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Effects of Reservoir Operation on Stream Water Quality

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During the past 15 years there has been a great increase in interest in conservation of our natural resources generally and of our water resources in particular. Many dams and reservoirs have been constructed by the Federal Government and many more have been authorized for construction for flood control, irrigation, navigation improvement and power production. Some of these projects have been built to serve a single purpose but there has been an increasing tendency toward multiple-purpose projects. In an increasing number of instances the possible effects of the developments, beneficial or detrimental, on water quality are being considered.

It is frequently possible and practical to make important improvements in water quality by proper operation of a reservoir. Such improvements may be obtained without any special provisions or they may require the specific allocation of storage. The benefits that may be obtained and the damages that may be caused must be considered if the maximum benefit is to be derived from any development. These benefits and damages can be evaluated, at least in part, in monetary terms and in some cases they are of considerable magnitude.

For the past 10 years the Public Health Service has cooperated with various district offices of the Army Corps of Engineers in investigating water quality aspects of water control projects. Certain general principles have been recognized in this work and are the subject of this discussion.

In general, where wastes are discharged into flowing streams, the seriousness and extent of the damage caused by the resulting pollution depends upon the volume of stream flow as related to the amount and strength of the wastes discharged. A variety of other factors are involved, so that it is not practicable to establish standard dilution ratios which would be generally applicable. Nevertheless, the dilution available is one of the most important factors governing the

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extent and seriousness of pollution. Therefore, pollution damage can be decreased either by reducing the waste load or by increasing the volume of dilution water. Since most streams have widely varying natural flows and since the design of waste treatment works must be based upon critical low-flow conditions, it frequently is possible to reduce the degree of treatment required by storing flood waters for release during low-flow periods.

Several important qualifications to these statements must be made. For many types of wastes, dilution alone cannot be considered an entirely satisfactory substitute for treatment, at least within the range of dilutions which it might be practicable to provide. For example, oils and other floating solids will be objectionable in many cases regardless of dilution water available. Settleable solids, either putrescible or inert, will not be greatly affected by added dilution. Sludge deposits from sewage or other organic wastes may exert an effect on the stream that is disproportionate to the pollution load in solution and in suspension in the stream. The wastes from coal washeries and from placer mining operations are examples of inert wastes.

Neither is dilution usually a suitable solution for problems of bacterial pollution. In such cases, the effect of dilution may be offset by the reduced time of flow from the source of pollution to the point of water use with fewer organisms dying in the shorter time period. For such pollution problems, waste treatment is essential to solution of the problem and although additional dilution may be a valuable supplement to treatment, it cannot be considered a satisfactory substitute.

Low-flow augmentation can benefit water quality in other ways than by reducing the degree of waste treatment needed. In most streams the water during low-flow periods is more highly mineralized and harder than during periods of high flow. By storing a part of the softer water from periods of high surface run-off and releasing it during low-flow periods, the chemical quality at downstream points often can be improved. In special cases other types of benefits may be important. For instance, in areas where acid mine drainage is an important type of pollution, it may be possible to reduce the acid damage by storing flood waters for release during low-flow periods when acid concentrations are highest. Certain periodically acid streams or portions of streams may even be made continuously alkaline in this way.

Temperature reduction due to increased low flows may be of major economic value where the stream is used intensively for cooling water. An outstanding example of this is the Mahoning River which serves the Youngstown steel district in northeastern Ohio. Natural minimum flows were only about one-twentieth of the cooling water demands, and water temperatures rose to about 120° F. due to the repeated re-use of the stream water. These high temperatures not only decreased the usefulness of the water for cooling and increased the cost of cooling but even forced a curtailment of plant operations. High water temperature also increases the difficulty and cost of organic pollution abatement by reducing the solubility of oxygen and increasing the rate of decomposition of organic matter. Heat, in this case, can be considered a polluting agent. Flow regulation has aided greatly in overcoming the stream water temperature problem in the Mahoning Valley. Although this is an unusual situation, other places exist where industrial water use is intensive, and resulting high water temperature is a problem.

