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Q Fever Studies in Southern California

V. Natural Infection in a Dairy Cow

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In the course of investigations on Q fever in the endemic area of Southern California, it was demonstrated that the causative agent, *Coxiella burneti* (Derrick), was abundantly present in the raw milk from four widely separated dairies in Los Angeles County. These findings have been reported by Huebner *et al.* (*1*).

Not only was the pooled milk from certain groups of cows on these dairies proved to be infectious, but in the study reported and in later tests, a number of individual cows were identified as shedding the organism. Most of these cows whose milk proved to be infectious when injected into guinea pigs gave a strongly positive complement-fixation reaction when their serums were tested with specific Q fever antigens.

Obviously, it was desirable to autopsy an infected cow in order to observe the gross and microscopic pathology, if any, and to see the extent of the infection in the tissues. The first autopsy on such a cow known to be shedding Q fever organisms in its milk was performed January 15, 1948, at the Q Fever Laboratory in Hondo, Calif. This cow bore the herd serial number 8704 and is so designated in our records and tissue samples.

An animal (8704) was selected from a large herd of purebred and grade Holstein and Guernsey dairy cattle which had been under observation since September 1947. There were nine cows in this herd which, on the basis of laboratory tests, were considered for the autopsy. (See

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table 1.) The final choice was made after a careful physical examination of the nine cows and with regard for data given in table 1.

Table 1. *Data on cows considered for autopsy*

Cow No.	Serology Nov. 19 1947	Serology Dec. 7 1947	Milk Nov. 19 1947
4273	+1-64	+1-64	+
4330	+1-32	+1-64	+
6832	+1-32	+1-128	+
7398	+1-32	----------	+
7427	+1-16	–	+
8683	+1-32	+1-8	+
8704	[1] +1-32	----------	+
8749	+1-32	+1-16	+
9364	+1-16	–	+

[1] Serum from this cow, taken at time of autopsy (Jan. 15, 1948) was positive at 1:32.

We wish to emphasize that cow 8704 was not unique in the herd insofar as pre-autopsy serology and other information were concerned. Probably several others of the nine cows would have been suitable for autopsy. A lactating and pregnant animal was preferred.

Laboratory Findings Prior to Autopsy

C. burneti was isolated from the pooled milk of string No. 34, which included cow 8704, when tested October 28, 1947. Milk from individual cows was tested November 19, and cow 8704 was one of those shown to be positive. The complement-fixation test on this animal was reported as positive for Q fever at a titer of 1:32 by the National Institutes of Health on a sample taken November 19, 1947. The sample taken at time of autopsy was again positive at a titer of 1:32.

History and Physical Findings

Cow 8704 was a grade Guernsey born May 17, 1942, in the San Fernando Valley, Los Angeles County, Calif. She had given birth to two calves, the last on May 7, 1947. She was bred July 27, 1947. The animal had been hand-milked during the present lactation and was lactating up to the time of autopsy.

A physical examination on January 14 showed the following: Unthrifty appearance in distinct contrast to most of the others in the herd; pulse 68; respiration 42; temperature 101.4; pregnancy; pleuritic friction sounds on left side; distinct fibrosis (Udall) in all quarters of the udder; numerous firm nodular cutaneous lesions on the udder, with a minimum amount of external discharge; pocklike skin lesions on udder and teats; supramammary lymph nodes palpable and enlarged; some redness and edema in the escutcheon area. No ticks were found on this cow although many animals in this herd, especially young stock in the corrals, were infected with spinose ear ticks, *Otobius megnini.*

Autopsy and Sampling of Tissues

The udder was washed and teats cleaned with cotton swabs soaked in 70 percent alcohol before separate milk samples were taken from each quarter. The animal was then restrained and anesthetized with an intravenous injection of chloral hydrate 36 grams and magnesium sulfate 30 grams in 250 cubic centimeters of water. Only light anesthesia resulted. The carotid artery was exposed and canulated and the cow was bled. Expiration was slow so the brain was pithed. The autopsy began at 2 p. m. and the final tissue samples for histological study and infectivity studies were taken and stored at 12 midnight.

A median incision was made from the mentis to the pubis, the skin laid back, and the legs on the right side reflected. The peritoneal and pleural cavities were opened and exposed. The tongue, larynx, trachea, lungs, and heart were removed together. Organs or systems were placed on large sterile trays and removed to another laboratory building where tissue samples were saved for animal injection or in fixatives for histological study. One set of autopsy instruments was used throughout in the gross autopsy without resterilization. Tissue samples were taken from each organ with a separate set of freshly boiled instruments to prevent cross infection. The udder and attached supramammary lymph nodes were the last organs to be removed from the carcass and sampled.

Gross Observations

Upper alimentary and respiratory tracts—The larynx showed a few petechiae and similar lesions were present at the level of the first three tracheal rings. The retropharyngeal lymph nodes were edematous. The mucosa of the mouth, nose, sinuses, trachea, and oesophagus were normal as were the salivary glands and the pharyngeal lymph glands.

Lungs—The lungs showed fibrinous deposits in the left pleural space and slight congestion of right upper lobe and left diaphragmatic lobe. The mediastinal lymph nodes were normal.

Liver—The liver was loosely adherent to the diaphragm at two points on the left side. In this area a scar was present in the liver parenchyma. The gall bladder appeared normal and its contents were aspirated aseptically for testing.

Spleen—The spleen was firm and small (animal bled before autopsy).

Pancreas—The pancreas appeared normal.

Kidneys—A small cortical area of the kidney showed petechiae. This may have resulted from ante-mortem rectal examination.

Ureters—The ureters appeared normal.

Adrenals—The adrenals showed slight cortical hemorrhage.

Heart—The pericardial fluid was present in slightly more-than-average quantity. The anterior portion of the left ventricle showed considerable subendocardial hemorrhage (possibly agonal). The other parts of the heart appeared normal.

Muscle—The muscles were normal. A section was taken from the adductor of the hind leg for testing.

Skin—From the base of a teat, a portion of skin containing nodular and pock-like lesions was taken for testing. Otherwise the skin was normal.

Gastrointestinal tract—The mesenteric vessels were congested and a fibrous piece of material was found free in the peritoneal cavity. The rectum showed some areas that were slightly hemorrhagic and these areas were attributed to rectal examination prior to autopsy. The gastric mucosa, rumen, reticulum, omasum, abomasum, and mesenteric lymph nodes appeared normal.

Genitourinary tract—A cystic follicle was present in the left ovary and the corpus luteum was present in the right ovary. A normal 5-month old fetus was present. The amniotic fluid was aspirated aseptically for testing. Fetal blood was collected for the complement fixation test and fetal tissues were saved for testing. The bladder was empty but normal and was flushed with sterile saline for testing. The uterus, vagina, and fetal membranes were normal.

