 40 1 2 0 0 0 0 0 0 South Carolina: 0 37 0 20 1 0 0 1 0 1 0 1	16
North Carolina:	19
Wilmington 0 0 2 1 1 0 3 Winston-Salem 0 1 1 40 1 2 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0	18
Winston-Salem 0 1 1 40 1 2 0 0 0 0 0 South Carolina: Charleston 0 37 0 20 1 0 0 1 0 1	7
Charleston 0 37 0 20 1 0 0 1 0 1	9
Columbia	20
Columbia	13
Georgia:	
Atlanta 3 21 3 255 10 8 0 4 0 1 Brunswick 0 0 51 1 0 0 0 0	100 3
Savannah 1 53 0 65 3 1 0 1 1 6	26
Florida: 0 1 1 41 3 1 0 1 2 3	30
Tampa	34
Kentucky:	
Ashland 0 3 0 0 0 0 1 Lexington 1 7 0 8 2 1 0 2 0 0	17
Louisville 3 0 2 8 29 0 0 0 20	65
Tennessee: 2 2 267 15 9 2 3 0 5	92
Nashville 2 0 75 6 4 0 0 0 26	3 6
Alabama: 1 5 2 72 6 4 0 7 0 0	68
Mobile 1 15 3 0 0 1 0 0	31
Montgomery 0 42 1 0 2 0 -	-
Arkansas:	
Fort Smith 0 31 0 0 0 0 1	i
Louisiana:	150
New Orleans 14 6 0 21 9 21 0 12 0 5 Shreveport 0 0 6 3 1 0 1 0 0	15 6 18
Oklahoma:	
Oklahoma City 6 16 0 6 9 0 0 0 0 0 0 Fexas:	48
Dallas 13 1 1 4 12 7 0 1 1 4	5 6
Dallas 13 1 1 4 12 7 0 1 1 4 Fort Worth 4 2 4 5 3 0 1 3 0 Galveston 0 0 0 2 0 0 1 0 0 Houston 5 0 9 8 4 0 2 0 0	3 5 1 4
	14 74
San Antonio 1 0 18 6 2 0 11 0 0	5 9
Montana:	~
Billings 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0	1
	4
Missoula 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0	8
dano: Boise	7 4 8 2

¹ Nonresident.

City reports for week ended Mar. 17, 1934—Continued

	Diph	.	fluenza	Mea-	Pneu-	Scar-	Small-	Tuber		Whooping	Dog (113)
State and city	theria cases	Case	es Deaths	sles cases	monia deaths	fever cases	pox cases	culosi: death:	former	cases	all causes
Colorado: Denver Pueblo	4 0	41	_ 0	136 0	11 0	10 6	5 0	20	0	85 24	81
New Mexico: Albuquerque Utah:	1		_ 0	3	0	6	0	7	0	1	14
Salt Lake City Nevada: Reno	0		- 9	326 0	3 0	6	0	1	0	46 0	43
Washington: Seattle	0 0	5	- 0	2 39 20	4 7 6 2	28 3 3	0 0 0 5	3 0 2	0 0	88 10 17	90 47 28
Salem California: Los Angeles Sacramento San Francisco	10 0 0	1 17 1 2	0 2 0 2	0 81 2 179	23 0 3	0 52 2 20	0 2 0 0	0 21 0 4	0 0 1 0	67 6 20	302 34 164
State and city	M	ening meni	ococcus ngitis	Polio- mye- litis		State and city				ococcus ngitis	Polio- mye-
•	G	ases	Deaths	Cases			•		Cases	Deaths	litis cases
New York: New York New Jersey: Trenton		1	0	1	Tenn	Maryland: Baltimore Tennessee: Memphis				0	0
Indiana: Indianapolis	- 1	1	o	0	Louis	iana:	eans		1	0	0
Illinois: Chicago Springfield		3	0	0	Texas	s: Dallas	onio		1	0	0
Wisconsin: Milwaukee Iowa:		1	1	0	Utah 8	alt Lak	e City		0	1	0
Des Moines Sioux City North Dakota:		23	0	0	Califo L	иша: os Ang	eles		1	0	5
Fargo		0	1	0				- 1	j		

² Two nonresidents.