Water impoundments may create certain hazards and have some damaging effects on water quality. Although these are generally of less importance than the beneficial effects, they cannot be disregarded. Impoundment of water may aggravate the pollution problems of towns or industries along the reservoir shoreline. The reaeration capacity of streams, and in fact the self-purification capacity of the streams, varies with the velocity of flow. By changing a free-flowing stream into an arm of a reservoir with little current, the quality of the water at and near the source of pollution may be seriously affected. This has been quite noticeable on canalized rivers. Where the size of the reservoir and the amount of water available for dilution of the wastes is great, this effect is less serious.

A hazard to water works intakes may also be created by water impoundments. On free-flowing streams a water works intake may be safely placed a short distance above sources of pollution. Where this has been done and the stream subsequently becomes part of a reservoir, there is danger that wastes which formerly were carried away from the water intake may be carried toward it. The situation at Harriman, Tenn., is a notable example of this. At Cincinnati, Ohio, there is evidence that pollution from the city has traveled upstream to the water works intake even though the downstream velocity in the Ohio River is normally much greater than would be found in most reservoirs.

Water supplies downstream from a reservoir may be adversely affected if conditions in the reservoir are favorable for heavy production of algae. Obnoxious tastes and odors may be imparted to the water and necessitate special treatment for their removal. The services of an expert aquatic biologist are generally needed to predict the probable importance of such effects.

Where flow regulation is complete enough to eliminate the seasonal scouring of deposited material, some detriment may result. However, such complete regulation is quite unusual.

The operation of reservoirs for power generation frequently gives rise to very great fluctuations in flow in accordance with power demand. These fluctuations may create pollution problems that would not otherwise be of concern. During low-flow periods, power reservoirs in systems where steam power is also used are generally operated to carry peak power loads and may discharge little or no water during off-peak hours. The resulting wave of discharge is dampened as the water flows downstream, but wastes discharged a short distance below the reservoir may receive little or no dilution during part of the day or on certain days such as week ends and holidays, even though the daily and weekly average flows may be comparatively high. Under such conditions, the resulting wide fluctuations in water quality are maintained for a much greater distance downstream than the fluctuations in discharge and may seriously affect water supplies and aquatic life. In such situations the use of average discharges and average pollution loads for estimating stream conditions is misleading. Small re-regulation pools may be provided to reduce the fluctuations in flow.

Estimating Benefits

In estimating the monetary value of water quality benefits to be expected from the provision of low-flow augmentation, various approaches may be made to the problem. One can attempt to estimate directly the benefits which would be derived or the damages avoided by each of the various water users affected. The principal trouble with this approach is the extreme difficulty of evaluating some of the benefits. For instance, what value shall be estimated for the benefits to health of cleaning up a polluted stream? Also, the value of eliminating water quality conditions detrimental to aquatic life can seldom be estimated with any degree of assurance. The assignment of a monetary value to benefits of improved recreational utility is correspondingly hard. The direct benefits to riparian landowners from increased property values are very difficult to determine satisfactorily. Finally, purely aesthetic considerations are important factors in most instances and their evaluation in monetary terms is not practical.

A number of attempts have been made to develop methods for estimating the monetary value of such benefits. Estimates which can be made fairly accurately will reflect only a part, and sometimes a very small part, of the actual benefits.

Another approach which can be used to estimate the value of water quality improvements due to low-flow control is based on estimating the cost of obtaining similar needed improvements by the most economical alternative method. Two principal objections may be made to this procedure: (1) increased low flow sometimes can provide improvements that cannot be accomplished in any other way, and (2) if applied rigidly the procedure may lead at times to excessive benefit estimates since it assumes that the improvement in water quality is justified regardless of its cost. The first objection can be overcome if it is possible to estimate directly the damages eliminated or the benefits obtained by the water quality improvement. The second objection can be avoided by considering only those improvements which reasonably can be justified in the light of present and prospective water uses, of available methods of waste and water treatment, and of legal obligations.

Such an analysis generally involves (1) a zoning of the stream into reaches where water uses are comparatively homogeneous, and (2) the determination of suitable standards of water quality for each zone. This has been done in a number of river basins by various State and interstate agencies concerned with water pollution control and it is being done in a number of additional areas. With this as a basis, one can outline a program which will restore the streams to the prescribed quality in each zone. Certain types of treatment of sewage and industrial wastes at the various sources of pollution will be required under unregulated stream flow conditions. With flow regulation a lesser degree of treatment may be required. The saving in cost of waste treatment may properly be considered as a benefit derived from flow regulation.