Udder—Specimens were taken from each quarter in the more dorsal area and ventrally near the gland cistern. Right hind quarter: Normal parenchyma was present in dorsal section and no sharp line of demarcation between fat and parenchyma was observed. The gland cistern appeared fibrosed. Right front quarter: Dorsal section appeared normal, area of replacement sharply defined, ventral section normal. Gland cistern showed less fibrosis than in right hind quarter. Left hind quarter: Dorsal and ventral sections normal; gland cistern with very slight fibrosis. Left front quarter: Normal; gland cistern with very slight fibrosis.

The supramammary lymph nodes were enlarged about double in size and were edematous. Peripheral hemorrhage was observed in the right node and some generalized hemorrhage was present in left node. A third node was less conspicuously affected and possibly not enlarged.

Brain—The brain was small in size and areas of pigmentation were observed in the meninges. Sections of hippocampus and cerebral cortex between first and second sulci were taken. Sections of cerebellum, medulla, and pituitary were also taken.

The gross pathology observed was nonspecific in character and essentially consisted of congestion in the lungs with apparent fibrinous

pleuritic deposits, swelling in the supramammary lymph nodes, and localized fibrosis in the mammary gland cistern.

Tissue samples for histological study were taken from nearly all the organs that were tested for infection. A series was preserved in each of three fixatives: Bouin's, 10 percent formalin, and Regaud's. These were sent to the Division of Pathology, National Institutes of Health. A detailed report on these tissues may be published later by Dr. T. L. Perrin, Jr., of that Division, who is making other studies on Q fever in bovines. We are indebted to Dr. Perrin for the following summary on the histopathology in this animal.

> "Diagnosis: Subacute and chronic focal mastitis.
> Slight focal cellular infiltration of kidney, lung and pituitary.
> Parasite infestation (*Sarcocystis*) of cardiac and skeletal musculature.
> Subacute inflammation of nasopharyngeal mucosa.
> Subacute focal inflammation of skin (udder) with suppuration.
> Hemosiderosis of spleen.
> Micro-organisms were not identified after careful search in appropriately stained sections."

To the best of our knowledge this was the first naturally infected cow to be autopsied. Therefore, no definite statement can be made that any of the gross or microscopic pathology observed was specifically the result of Q fever infection.

Test of Tissues for Infectivity

The tissue samples to be tested for infectivity were stored immediately at below freezing temperature in glass jars. On January 19, guinea pigs were injected with samples of milk and with suspensions of udder tissue from each quarter. One animal was injected intraperitoneally and one subcutaneously with each inoculum. In some of those injected by the latter route characteristic subcutaneous indurated lesions became evident after 10 days; suggesting positive results. The other tissues were then removed from storage, tissue suspensions prepared and injected into guinea pigs. A duplicate set of tissues was shipped to the Rocky Mountain Laboratory under dry ice refrigeration. They were received January 27, 1948, and all specimens were partially or completely frozen and well preserved. These tissues were tested immediately in guinea pigs.

Animals at the Q Fever Laboratory injected with milk, udder tissue, and supramammary lymph-node tissue were bled on the twentieth day and the serums tested for complement fixation, whereas animals injected with other tissues were bled on the thirty-fifth day after injection.

All test animals at the Rocky Mountain Laboratory were bled on the twenty-ninth, thirtieth, or thirty-first day.

The tissues which were tested and the results of these tests at both laboratories are listed in table 2.

Table 2. *Results of infectivity tests on tissues*

	Rocky Mountain Laboratory (2 guinea pigs)	Q Fever Laboratory (2 guinea pigs)
NERVOUS SYSTEM:		
1. Brain	− −	− −
RESPIRATORY SYSTEM:		
2. Cartilage from turbinates and mucosa of septum	− −	− O
3. Lung	− −	− +
4. Congested area of lung	− −	− O
DIGESTIVE SYSTEM:		
5. Salivary gland	− −	− −
6. Rumen	− −	− −
7. Ileum, stomach duodenum	− −	− −
8. Liver	− −	− O
9. Pancreas	− −	− −
10. Gall bladder	− −	− O
11. Bile	− O	O O
UROGENITAL SYSTEM:		
12. Kidney	− −	− −
13. Bladder	− −	− O
14. Saline washing of empty urinary bladder	− −	
15. Ovary	− −	− O
16. Uterus	− −	− O
17. Vagina	− −	− −
LYMPHATIC SYSTEM:		
18. Spleen	− −	− −
19. Retropharyngeal node	− −	− −
20. Mediastinal node	− −	− O
21. Hepatic nodes	− O	− −
22. Mesenteric nodes	− −	− −
OTHER TISSUES:		
23. Heart	− −	− −
24. Muscle	− −	− −
25. Adrenal	− −	
26. Skin	− −	− −
27. Citrated blood		− −
EMBRYO TISSUES:		
28. Blood	− −	− −
29. Spleen	− O	− −
30. Kidney	− −	
31. Amnion	− −	
32. Amniotic fluid	− −	− −
MILK BY QUARTER OF UDDER:[1]		
33. Right front	+ +	+ −
34. Left front	+ +	+ +
35. Right rear	+ +	+ +
36. Left rear		+ +
MAMMARY TISSUE BY LEVEL AND QUARTER OF UDDER:[1]	4 guinea pigs	
Right front upper	+ + + +	+ +
Right front lower	+ − + +	
Left front upper	+ + + +	+ +
Left front lower	+ + + +	
Right rear upper	+ + + +	+ O
Right rear lower	+ + + +	
Left rear upper	+ + + +	+ +
Left rear lower	+ + + +	
	2 guinea pigs	
SUPRAMAMMARY LYMPH NODES	+ +	+ +

+=Positive results. −=Negative results. O=Test not completed or animal dead.

[1] Milk samples from rear quarters pooled at Rocky Mountain Laboratory. Mammary-tissue samples from upper and lower levels of each quarter pooled at Q Fever Laboratory.

Positive results were obtained in 13 of the 14 animals injected with milk from individual quarters. Positive results were obtained from all quarters. Forty animals were injected with udder-tissue suspensions of individual quarters and of these, 35 gave positive results. Again the positives represented all quarters of the udder. One animal was negative and four died before the test period was completed. Serums of 11 of these guinea pigs which had been injected with udder tissue at the Rocky Mountain Laboratory were tested for full end-titer of antibodies against two Q fever antigens (the Henzerling-Italian strain and a Nine-Mile strain). The titers as determined are given in table 3. These serums were taken on the twenty-ninth or thirtieth day after injection.

Table 3. *Q fever titers in guinea pigs injected with mammary tissue suspensions*

Guinea pig No.	Henzerling antigen	Nine-Mile antigen
C5680	1:4096	1:4096
C5686	1:2048	1:4096
C5688	1:2048	1:1024
C5694	1:1024	1:2048
C5710	1:8192	1:8192
C5713	1:512	1:256
C5715	1:2048	1:512
C5718	1:4096	1:4096
C5721	1:4096	1:2048
C5724	1:2048	1:512
C5725	1:4096	1:2048

All four animals injected with suspensions of the supramammary lymph nodes were positive.