Lethargic encephalitis.—Cases: New York, 3; Madison, 1; Washington, 1; Birmingham, 1; New Orleans, 1. Pellagra.—Cases: Philadelphia, 1; Raleigh, 1; Atlanta, 1; Tampa, 1; Mobile, 1; New Orleans, 1. Typhus fever.—Cases: Atlanta, 1; Savannah, 1; Houston, 1.

FOREIGN AND INSULAR

CANADA

Provinces—Communicable diseases—2 weeks ended March 10, 1934.—During the 2 weeks ended March 10, 1934, cases of certain communicable diseases were reported by the Department of Pensions and National Health of Canada, as follows:

Disease	Prince Ed- ward Island	Nova Scotia	New Bruns- wick	Que- bec	Onta- rio	Mani- toba	Sas- katch- ewan	Alber- ta	British Colum- bia	Total
Cerebrospinal meningitis Chicken pox Diphtheria Dysentery		11 2	1 1	1 259 43	376 22	70 7	2 31 4	20 1	90	5 858 80 1
Erysipelas Influenza Measles Mumpe Paratyphoid fever	6	61	1 4	18 13 234	9 28 55 354 2	3 12 396 16	5 4 115 7	3 1	37 34 165	35 155 844 550 3
Pneumonia Poliomyelitis		14			33		7 1		18	72 1 839
Scarlet fever		41	14	171	314	39 1	23 1	16	221	1 25
Tuberculosis Typhoid fever Undulant fever	5	3	15 2	133 49 1	101 12 2	21 5	47 5	5	51 1	381 74 3
Whooping cough		13	3	331	283	25	49	60	27	791

Quebec Province—Communicable diseases—2 weeks ended March 10, 1934.—The Bureau of Health of the Province of Quebec, Canada, reports cases of certain communicable diseases for the 2 weeks ended March 10, 1934, as follows:

Disease	Cases	Disease	Cases
Cerebrospinal meningitis Chicken pox Diphtheria Dysentery Erysipelas German measles Influenza Measles	1 259 43 1 18 30 13 204	Puerperal septicemia Scarlet fever Tuberculosis Typhoid fever Undulant fever Vincent's angina Whooping cough	2 171 133 40 1 2 331

CUBA

Provinces—Notifiable diseases—4 weeks ended November 25, 1933.—During the 4 weeks ended November 25, 1933, cases of certain notifiable diseases were reported in the Provinces of Cuba, as follows:

Disease	Pinar del Rio	Habana	Matan- zas	Santa Clara	Cama- guey	Oriente	Total
Cancer Chicken pox Diphtheria Hookworm disease		1 4	1	7	1	1	3 1 16
Malaria. Measles Tuberculosis. Typhoid fever.	114 1 2 5	66 4 14	352 14 12	1, 904 1 37 62	222 11 20	1, 568 25 17	4, 226 2 93 130

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

(NOTE.—A table giving current information of the world prevalence of quarantinable diseases appeared in the Public Health Reports for Mar. 30, 1934, pp. 438-450. A similar cumulative table will appear in the Public Health Reports to be issued Apr. 27, 1934, and thereafter, at least for the time being, in the issue published on the last Friday of each month.)

Cholera

Philippine Islands.—During the week ended March 24, 1934, cholera was reported in the Philippine Islands as follows: Bohol Province—Inabanga, 1 case, 1 death; Tubigon, 11 cases, 5 deaths. Occidental Negros Province—Escalante, 28 cases, 15 deaths; San Carlos, 6 cases, 4 deaths. Oriental Negros Province—Bais, 2 cases, 2 deaths; Tanjay, 3 deaths.