A variety of types of data must be used in making an estimate of this sort. It is necessary to have enough information on the physical, chemical, and bacteriological quality of the water and on the amount and character of wastes entering the stream to permit the determination of the self-purification characteristics of the stream. In the absence of this information, assumptions based upon experience must be made.

It is often not economically feasible nor desirable to design waste treatment facilities to maintain the desired condition under absolute minimum stream flow conditions. It becomes necessary to adopt a minimum design stream flow which will insure that the desired quality is maintained during most periods but which may permit an occasional failure to maintain such a quality. The permissible frequency of such failure depends largely upon the seriousness of the consequences of failure. In most parts of the country critical conditions occur during the late summer months. Although stream flows may be lower during the fall and winter, the lower temperatures at such times usually make pollution effects less serious, except where winters are severe and ice coverage is complete enough to reduce reaeration greatly. The most common design flow used in studies by the Public Health Service has been the minimum monthly summer (June to September) flow which is equaled or exceeded, on the average, 9 years in 10. On streams where water quality is difficult to maintain and water uses do not require a high quality, greater frequency of failure may be permitted. Where seasonal industrial operations, such as canneries, beet sugar refineries, or distilleries, contribute important quantities of wastes, critical stream conditions may occur during their peak seasons and the design stream flow would be chosen accordingly.

In determining the cost of treatment, it is generally not feasible to make individual studies of each community or industry concerned. If consulting engineers' estimates are available, they can be used with suitable adjustments for changes in costs. Otherwise it is generally necessary to use average cost figures from existing plants. As pointed out previously, flow regulation cannot be considered an adequate substitute for primary treatment of sewage or of many kinds of industrial wastes. Consequently, flow regulation in such cases will effect a saving in waste treatment cost only insofar as it makes possible the elimination or a reduction in the degree of more complete treatment than plain sedimentation. The costs of providing this additional treatment are less variable than the costs of intercepting the wastes and providing the site and initial units for the treatment plant. Consequently, the errors introduced through the use of average cost figures will not usually be serious.

Estimates of the benefits due to reduction in water hardness can be made by determining the reduction in hardness at various downstream points due to flow regulation. From knowledge of water uses for which hardness is troublesome, estimates can be made of the cost of an equivalent improvement in quality by water treatment or of the extra cost of soap due to usage of harder water. A similar procedure can be followed in estimating benefits due to reduction in mineral content.

Careful studies of the damages due to acid mine drainage in the upper portions of the Ohio River drainage basin have been made in connection with the Ohio River Pollution Survey.[1] Heavy acid loads and severe corrosion damage would seldom be encountered in other parts of the country, but in this area it appears that both flow regulation and other corrective measures will be required to achieve the best results. The damages from acid mine drainage include increased hardness and acidity in municipal and industrial water supplies, and accelerated corrosion of river boats and barges, river and harbor structures, and power plants. During the Ohio River pollution survey, information on damages and costs of treatment was obtained directly from the municipalities, industries, transportation and power companies concerned and from the United States Engineer Office. This information has afforded a basis for evaluating the benefits due to acid reduction which can be accomplished by low-flow regulation. In estimating such benefits a reservoir operating schedule is used which

[1] House Document 266, 78th Cong., 1st ses.

provides for storage of alkaline water during high flows and for release of this water to neutralize acidity during low-flow periods.

Water-temperature-reduction benefits can be estimated in a similar manner to those due to hardness, mineral content, and acid reduction. A study is made of water uses where temperature is an important consideration, the losses experienced due to high temperatures are determined, and the benefits at various points estimated from a knowledge of the rate of heat loss from water to air, the water and air temperatures, the water uses, and the stream flow. The principal industrial damages experienced on the Mahoning River were due to reduced power plant efficiencies because of poorer condenser operation, greater pumping costs and higher maintenance costs on various parts of the plant where the high temperature water was used.[2] Estimates of such damages were obtained from the industries affected. In addition, the degree of sewage treatment required to obtain a given stream water quality was increased because of the high temperatures.

Estimates of damage due to water impoundments can be made in similar ways. Reduction in reaeration capacity may require a higher degree of waste treatment than would be required without impoundment. The additional treatment cost is properly chargeable as a damage due to the impoundment. Where the reservoir endangers a water supply by making possible its pollution by wastes discharged downstream from it, the cost of correcting the situation by moving the water intake, the waste outlet, or by some other effective method, is a measure of the damage done.