One of the animals that had been injected with a suspension of lung tissue taken from a normal-appearing area of the lung was bled on the thirty-fifth day after injection and was positive by complement fixation. It was re-bled on the fortieth day and was again positive. This was the only test animal, with the exception of those injected with milk, udder, or supramammary lymph nodes, to give positive results. For this reason, other samples of lung tissue which had been stored below freezing temperatures were tested. Four guinea pigs were injected with a suspension of lung from a normal-appearing area, and four were injected with a suspension of lung from an obviously congested area. All eight guinea pigs were negative when tested on the thirty-third day for complement-fixing antibodies.

There were 25 other specimens of fluids or tissues from the cow and 5 from the embryo tested at one or both laboratories. At least one, usually three or all four, of the animals injected with each tissue survived the test period and were bled. Bile was toxic and killed

three of four test animals. All surviving animals were negative on complement-fixation test, indicating lack of infection in all organs of the cow except the udder and its associated lymph nodes. The only exception was the one positive from lung tissue which on retest of the tissue, was not substantiated.

Controls

Thirty-one normal guinea pigs were distributed among the cages of the test animals at the Q Fever Laboratory at the time of their injection. These controls were kept in the same cans with the test animals for the duration of the experiment. There were three deaths in control animals from undetermined causes. The remaining 28 control guinea pigs were tested for complement-fixing antibodies at the conclusion of the experiment and all were negative.

Summary

A mature dairy cow known to be shedding Q fever rickettsiae in its milk and whose serum gave a positive complement-fixation test with Q fever antigen was autopsied. Both on the basis of serology, and discharge of the infectious agent in milk, infection had been present over a period of at least 2 months. The few lesions observed were nonspecific and could not be attributed definitely to Q fever, since the pathology of acute or chronic *Coxiella burneti* infection in cattle is practically unknown.

Tests of milk and numerous tissues for infectivity by maceration and injection into guinea pigs demonstrated *C. burneti* in the milk and udder tissues of all four quarters and in the supramammary lymph nodes proximal to the udder.

One of eight experimental animals injected with lung tissue developed antibodies to Q fever but on a repeat test of two samples of lung tissue, all eight test animals remained negative.

No infection was demonstrated in the other tissues or fluids from this cow or from blood, spleen, kidney, amnion or amniotic fluid from its 5-month old fetus.

REFERENCE

(1) Huebner, R. J., Jellison, W. L., Beck, M. D., Parker, R. R., and Shepard, C. C. Q fever studies in Southern California. I. Recovery of *Rickettsia burneti* from raw milk. Pub. Health Rep. **63**:214–222 (1948)

Q FEVER STUDIES IN SOUTHERN CALIFORNIA SERIES

This article is the third in a series of five on Q fever studies in California, appearing in the PUBLIC HEALTH REPORTS. Already published in Vol. 63, are: I. Recovery of *Rickettsia burneti* from raw milk, pp. 214–222; IV. The occurrence of *Coxiella burneti* in the spinose ear tick, *Otobius megnini*, pp. 1483–1489. To be published are: II. An epidemiological study of 300 cases; III. Effects of pasteurization on the survival of *Coxiella burneti* in naturally infected milk.

Carbon Tetrachloride Poisoning

A Report on Ten Cases at the U. S. Marine Hospital, Seattle, Washington, Since 1937

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Ten patients have been admitted to the U. S. Marine Hospital, Seattle, Washington, since 1937, with carbon tetrachloride poisoning; of these, four patients died. Most of the cases could have been avoided by the precaution of proper ventilation.

Carbon tetrachloride is one of the most widely used of the organic solvents. It is employed as a dry cleaning fluid, a metal degreaser, rubber solvent, fire extinguishing agent, household cleaner, and is used for a variety of other industrial and nonindustrial applications (*1*). That Carbon tetrachloride is toxic and occasionally fatal has been realized since 1909 (*2*). Numerous cases have been reported in the literature, but the reported cases probably are only a small fraction of the actual number of cases of poisoning. In most instances, fatal poisoning has occurred in isolated or individual use of carbon tetrachloride as contrasted to its use in industry (*3*).

The present allowed standard maximum is 100 parts of carbon tetrachloride per 1,000,000 of inspired air. Toxic reactions to carbon tetrachloride may result from a single brief exposure to a high concentration of the vapor, from prolonged or repeated exposure to a moderately high concentration or from regular daily exposure to lower concentrations; also from repeated contact with the skin or from ingestion. Obese persons, alcoholics, or undernourished individuals are likely to be especially susceptible to carbon tetrachloride injury, as are those with diabetes, liver or kidney disease, jaundice, pulmonary or heart disease, or peptic ulcers (*1*).

The signs and symptoms of carbon tetrachloride poisoning differ somewhat according to the nature of exposure (*1*).

Acute intoxication resulting from a single exposure to a high concentration results in mental confusion, feeling of fullness in the head, headache, dizziness, nausea, stupor, or loss of consciousness. Death from respiratory failure may occur. There may be a delayed appearance of systemic poisoning with liver and kidney symptoms.

Repeated or prolonged inhalation may cause headache, fatigue, nausea, vomiting, dizziness, visual disturbances, and bleeding from the mucous membranes followed by severe acute nephritis and toxic hepatitis. It may be injurious to the central nervous system.

Some cases are caused from ingestion of carbon tetrachloride and result in nausea, vomiting, abdominal distress, diarrhea, bloody stools, and coma followed by hepatitis, jaundice and nephritis.

The general clinical picture is headache, nausea, vomiting, hematemesis, hematuria, icterus, oliguria, and retention of urine. Pulmonary complications may occur as late as a week after exposure. Although experimental proof is lacking, it is felt that carbon tetrachloride is removed from the body by the expired air as well as by the kidneys. Some may be detoxified by the liver.

It is stated that anuria and oliguria are often the primary physiological disturbances in man and the development of pulmonary edema is often the immediate cause of death (*3*).

The cases presented here are brief and only the significant findings are given.

CASE No. 1. A 38-year-old veteran, a cleaner and dyer by occupation, was admitted to the hospital November 3, 1937, complaining of nausea and vomiting, abdominal cramps, anuria, blindness, convulsions, and mental confusion. For 2 weeks prior to admission, the patient had been exposed to large vats of carbon tetrachloride used in his trade. The first symptoms were headache and dizziness. The patient had a history of being a heavy drinker. Upon admission to the hospital he was acutely ill with convulsions, blindness, and anuria. On admission the blood urea nitrogen was 80 mg. percent; the following day it was 200 mg. percent. The patient died 3 days after admission. The autopsy revealed parenchymatous nephritis, fatty degeneration of the liver, and pulmonary edema.

CASE No. 2. A 29-year old white male was admitted March 2, 1945, with the chief complaints of nausea, vomiting, oliguria, hematuria, edema of the face and hands, pain over the liver area, and clay-colored stools. The patient had a history of being a mild alcoholic. He stated he was in a cellar where a friend was cleaning clothes with carbon tetrachloride. He was drinking at that time, and after about an hour began to feel dizzy and have a headache. The following day he began to have oliguria, hematuria, and edema of the face and hands which progressed. The patient was a refrigerator worker by trade. He was seriously ill upon admission. Blood urea nitrogen was 150 mg. percent. He died March 7, 1945. The autopsy revealed an acute, diffuse, parenchymatous nephritis, toxic hepatitis, pulmonary edema, hydrothorax, and ascites.

CASE No. 3. A 29-year old merchant seaman was admitted February 22, 1946, complaining of hiccups, multiple bleeding, oliguria, headaches, dizziness, nausea and vomiting. He stated that for 2 or 3 years prior to admission he had been cleaning a mimeograph machine with carbon tetrachloride three times a day in a poorly ventilated compartment aboard ship. He was a steward by trade and was a

heavy drinker. His blood urea nitrogen was 140 mg. percent. He died 3 days after admission. The autopsy revealed a diffuse, toxic nephritis, pulmonary edema, multiple hemmorrhages, and a large fibrotic liver.

CASE No. 4. A 52-year old merchant seaman, an electrician by trade, was admitted to the hospital May 15, 1946. Three days prior to admission he had cleaned one of the ship's generators using a spray gun filled with carbon tetrachloride. He estimated that he used about 10 gallons. The engine room was poorly ventilated. The next evening he developed a headache, dizziness, weakness, nausea, and vomiting. There was a history of alcoholism. Upon admission he was jaundiced. His liver was enlarged and tender. He had edema of the extremities. The urine contained albumin, casts, and red blood cells. There was oliguria with an output of about 50 cubic centimeters of urine daily. He gradually became compensated and left the hospital. The diagnosis was nephritis and hepatitis due to carbon tetrachloride poisoning.

CASE No. 5. A 29-year old colored merchant seaman, fireman by trade, was admitted August 17, 1946. He had the history of being a heavy drinker. He stated that he had been continuously exposed to carbon tetrachloride aboard ship. He complained of headaches, abdominal pains, jaundice, nausea and vomiting. Upon admission to the hospital he was jaundiced. The blood urea nitrogen was 140 mg. percent. He died 4 days after admission. The autopsy revealed an acute, diffuse, parenchymatous nephritis, acute hepatitis, pulmonary edema and acute hemorrhagic gastritis.

CASE No. 6. A 27-year-old white merchant seaman, radar operator, was admitted May 15, 1947, with a history of moderate drinking. His chief complaints were subsiding jaundice, weakness, nausea, vomiting, and headaches. He gave a history of a few days prior to the onset of the jaundice, along with two friends, sniffing carbon tetrachloride for the purpose of "getting a jag on." The cephalin flocculation test was 4+. He had the signs and symptoms of subsiding hepatitis. He slowly improved with apparent full recovery.

CASE No. 7. A 30-year-old merchant seaman was admitted on June 21, 1947, complaining of nausea, vomiting, malaise, and headaches. He gave the history of having become mildly intoxicated on June 18, 1947. The following day he spent 2 hours cleaning clothes with carbon tetrachloride in a poorly ventilated room. He had a history of moderate drinking. The urine contained albumin, casts, and red blood cells. The blood urea nitrogen was 95 mg. percent. The blood creatinine was 7.2 mg. percent. The diagnosis was acute nephritis due to carbon tetrachloride poisoning. The patient recovered and was discharged on July 17, 1947.

CASE No. 8. A 37-year-old merchant seaman was admitted on October 23, 1947, complaining of blurring of vision, memory loss, nervousness, weakness, malaise, and jaundice. He had the history of being a heavy drinker. He stated that he had been daily exposed to carbon tetrachloride for the past 2 years aboard ship. The icteric index was 42 on admission. The liver was enlarged and there was albumin in the urine. A diagnosis of toxic hepatitis was made. The patient slowly recovered.

CASE No. 9. A 44-year-old merchant seaman was admitted October 13, 1947, complaining of weakness, dizziness, and pains in the right upper quadrant. There was a history of alcoholism. The patient

Signs and Symptoms	Occupation and Place of Exposure	Important Findings
1. Nausea and vomiting, abdominal cramps, anuria, blindness, convulsions and mental confusion.	Cleaner and dyer. Age 38. Exposed to large vats of carbon tetrachloride. Heavy drinker.	Anuria. B. U. N. up to 200 mg. %. Autopsy: Parenchymatous nephritis, fatty degeneration of liver and pulmonary edema.
2. Nausea, vomiting, oliguria, hematuria, edema of the face and hands, pain over the liver and clay-colored stools.	Refrigerator mechanic. Age 29. Cleaning clothes with carbon tetrachloride in a cellar room. Mild alcoholic.	Oliguria, hematuria, edema of the face and hands. B. U. N. 150 mg. %. Autopsy: Acute, diffuse, parenchymatous nephritis, toxic hepatitis, pulmonary edema, hydro thorax and ascites.
3. Hiccups, hemorrhages, oliguria, headaches, dizziness, nausea and vomiting.	Merchant seaman, steward. Age 29. For 3 years had been cleaning a mimeograph machine with carbon tetrachloride 3 times a day in a poorly ventilated compartment aboard ship. Heavy drinker.	B. U. N. 140 mg. %. Expired 3 days after admission. Autopsy: Diffuse, toxic nephritis, pulmonary edema, multiple hemorrhages and a large fibrotic liver.
4. Headache, dizziness, weakness, nausea and vomiting.	Merchant seaman, electrician. Age 52. Three days prior to admission had cleaned ship's generator with spray gun filled with carbon tetrachloride. Room poorly ventilated. History of alcoholism.	Jaundiced, liver large and tender, edema of extremities. Albumin, casts and red blood cells in urine. Oliguria. Became compensated and left hospital. Diagnosis: Nephritis and hepatitis.
5. Headaches, abdominal pains, jaundice, nausea and vomiting.	Merchant seaman, fireman. Age 29. Colored. Continuously exposed to carbon tetrachloride aboard ship. Heavy drinker.	Jaundice. B. U. N. 140 mg. %. Liver enlarged and tender. Expired 4 days after admission. Autopsy: Acute, diffuse, parenchymatous nephritis, acute hepatitis, pulmonary edema, acute hemorrhagic gastritis.
6. Jaundice, weakness, nausea, vomiting, and headaches.	Merchant seaman, radar operator. Age 27. Few days prior to onset of jaundice sniffed carbon tetrachloride for purpose of "getting a jag on". Moderate drinker.	Function test revealed impairment of liver. Jaundiced. Diagnosis: Hepatitis. Discharged apparently recovered.
7. Nausea, vomiting, malaise and headaches.	Merchant seaman. Age 30. Two hours cleaning clothes with carbon tetrachloride in poorly ventilated room two days prior to admission. Moderate drinker.	Albumin, casts and red blood cells in urine. B. U. N. 95 mg. %. Blood creatinine 7.2 mg. %. Diagnosis: Acute nephritis due to carbon tetrachloride poisoning. Recovered.
8. Blurring of vision, memory loss, nervousness, weakness, malaise and jaundice.	Merchant seaman, Second Officer. Age 37. Daily exposure to carbon tetrachloride for past 2 years aboard ship. Heavy drinker.	Icteric index 42, enlarged liver, albumin in urine. Diagnosis: Toxic hepatitis. Recovered.
9. Weakness, dizziness, pains in right upper quadrant.	Merchant seaman, oiler. Age 44. Worked with carbon tetrachloride for 2 weeks aboard ship. Moderate drinker.	Liver function test revealed mild liver damage. Diagnosis: Hepatitis. Recovered.
10. Nausea, vomiting and abdominal pains, diarrhea, oliguria.	Merchant seaman, electrician. Age 34. Daily exposure to carbon tetrachloride vapors in poorly ventilated room aboard ship. Moderate drinker.	Albumin and casts in urine. B. U. N. 95 mg. %. Oliguria. Diagnosis: Toxic nephritis. Recovered.