In considering any of these benefits or damages, it is necessary to make estimates of probable future conditions. This presents no unusual difficulties in so far as domestic sewage is concerned but may be very difficult in the case of industrial waste discharges. Information on the plans of specific industries for expansion or process changes, information on new establishments expected, and study of past trends in industrial activity, may be used as guides in estimating future industrial waste loads. Increased dilution water during low-flow periods is of unquestioned value and benefit to water quality and it is believed that the methods adopted for estimating its monetary value provide a rational and conservative approach to the problem. Intangible benefits which are not evaluated are also important.

[2] The method used in dealing with this problem is discussed in detail in the article, Cooling-Water Benefits From Increased River Flows, by M. LeBosquet, Jr., Journal of the New England Waters Works Association, 60: 111-116, June 1946.

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Incidence of Q Fever in Eastern Washington

— A Serological Survey —

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Among the advances in knowledge of Q fever made during and after the recent war is the addition of new geographic areas to those in which it was previously known to occur. Prior to the last war the natural occurrence of this disease in man was considered limited to Australia (*3*, *8*) although the infectious agent was recovered from ticks in Montana by Davis and his colleagues as early as 1938 (*7*, *10*). However, one naturally acquired case of Q fever was reported from Montana in 1941 (*9*) and a few suspected cases (*5*). Recently the disease has been reported from scattered parts of the United States. The etiological agent *Coxiella burnetii* has also been recovered from different species of ticks in widely separated areas.

Although Washington presents geographic and climatic conditions differing from those of areas where natural outbreaks have been reported previously, it is one of the Northwestern States offering large areas suitable for cattle raising and has a proportionately higher percentage of cattle than many of the Eastern or other Western States. Cattle are thought to be a reservoir of the disease. Several species of ticks found in this area are identical with those known to harbor the rickettsia in other areas. Therefore, there is no reason to believe that the disease should not occur here.

Furthermore, the etiological agent of Q fever has been recovered from the Rocky Mountain wood tick *Dermacentor andersoni* in Montana (*7*) and from the Pacific coast tick *Dermacentor occidentalis* in Oregon (*5*). Sporadic natural infections have also been reported from Montana and Idaho. Since Idaho and Oregon border the State of Washington on the east and south, respectively, it was felt that a survey of the sera of animals in this area might be of value to determine if any reservoir of this disease is present.

The main objective was to determine whether this disease exists in eastern Washington, and if so, to estimate its prevalence. Our knowledge of the effects of Q fever in cattle and other domestic animals is obscure, although it is known that the disease causes an inapparent or mild infection in cattle (*11*, *12*). Recent publications reveal that the primary interest in Q fever in cattle is due to its public health aspects and to economic considerations. Occasional deaths may occur among

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infected animals. Infection may spread to associated animals as well as to man, and infected milk and meat products eventually may be excluded from certain markets.

The explosive nature of the human respiratory disease caused by *C. burnetii* as manifested in the recent outbreaks in the United States (*6*, *14*, *15*) and other countries (*13*) confronts public health officials with the problem as to how it best may be controlled or eradicated. The symptoms in many cases of Q fever in humans are so mild or inapparent that they escape diagnosis. Fortunately, the complement-fixation test is sufficiently accurate and sensitive to detect the specific antibodies in the sera of man and animals which are or have been infected by *C. burnetii*. Although the incidence of human Q fever in the United States appears to be low at present, the disease is considered important enough by leading public health officials to warrant thorough investigation. Therefore, it is considered that this study may be of some value to the people of this part of the State, and further investigation may protect the livestock industry against economic losses in the future.

Experimental Procedure

The variety of manifestations which accompany this disease entity often does not permit a specific diagnosis on clinical grounds alone. Therefore, the need for accurate serological tests to aid in the diagnosis of Q fever is apparent. Diagnosis of Q fever by serologic means is highly satisfactory (*1*, *2*). Both complement-fixing and agglutinating antibodies appear in the sera of the patients. Complement-fixing antibodies appear in low dilutions as early as the seventh day of illness but may not be present until the thirteenth day. There is a progressive rise in antibody titer in each case until 21–30 days, after which it gradually falls. Unfortunately, very little information is available in the literature concerning the maximum period of persistence of antibodies following Q fever acquired under natural conditions.