gave a history of working with carbon tetrachloride for 2 weeks aboard ship. About 10 days after this, he became ill with the above complaints. A fellow worker became jaundiced. The patient was hospitalized in Alaska and was seen here several weeks after exposure. The only finding indicative of liver damage was a 3+ cephalin flocculation test. A diagnosis of probable carbon tetrachloride poisoning with slow recovery was made.

CASE NO. 10. A 34-year-old white merchant seaman, electrician, was admitted January 26, 1948, complaining of nausea, vomiting, abdominal pains, diarrhea, and oliguria. He had the history of being a moderate drinker. He gave a history of daily exposure to carbon tetrachloride vapors in a poorly ventilated room aboard ship. The urine contained 3+ albumin and casts. The blood urea nitrogen was 62 mg. percent and progressed up to 95 mg. percent. The cephalin flocculation test was negative. The patient had oliguria for several days but gradually returned to normal. The impression was that of toxic nephritis.

In addition to the above cases, the authors are personally aware of several cases of carbon tetrachloride poisoning which were never admitted to the hospital. Also, their experience in other hospitals is not included.

Treatment

A few words of caution are: never give alcohol, fats, oils, or epinephrine to a suspect of carbon tetrachloride poisoning. Intravenous hypertonic glucose and Hartmann's solution should be given to combat acidosis and liver dysfunction. The patient should be watched closely for pulmonary edema. Methionine and choline may be given and oxygen and blood as indicated. Papaverine may be given to combat vasospasm. When the patient is able to tolerate food orally, he should be given a high carbohydrate and protein, low fat diet along with choline, vitamin B and vitamin K.

Summary

Alcohol seemed to be a predisposing factor in eight of the ten cases. Seven of the cases presented severe kidney damage. Anuria and pulmonary edema were the clinical phenomena most serious and were the chief causes of death in the four fatal cases. In most of these cases the users of the carbon tetrachloride were not aware of its danger, and apparently the operators of the ships or industries were not aware of the danger involved in its use. In all of these cases improper ventilation was a factor.

It is felt that the incidence of carbon tetrachloride poisoning is increasing, either due to the fact that it is being more widely used and less precautions are being taken, or else the diagnosis is being made more frequently.

It is not the purpose of this article to emphasize the diagnosis and treatment, but to call attention to the frequent and preventable occurrence of carbon tetrachloride poisoning. It is felt that ship operators and small business concerns are improperly educated as to the dangers of carbon tetrachloride. Carbon tetrachloride should be properly labeled with instructions explaining the dangers in its use. It should always be used in a properly ventilated room. It is believed that investigation will show that these precautions are not being observed.

REFERENCES

(*1*) Safety Research Institute: Recognition and treatment of carbon tetrachloride poisoning. Council on Industrial Health, J. A. M. A. 132:786 (1946).

(*2*) Hamilton, Alice: Industrial Toxicology. 1934, Harper and Brothers, New York, 329 pp.

(*3*) Martin, William B., Dyke, Louis H., Coddington, Fred L. and Snell, Albert M.: Carbon tetrachloride poisoning; A report of one case with necropsy and one nonfatal case with clinical laboratory studies, Ann. Int. Med. 488 (1946).

The Serological Characterization of North Queensland Tick Typhus[1]

By DAVID LACKMAN, *Senior Assistant Scientist*, and R. R. PARKER, *Director*

Serological Analysis by the Rabbit-Injection Method of Felix

Felix (*1*) first suggested in 1933 the use of rabbits as test animals in studying the antigenic structure of rickettsiae. Davis (*2*) in 1935 used rabbits to determine antigenic relationships among rickettsiae. These studies were based on the observation that when rabbits are injected with most strains of rickettsiae they respond with the production of Weil–Felix agglutinins. When the rabbits are given a second injection about 30 days following the original injection, there is further production of Weil–Felix agglutinins, providing the second injection is made with a strain heterologous to the original strain; if the strain of rickettsia used in the "challenge" injection possesses the same antigenic structure as the original strain, there is no further increase in agglutinins.

In an attempt to apply this procedure to an analysis of the antigenic structure of North Queensland tick typhus, 30 rabbits were injected, intraperitoneally, with 2.0 cubic centimeters of tunica suspension from guinea pigs infected with the "Phillips" strain.[2] The rabbits were

[1] From the Rocky Mountain Laboratory, Hamilton, Mont., Division of Infectious Diseases, National Institutes of Health.

[2] The strain of North Queensland tick typhus used was originally sent from Australia by Dr. F. M. Burnet to Dr. J. Smadel at the Army Medical School. It was obtained from Dr. Smadel by Dr. C. B. Philip of this laboratory. It has been carried by serial passage in guinea pigs since the date of its receipt (Dec. 29, 1945).

bled 14, 16, and 27 days following inoculation and the serums were tested in the Weil-Felix test for agglutinins to Proteus OX19, Proteus OX2, Proteus OXK, and Proteus XLL. They were also tested by complement fixation against antigens of epidemic typhus, murine typhus, Rocky Mountain spotted fever, the 9–Mile strain of American Q fever, boutonneuse fever, and North Queensland tick typhus.

Twenty-seven days following the initial injection, the rabbits were divided into groups and challenged with Rocky Mountain spotted fever, boutonneuse fever, and epidemic typhus. They were bled 14, 16, 20, and 30 days following challenge and the sera were subjected to the same tests as used on the serums following the initial inoculation. Five normal rabbits were injected with each of the challenge strains to serve as controls.

Results

Twenty-four of the rabbits survived the inoculations and bleedings. The results of the complement-fixation test on the serums from the rabbits following the initial injection with North Queensland tick typhus are summarized in table 1.

Table 1. *Summary of results of complement fixation with sera from rabbits injected with North Queensland tick typhus*

Antigen Used					
Q fever	Epidemic typhus	Murine typhus	Rocky Mountain spotted fever	Boutonneuse fever	North Queensland tick typhus
*0/24	0/24	0/24	22/24	12/24	13/24

*Numerator=number of "positive" rabbits (those showing an increase in complement-fixing activity over that present in the preinoculation specimens).
Denominator=total number of rabbits in group.

No significant agglutination was obtained in the Weil-Felix test with any of these sera.

The results of the serological tests on the serums from the rabbits following challenge are summarized in table 2.

Discussion

Following the initial injection with North Queensland tick typhus the sera from 22 out of 24 rabbits gave significant complement fixation with Rocky Mountain spotted fever antigen. The sera from 12 out of 24 and 13 out of 24 gave significant reactions with boutonneuse fever and North Queensland tick typhus, respectively. No significant reaction was obtained with antigens of Q fever, epidemic typhus, and murine typhus. These results indicate that North Queensland tick typhus belongs in the spotted fever group of rickettsiae. This is in

Table 2. *Results of complement-fixation and Weil Felix agglutination tests with sera from rabbits injected with North Queensland tick typhus and challenged with epidemic typhus, Rocky Mountain spotted fever, and boutonneuse fever*

Rickettsia used for challenge					
Epidemic typhus		Rocky Mountain spotted fever		Boutonneuse fever	
[1] CF	[2] WF	CF	WF	CF	WF
[3] 10/10	10/10	4/4	3/4	5/5	0/5
[4] 9/10	7/10	4/5	4/5	5/5	0/5

[1] Complement-fixation test with homologous antigen.
[2] Weil-Felix agglutination performed with the four strains of proteus previously mentioned but results based on agglutination obtained with Proteus OX19, Proteus OX2, and Proteus XLL.
[3] Numerator=number of positive animals. Denominator=total number of animals in group.
[4] The figures in this row are for the control rabbits. These animals did not receive an initial injection of North Queensland tick typhus.

disagreement with the serological findings of Plotz and co-workers (*3*) with this rickettsia. They failed to obtain any cross reactions in complement-fixation tests with convalescent guinea pig serum. However, our results in rabbits do not enable one to make a differentiation within this group. The failure of the initial inoculation of North Queensland tick typhus to produce Weil–Felix agglutinins in rabbits resembles the findings of Davis (*2*) with boutonneuse fever.

Following challenge, the majority of the rabbits developed complement-fixing antibodies against the strain injected. In the case of the rabbits challenged with Rocky Mountain spotted fever and epidemic typhus, Weil–Felix agglutinins were produced. The rabbits challenged with boutonneuse fever failed to show Weil–Felix agglutinins in conformity with the results of Davis (*2*).

These results fail to give any indication as to the identity of North Queensland tick typhus within the "spotted fever" group because of its failure to produce Weil–Felix agglutinins following inoculation into rabbits.

When the rabbit sera were tested with heterologous antigens, there was considerable cross-reactivity in the complement-fixation test. This characteristic of rabbit sera has been noted by other investigators, particularly Davis (*4*). He points out that a number of investigators have called attention to the nonspecific fixation of complement with lipoidal and bacterial antigens when working with normal rabbit, dog, and mule sera, and that the injection of concomitant antigens probably stimulates the property of normal rabbit serum to fix complement with lipoidal antigens. On the basis of the results obtained in this study and the experience of others, we would not recommend the use of rabbits in studies of rickettsiae where it was desirous to use the complement-fixation reaction to study the antibody content of the sera.

Serological Analysis by Determining the Complement-Fixing Antibodies Present in Convalescent Guinea Pig Sera

Complement-fixation results employing as antibody sera obtained from guinea pigs convalescent from various rickettsial diseases,[3] and using as antigen rickettsial suspensions and soluble antigen prepared from infected yolk sacs according to the method of Topping and Shepard (*5*), are given in table 3.

Table 3. *Average titer obtained in complement-fixation tests* [1] *between North Queensland tick typhus antigen and convalescent guinea pig sera*

Guinea pig serum against—	Antigen		Titer of antiserum with its homologous rickettsial suspension
	North Queensland tick typhus		
	Soluble antigen	Rickettsial suspension	
North Queensland tick typhus	1:248	1:126	
South African tick-bite fever	1:56	1:3	1:64
Boutonneuse fever	1:43	1:10	1:336
Rocky Mountain spotted fever	1:103	1:18	1:240
Rickettsialpox	1:200	1:15	1:166
Maculatum disease	1:64	0	1:328
Murine typhus	0	0	1:512
Q fever (9-Mile strain)	0	0	1:416

[1] Average of 20 tests.

Discussion

The results obtained with soluble antigen indicate that this strain of rickettsia belongs to the "spotted fever" group. The higher titer obtained in the reaction between North Queensland tick typhus rickettsial suspension and the homologous antiserum indicates that it represents a new strain within this group. The final column of figures in table 3 gives the reactions of the antisera with their homologous antigens.

A study, similar in nature to the one reported above, was carried out by Plotz and co-workers (*6*) in differentiating boutonneuse and Rocky Mountain spotted fevers on the basis of the comparative complement-fixing activity of soluble antigens and rickettsial suspensions with convalescent guinea pig sera.

The previous knowledge concerning North Queensland tick typhus is presented in an editorial in the Bulletin of the United States Army Medical Department (*7*).

The results of cross-immunity tests in guinea pigs have been reported by Plotz and co-workers (*3*). They showed that guinea pigs, convalescent from North Queensland tick typhus, from South African tick bite fever, or from murine typhus, were solidly immune to rein-

[3] Guinea pigs bled about 20 days following inoculation with living rickettsiae.

fection with the homologous strains. However, they displayed considerable resistance to infection with the heterologous agents. These workers make the point that the resistance to heterologous strains may not be evidence of specific immunity but may be an acquired cellular resistance dependent upon other factors. Some of their data show that immunity to heterologous strains gradually disappears while that against the homologous agent persists for a longer period of time. Hence, the time interval between the original inoculation and the challenge inoculation is of utmost importance. Cross-immunity experiments with North Queensland tick typhus were performed by us, but the conditions were such that the criticisms advanced by Plotz in connection with his own experiments apply equally well to ours.

Conclusions

The studies in rabbits and the results of complement fixation with soluble antigen indicate that North Queensland tick typhus belongs to the "spotted fever" group of rickettsial diseases. The reactions obtained with North Queensland tick typhus rickettsial suspensions and sera of guinea pigs convalescent from various rickettsial infections would seem to indicate that this strain of rickettsia is not synonymous with any of the known members of the group (Rocky Mountain spotted fever, Maculatum disease, South African tick-bite fever, rickettsialpox, and boutonneuse fever) but represents a new strain within the "spotted fever" group.

REFERENCES

(*1*) Felix, A.: The rabbit as experimental animal in the study of the typhus group of viruses. Tr. Roy. Soc. Trop. Med. & Hyg. **26:** 365–378 (1933).
(*2*) Davis, Gordon E.: The Weil-Felix reaction in experimental Rocky Mountain spotted fever and certain other typhus-like diseases. Pub. Health Rep. **50:** 404–412 (1935).
(*3*) Plotz, Harry, Smadel, Joseph E., Bennett, Byron L., Reagan, Reginald L., and Snyder, Merrill J.: North Queensland tick typhus: Studies of the etiological agent and its relation to other rickettsial diseases. M. J. Australia **2:** 263–277 (1946).
(*4*) Davis, Gordon E.: Complement fixation in yellow fever in monkey and man. Am. J. Hyg. **13:** 79–128 (1931).
(*5*) Topping, Norman H., and Shepard, Charles C.: The preparation of antigens from yolk sacs infected with rickettsiae. Pub. Health Rep. **61:** 701–707 (1946).
(*6*) Plotz, Harry, Reagan, Reginald L., and Wertman, Kenneth: Differentiation between fievre boutonneuse and Rocky Mountain spotted fever by means of complement fixation. Proc. Soc. Exper. Biol. & Med. **55:** 173–176 (1944).
(*7*) Editorial: North Queensland tick typhus. Bull. U. S. Army M. Dept. **7:** 4–5 (1947).