The procedure followed in the complement-fixation test in this study was essentially as described by Bengston in 1944 (*2*). Instead of 1-hour fixation as recommended, the tests were placed in the refrigerator overnight, as is done in the Rocky Mountain Laboratory, Hamilton, Mont. The overnight fixation gives a somewhat higher titer and does not seem to increase nonspecific reactions over those obtained with 1-hour fixation. The sera inactivated by heating at 56° C. for 30 minutes were diluted serially in 0.2 ml. amounts. Two complement-fixing units of antigen were added in a volume of 0.2 ml. and followed by two full units of complement, likewise in a volume of 0.2 ml. The tubes were shaken and placed in the refrigerator overnight. The following morning 0.4 ml. of sensitized sheep red blood cells was added to each tube. The tubes were incubated in a water bath at 37° C. for one-half hour and the results were read.[1]

Sources of Serum Specimens Examined

During the course of an 8-month period, 675 samples of blood sera of man and animals were examined to determine the presence of specific Q fever antibodies by complement-fixation procedure.[2] The samples were collected irrespective of the clinical diagnosis, though particular attention was focused on the sera of the patients suffering from respiratory infections. Since most of the outbreaks of Q fever have been found among persons in close contact with animals, it was thought advisable to study the blood sera of the junior and senior students of the College of Veterinary Medicine, as they had been in contact with animals for 2–4 years.

Samples of the blood sera from most of the animals of the State College of Washington beef and dairy herds were examined in addition to the usual routine blood samples obtained from the veterinary clinic. Since the brucellosis investigation unit of the college was expanding its program during the time of this study, it was possible to procure through its cooperation bovine sera from various ranches in the vicinity of Pullman. Through the cooperation of the United States Department of Agriculture and the Idaho Bureau of Animal Industry, Boise, Idaho, some animal sera were obtained from them also and tested for Q fever complement-fixing antibodies. Though main attention was focused in this study on cattle sera, examinations also were made of blood sera of horses, dogs, sheep, and foxes.[3]

The number of sera, the types of sera, and the percentage of positives are shown in table 1; human case histories with suggested source of

Table 1. *Summary of results of serological studies of sera from different sources*

Type of sera	Number of sera examined	Number of positive sera	Number of negative sera	Percentage of positive	Serum dilutions*					
					1:8	1:16	1:32	1:64	1:128	1:256
Human	289	6	283	2.076	0	3	1	1	1	0
Bovine	327	9	318	2.446	3	2	2	1	1	0
Canine (10 foxes)	27	0	27	0	0	0	0	0	0	0
Others (13 horses, 7 hogs, 12 sheep)	32	0	32	0	0	0	0	0	0	0
Totals	675	15	660	0	3	5	3	2	2	0

*Titers shown are highest dilutions in which sera reacted.

[1] The Q fever antigens used in this study were obtained from the Rocky Mountain Laboratory through the courtesy of Dr. R. R. Parker, Director, and David B. Lackman, Senior Scientist, Public Health Service, Hamilton, Mont.

[2] The samples of human sera were obtained from Finch Memorial Hospital, Pullman, Wash., through the courtesy of the hospital authorities.

[3] The samples of sera of foxes were obtained from the Fur Animal Disease Investigation Laboratory, Pullman, Wash.

infection and end titers of the sera in table 2; and descriptive histories of cattle and the end titers of the sera in table 3.

In this study all the sera, both human and animal, showing complement fixation in dilutions of 1:8 and above have been considered positive. Shepard and Huebner (*15*) consider complement fixation in dilutions of 1:4 and above in human sera and in dilutions of 1:8 and above in animal sera, as positive for Q fever.

Table 2. *Human case histories with suggested source of infection*

No.	Sex	Age	Period of occupational exposure with animals	Probable source of infection	Probable date of illness	End titer of the sera
1	F	26	4 years	Occupational [1]	Spring 1947	1:64
2	M	28	do	Occupational [1] or arthropod	Summer 1947	1:128
3	M	27	6 years	Occupational [1]	Spring 1947	1:32
4	M	22	Nil	Not known	Not known	1:16
5	M	21	do	do	do	1:16
6	F	20	do	do	do	1:16

[1] Student of the College of Veterinary Medicine, State College of Washington.

Table 3. *Descriptive histories of cattle showing antibodies specific for Q fever*

Case no.	Identification No.	Location	Breed	Sex	Age in years	End titer of sera
1	9117	Coeur d'Alene, Idaho	Holstein	F	8	1:64
2	A 26	State College of Washington beef herd.	Angus	F		1:128
3	Vet. Clinic [1] 4771	Palouse, Wash	Shorthorn	M	3	1:32
4	32	State College of Washington beef herd.	do	F	15	1:8
5	A 71	do	Angus	F	1	1:16
6	A 67	do	do	F	2	1:8
7	14946	Palouse, Wash	Guernsey	F	5	1:16
8	Vet. Clinic [1] 30019	Colfax, Wash	Shorthorn	F	4	1:32
9	Vet. Clinic [1] 4675	Vet. Clinic [1]	Jersey	M		1:8

[1] Veterinary Clinic of the College of Veterinary Medicine, State College of Washington.

Discussion

Out of 289 samples of human sera examined, 6 were found to contain antibodies specific for Q fever with titers varying from 1:8 to 1:128. The percentage of positive sera among all samples from eastern Washington and neighboring Idaho, proved to be 2.076 percent in human beings, and 2.446 percent in cattle. The sera of all other species of animals were negative. It is interesting to note that the three positive human sera which showed higher titers were obtained from the students of the College of Veterinary Medicine. All three reported a past history of respiratory infection.

Case 1: A female native of Lewiston, Idaho, and a senior student in the College of Veterinary Medicine had a severe infection of upper respiratory tract, which she considered as "flu", during the spring of 1947 while in Pullman. The symptoms were those of cold and "flu" with persistent headache. The duration of the illness was about a week and she was not hospitalized.

Case 2: A male senior student of the College of Veterinary Medicine had an attack of influenza-like infection while in Walla Walla during the summer of 1947. He had frequently visited the Blue Mountains for fishing prior to his illness. His illness lasted about 10 days.

Case 3: A male native of Ventura County, Calif., a junior student in the College of Veterinary Medicine had been in close contact with animals since 1940. He had worked in a packing plant during the summer of 1942 and in citrus pest control the succeeding year. He had an influenza-like infection with severe headache in the spring of 1947 while in Pullman.

Cases 4, 5 and 6: In the remaining three persons, the probable source of infection could not be traced since these persons had no occupational contact with animals. These three sera were obtained from patients in the Finch Memorial Hospital where they were undergoing treatment for conditions other than respiratory infections. These patients probably had past infection of Q fever since their sera showed low titers of complement-fixing antibodies and since there was no increase in the titers during the hospitalization period.

Most of the samples of the positive sera were sent to Dr. David B. Lackman, Rocky Mountain Laboratory, Hamilton, Mont., for confirmation of the results obtained and for further studies to determine any possible cross reactions of the sera with other rickettsial diseases. The sera were examined also for antibodies against *Pasteurella tularensis* or *Brucella tularensis* and the results were negative. The results reported by Dr. Lackman corresponded with those obtained here except for slight variations in the reading of the end titers of the sera. The sera showed no cross reactions with other rickettsial diseases.

A striking feature of the epidemiology of Q fever is its peculiar relationship to occupation or contact with animals. In this study the sera of the persons who had apparently the greatest exposure exhibited higher titers of complement-fixing antibodies specific for Q fever. They also showed a definite history of past infection of the upper respiratory tract. The patients whose sera showed lower titers failed to give any definite history of attacks particularly suggestive of Q fever. They might have undergone mild or inapparent attacks of this disease.

In the first three cases there had been definite contact with animals of various species, both in their previous history and in their work in the veterinary clinics. In case 3, a history of work in a meat packing plant and citrus pest control also was obtained. It is questionable whether these factors were related to the source of infection, since the person worked in these places about 6 to 7 years previously. The titer of antibodies probably indicates recent infection since antibodies are reported to disappear within 3 to 4 years. From the histories of the cases, the persons probably acquired their antibodies in the course of their work in the veterinary clinics. The sera from a number of the students of veterinary medicine showed fixation in very low dilution. Such reactions, though below the diagnostic level, might be indicative of residual titers from pest infection.