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 NOVEMBER 20, 1948

A total of 651 cases of poliomyelitis was reported, as compared with 669 last week, 463 for the corresponding week in 1946, and a 5-year (1943–47) median of 256. A decline of 82 cases in 9 of the 17 States reporting currently 10 or more cases was more than offset by the increase of 89 cases in the other 8 States. The 6 States reporting more than 18 cases are as follows (last week's figures in parentheses): California 245 (198), South Dakota 65 (90), Minnesota 45 (30), Texas 33 (27), New York 27 (20), and Wisconsin 21 (20). The total reported for the year to date is 25,692, as compared with 10,070 in 1947, and 23,894 in 1946, the lowest and the highest figures for the corresponding periods of the past 5 years.

A total of 4,036 cases of measles was reported (last week 2,761, 5-year median 1,696). The largest increases reported currently were in Massachusetts (522 to 737 cases), Michigan (170 to 306), Pennsylvania (96 to 206), Washington (50 to 155), Colorado (61 to 165), and Wisconsin (135 to 237). Both the current figure and the cumulative total since the average seasonal low week of the disease (17,776) are above the corresponding figures for all prior years since 1943.

Of the current total of 2,067 cases of influenza reported (last week 1,890, 5-year median 2,104), no State reported more than 75 cases except Texas (1,127, last week 1,004), South Carolina (314, last week 278), Virginia 220 (last week 250), and Arizona (110, last week 56). The total reported since July 31 is 21,343, 5-year median 16,565.

One case of anthrax was reported during the week, in New Jersey, and 2 cases of Rocky Mountain spotted fever, 1 each in Georgia and Oklahoma. Of 16 cases of tularemia reported in 12 States, Indiana and Arkansas each reported 3 cases. Of 85 cases of typhoid fever (last week 61), New Mexico reported 28 (last week 8).

Deaths recorded during the week in 93 large cities in the United States totaled 9,217, as compared with 8,539 last week, 9,212 and 8,951, respectively, for the corresponding weeks of 1947 and 1946, and a 3-year (1945–47) median of 8,951. The total for the year to date is 430,573, as compared with 430,544 for the corresponding period last year. Infant deaths totaled 686, as compared with 625 last week and a 3-year median of 641. The cumulative figure is 31,245, same period last year, 34,525.

Telegraphic case reports from State health officers for week ended November 20, 1948

[Leaders indicate that no cases were reported]

Division and State	Diphtheria	Encephalitis, infectious	Influenza	Measles	Meningitis, meningococcal	Pneumonia	Poliomyelitis	Rocky Mt. spotted fever	Scarlet fever	Smallpox	Tularemia	Typhoid and paratyphoid fever [d]	Whooping cough	Rabies in animals
NEW ENGLAND														
Maine	2			203	1	9	2		16				25	
New Hampshire				2		1	1		4				6	
Vermont				34		1	1		6			1	12	
Massachusetts	10			737	1	14	1		106			1	58	
Rhode Island				2		1			2				3	
Connecticut			2	61	1	45	1		23			1	15	
MIDDLE ATLANTIC														
New York	13	2	[b] 4	264	6	230	27		[c] 112			4	123	13
New Jersey	3		2	71		42	10		56		1	2	30	4
Pennsylvania	2		(b)	206	3		12		100			2	99	
EAST NORTH CENTRAL														
Ohio	9		5	52	2	44	10		161			1	36	20
Indiana	15		12	35		20	3		32		3	1	16	12
Illinois	1	1	5	21	6	97	17		113		1	2	46	3
Michigan [a]	10		1	306	1	38	10		124				18	2
Wisconsin			2	237	1	4	21		31				20	
WEST NORTH CENTRAL														
Minnesota	3			6	1	7	45		51				1	
Iowa				7		6	18		18				3	1
Missouri	4		1	108	2	12	5		29			3	15	
North Dakota			2	33	1		1		14					
South Dakota				4		4	65		1					
Nebraska	3			6			3		22				3	
Kansas		1	1	7		6	4		20				1	
SOUTH ATLANTIC														
Delaware				2					2					
Maryland [a]	2			92	1	36	5		14			3	15	
District of Columbia				4		11	3		5					
Virginia	4		220	54	3	24	7		5		1	3	13	1
West Virginia	6		9	29	1	3	3		31			6	30	
North Carolina	14			30	3		13		30			2	14	
South Carolina	17		314	16	1	80	2		8			1	12	1
Georgia	15	1	8	1	1	15	5	1	37			7	1	7
Florida	11	1	15	25	7	17	4		8		1	1	3	14

EAST SOUTH CENTRAL														
Kentucky	6		6	85	1	8			52				36	
Tennessee	7	1	17	6	1	43	11		53			3	6	
Alabama	34		3	52	3	23	1		35			1	1	10
Mississippi [a]	5		2	4		12	1		17		1	2		
WEST SOUTH CENTRAL														
Arkansas	2		75	43	2	21	1		6		3		7	3
Louisiana	8		9	10		33	4		9		1	2		
Oklahoma	2		25	6	1	10	6	1	25		1			3
Texas	27		1,127	276	6	147	33		26		1	3	61	24
MOUNTAIN														
Montana			5	6		1			10				7	
Idaho	1		23	9		15	7		[c] 16				2	
Wyoming			2	58		2	2		4			1		
Colorado	3		42	165	1	24	2		16				8	
New Mexico	2		2	20		14			3			28		
Arizona	1		110	41		15	2		1		1	1	10	
Utah [a]			2	159		2	14		8		1		7	
Nevada									1					
PACIFIC														
Washington	5		2	155	1	4	13		26				6	
Oregon	2		4	96		35	10		28				14	
California	6		8	190	5	24	245		83			3	48	1
Total	255	7	2,067	4,036	64	1,200	651	2	1,600		16	85	831	
Median, 1943–47	408	12	2,104	1,696	107		256	1	2,683	8	10	75	2,268	
Year to date 46 weeks	8,475	509	160,158	569,190	2,887		25,692	514	67,195	54	847	3,288	68,143	
Median, 1943–47	11,921	589	206,762	561,941	7,314		12,928	461	121,996	319	714	4,531	111,507	
Seasonal low week ends	(27th) July 10		(30th) July 31	(35th) Sept. 4	(37th) Sept. 18		(11th) Mar. 20		(32d) Aug. 14	(35th) Sept. 4		(11th) Mar. 20	(39th) Oct. 2	
Since seasonal low week	3,865		21,343	17,776	451		25,342		11,579	4		2,815	5,220	
Median, 1943–47	5,657		16,565	10,258	819		12,531		22,136	40		3,907	13,971	

[a] Period ended earlier than Saturday.
[b] New York City and Philadelphia only, respectively.
[c] Including cases reported as streptococcal infections and septic sore throat.
[d] Including paratyphoid fever, reported separately, as follows: New York 2; Indiana 1; Missouri 1; Virginia 1; Louisiana 1; Texas 1; Arizona 1; California 1.
Salmonella infections, not included were reported, as follows: Massachusetts 1; New York 1.