In case 2, a blood-sucking anthropod vector might have been the source of infection, as the patient had visited the Blue Mountains, Walla Walla, for fishing many times prior to his illness.

Since cattle, sheep, and goats are the common animals reported to be naturally infected with Q fever, it seems logical to believe that these persons might have contracted the disease in the veterinary clinics through contact while attending the animals. Inasmuch as the number of sheep and goats treated in the clinics is small when compared to that of cattle, our attention is directed primarily on cattle. This is substantiated by the presence of complement-fixing antibodies specific for Q fever in the blood sera of cattle of this locality.

In cases 4, 5, and 6, there was no apparent association with the livestock industry. This suggests that the sources of infection or modes of spread of the disease are not limited to this particular industrial occupation. There is also evidence in the literature that Q fever is rarely, if ever, transmitted directly from one human to another. Dairy cows have been found infected with Q fever, and *C. burnetii* has been recovered from raw milk (*12*, *15*). Hence, in the absence of evidence of a probable source of infection in the last 3 cases, it is reasonable to believe that infected raw milk or air-borne dust might have been the cause.

Animal Sera: Of the total nine positive sera, the first two showed fairly high titers of 1:64 and 1:128, respectively. Of the cattle sera studied, 2.446 percent showed antibodies specific for Q fever. The disease appeared to be present in the beef herd and absent from the dairy herd, although the absence of positive tests for Q fever in the dairy herd may be due to good sanitary practices. Also in contrast to the beef herd, the dairy herd, not put out to pasture, was not exposed to tick infestation. Out of more than 80 samples of the beef herd examined, 4 sera were found to have complement-fixing antibodies specific for Q fever with diagnostic titers. The serologically positive animals appeared healthy. No ticks were found on the animals; however, the examinations were made during winter when ticks are rarely present.

The serum of case 1 was obtained from the Idaho Bureau of Animal Industry and is one of the two which showed high titers. The animal blood samples of cases 3, 7, 8, and 9 were obtained from the veterinary clinic where the animals were undergoing treatment for various ailments. Two of these animals were from the nearby town of Palouse and one from Colfax, Wash.

The breed, age, and sex of the animals were not considered to be of interest, since complement-fixing antibodies were found in both sexes and in ages varying from 1 to 15 years.

The evidence presented indicates that Q fever exists in the eastern part of Washington and that cattle are an important reservoir.

Summary

1. Q fever exists in eastern Washington in both humans and animals.

2. Six of 289 samples of human sera examined showed Q fever complement-fixing antibodies, in titers of 1:8 to 1:128.

3. Three of the cases in humans were among students of the College of Veterinary Medicine who had been in close contact with animals.

4. Three of the persons whose sera were positive by the complement-fixation test for Q fever had no occupational contact with animals, and their histories gave no indication of previous respiratory infection suggestive of Q fever.

5. Nine of 327 samples of cattle sera showed Q fever antibodies, 2 in high titers.

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(*14*) Shepard, C. C.: An outbreak of Q fever in a Chicago packing house. Am. J. Hyg. **46:** 185–192 (1947).

(*15*) Shepard, C. C. and Huebner, R. J.: Q fever in Los Angeles County. Am. J. Pub. Health **38:** 781–788 (1948).

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 SEPTEMBER 10, 1949

A decline was recorded in the reported incidence of poliomyelitis for the third consecutive week. A total of 2,701 cases was reported, as compared with 3,197 last week, a decrease of approximately 15 percent. The 5-year (1944–48) median is 1,498, and the figure for the corresponding week last year was 1,526, representing an increase of 21 cases (1.4 percent). Currently, net declines were reported in the New England area (from 343 cases last week to 284), the Middle Atlantic (753 to 518), the East North Central (902 to 714), the West North Central (522 to 452), and the East South Central (115 to 113). In the South Atlantic, West South Central, Mountain, and Pacific areas, each showing a slight net increase, an aggregate of 620 cases was reported (last week 562).