Alaska: Meningitis 1; scarlet fever 6.
Territory of Hawaii: Measles 198; whooping cough 4.
Anthrax: New Jersey 1 case.

FOREIGN REPORTS

CANADA

Provinces—Communicable diseases—Week ended October 30, 1948.—During the week ended October 30, 1948, cases of certain communicable diseases were reported by the Dominion Bureau of Statistics of Canada as follows:

Disease	Prince Edward Island	Nova Scotia	New Brunswick	Quebec	Ontario	Manitoba	Saskatchewan	Alberta	British Columbia*	Total
Chickenpox		29	1	126	240	62	56	94	368	976
Diphtheria			1	18	3	6		2	6	36
Dysentery, bacillary						1			1	2
Encephalitis, infectious							1			1
German measles				6	21			3	19	49
Influenza		9			7	1			3	20
Measles		49	1	63	99	20	49	11	75	367
Meningitis, meningococcal			1	1			2		2	6
Mumps		5		84	123	35	70	20	51	388
Poliomyelitis		3		1	6	4	5	8	3	30
Scarlet fever		13	6	83	63	5	11	11	13	205
Tuberculosis (all forms)		4	4	79	26	18	14	72	103	320
Typhoid and paratyphoid fever				8	1			1	1	11
Undulant fever		1			1					2
Venereal diseases:										
Gonorrhea		16	11	90	104	24	20	34	128	427
Syphilis		9	12	96	58	10	4	6	27	222
Other forms									2	2
Whooping cough				55	25	2	13	5	5	105

*Figures for British Columbia are for two-week period, ended October 30, 1948.

FINLAND

Notifiable diseases—September 1948.—During the month of September 1948, cases of certain notifiable diseases were reported in Finland as follows:

Disease	Cases	Disease	Cases
Cerebrospinal meningitis	8	Paratyphoid fever	124
Diphtheria	247	Poliomyelitis	18
Dysentery	7	Scarlet fever	258
Gonorrhea	1,158	Syphilis	236
Malaria	1	Typhoid fever	16

MEXICO

Mexico City—Poliomyelitis.—During the week ended November 6, 1948, 15 cases of poliomyelitis were reported in Mexico City, Mexico.

NETHERLANDS

Amsterdam—Psittacosis.—During the week ended October 30, 1948, 1 case of psittacosis was reported in Amsterdam, Netherlands.

NEW ZEALAND

Notifiable diseases—4 weeks ended October 30, 1948.—During the 4 weeks ended October 30, 1948, certain notifiable diseases were reported in New Zealand as follows:

Disease	Cases	Deaths	Disease	Cases	Deaths
Cerebrospinal meningitis	10	1	Ophthalmia neonatorum	1	
Diphtheria	15		Puerperal fever	6	
Dysentery:			Poliomyelitis	85	3
Amebic	3		Scarlet fever	80	
Bacillary	5		Tetanus	3	1
Erysipelas	14		Trachoma	1	
Food poisoning	4		Tuberculosis (all forms)	134	58
Lethargic encephalitis	4	2	Typhoid fever	5	1
Malaria	1		Undulant fever	3	

REPORTS OF CHOLERA, PLAGUE, SMALLPOX, TYPHUS FEVER, AND YELLOW FEVER RECEIVED DURING THE CURRENT WEEK

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

China—Kwangtung Province.—It is reported that an epidemic of cholera is raging in Waiyang district, Kwangtung Province, on the East River. It is stated that precautionary measures have been taken by the local authorities.

India—Madras.—During the week ended November 13, 1948, 11 cases of cholera were reported in the city of Madras, India.

Pakistan—Lahore.—During the period September 25–October 23, 1948, 105 cases of cholera, with 19 deaths, were reported in the city of Lahore, Pakistan.

Plague

Ecuador—Loja Province—Macara County.—Plague has been reported in Macara County, Loja Province, Ecuador, as follows: During the period September 16–30, 1948, 1 case in Curitachi; during the period October 1–15, 1 case in Larama, 1 case in Potrerillo.

India—Bombay.—Information dated November 9, 1948, states that 6 cases of bubonic plague have been reported in the city of Bombay, India. During the week ended November 13, 2 cases were reported. During the week ended November 6, 1948, 15 plague-infected rats were reported caught in the urban area of Bombay.

Java—Surabaya.—Information dated November 19, 1948, states that 6 nonimported cases of plague have been reported in Surabaya, Java.

Union of South Africa—Orange Free State.—During the week ended November 6, 1948, 1 case of plague was reported at Verdeeld Farm, Kopies District, Orange Free State, Union of South Africa.

Smallpox

Sierra Leone.—For the week ended September 25, 1948, 16 cases of smallpox, with 5 deaths, were reported in Sierra Leone.

Syria.—During the week ended October 23, 1948, 38 cases of smallpox were reported in Syria, including 10 cases in Aleppo, and 4 cases in Damascus.

Typhus Fever

Egypt—Alexandria.—During the week ended October 23, 1948, 37 cases of typhus fever were reported in Alexandria, Egypt.

Correction: For the week ended October 2, 1948, only 1 case of typhus fever was reported in Alexandria instead of 16, as stated in Public Health Reports for November 5, 1948 (p. 1, 481).

India—Calcutta.—During the week ended August 14, 1948, 1 fatal case of typhus fever was reported in Calcutta, India.

Italy—Sicily.—During the period July 1–31, 1948, 4 cases of typhus fever were reported in Sicily, and during the period August 1–31, 9 cases were reported.

NOTE: No reports were received on yellow fever.

DEATHS DURING WEEK ENDED NOVEMBER 13, 1948

[From the Weekly Mortality Index, issued by the National Office of Vital Statistics]

	Week ended Nov. 13, 1948	Corresponding week, 1947
Data for 93 large cities of the United States:		
Total deaths	8,539	9,342
Median for 3 prior years	8,836	
Total deaths, first 46 weeks of year	421,356	421,332
Deaths under 1 year of age	625	721
Median for 3 prior years	721	
Deaths under 1 year of age, first 46 weeks of year	30,559	33,884
Data from industrial insurance companies:		
Policies in force	70,814,298	67,065,060
Number of death claims	10,597	10,320
Death claims per 1,000 policies in force, annual rate	7.8	8.0
Death claims per 1,000 policies, first 46 weeks of year, annual rate	9.2	9.2