The 32 States reporting more than 18 cases each are as follows (last week's figures in parentheses): *Increases*—Maine 47 (37), Vermont 21 (15), Ohio 178 (171), Indiana 68 (61), Iowa 75 (72), South Dakota 44 (38), Nebraska 60 (39), West Virginia 27 (23), Georgia 21 (5), Mississippi 28 (6), Oklahoma 86 (50), Texas 65 (63), Idaho 28 (27), Colorado 77 (55), California 121 (89); *decreases*—Massachusetts 145 (194), Connecticut 43 (56), New York 336 (538), New Jersey 105 (137), Pennsylvania 77 (78), Illinois 191 (282), Michigan 195 (287), Wisconsin 82 (101), Minnesota 128 (168), Missouri 75 (101), North Dakota 23 (52), Kansas 47 (52), Virginia 21 (27), Kentucky 41 (60), Tennessee 32 (36), Arkansas 34 (44), Washington 34 (50). For the year to date, 26,457 cases have been reported, as compared with 14,183 for the same period last year and a 5-year median of 10,972.

One case of smallpox was reported during the week, in Kentucky. The total for the year to date is 42, same period last year 50, 5-year median 275.

A total of 7,776 deaths was recorded during the week in 93 large cities of the United States, as compared with 8,405 last week, 7,804 and 8,230, respectively, for the corresponding weeks of 1948 and 1947, and a 3-year (1946–48) median of 8,230. The total for the year to date is 329,380, as compared with 332,340 for the corresponding period last year. Infant deaths totaled 550, last week 656, same week last year 604, 3-year median 680. The cumulative figure is 23,006, same period last year 23,632.

Telegraphic case reports from State health officers for week ended Sept. 10, 1949

(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 [e]	Whooping cough	Rabies in animals
NEW ENGLAND														
Maine	----	----	----	2	----	14	47	----	2	----	----	1	20	----
New Hampshire	----	----	----	----	----	----	18	----	----	----	----	----	4	----
Vermont	----	----	----	2	----	----	21	----	[d] 2	----	----	----	10	----
Massachusetts	----	----	----	4	1	----	145	----	7	----	----	----	98	----
Rhode Island	----	----	----	1	----	----	10	----	1	----	----	----	3	----
Connecticut	----	----	----	8	----	26	43	----	1	----	----	1	31	----
MIDDLE ATLANTIC														
New York	2	1	(c)	59	2	79	336	2	[d] 15	----	----	6	172	11
New Jersey	1	2	3	19	2	58	105	----	• 1	----	----	2	107	----
Pennsylvania	4	----	(c)	34	3	31	77	1	11	----	----	8	151	----
EAST NORTH CENTRAL														
Ohio	3	----	----	10	5	21	178	----	19	----	----	6	56	7
Indiana	5	2	----	9	----	3	68	----	10	----	----	2	44	4
Illinois	----	1	----	12	3	48	191	1	9	----	----	1	72	----
Michigan [a]	1	1	----	23	2	18	[1] 195	----	6	----	----	1	123	----
Wisconsin	----	1	3	36	----	2	82	----	2	----	----	----	46	----
WEST NORTH CENTRAL														
Minnesota	2	----	----	3	1	1	128	----	5	----	----	2	12	1
Iowa	----	----	----	4	----	----	75	----	3	----	----	----	1	1
Missouri	2	----	----	1	6	4	75	----	----	----	2	1	7	----
North Dakota	----	6	----	----	1	----	23	----	----	----	----	----	----	----
South Dakota	----	5	----	2	----	1	44	----	4	----	----	----	7	----
Nebraska	1	1	4	7	----	3	60	----	12	----	----	----	----	----
Kansas	1	----	----	52	----	4	47	1	6	----	----	1	10	----
SOUTH ATLANTIC														
Delaware	----	----	----	1	1	----	2	----	----	----	----	----	1	----
Maryland [a]	1	----	2	7	----	8	[1] 5	1	[d] 3	----	----	2	32	----
District of Columbia	----	----	----	----	----	7	11	----	----	----	----	----	----	----
Virginia	4	----	160	17	2	48	21	6	16	----	1	3	29	1
West Virginia	4	----	----	7	----	3	27	----	12	----	----	8	13	----
North Carolina	16	----	----	7	----	----	4	2	14	----	1	2	25	----
South Carolina	8	----	4	5	1	10	3	----	4	----	----	2	1	3
Georgia	12	----	2	1	1	6	21	----	12	----	4	5	2	6
Florida	2	----	----	1	1	16	12	----	----	----	2	1	2	----

EAST SOUTH CENTRAL														
Kentucky	8			2	3	37	41	1	11	1		8	60	8
Tennessee	2	1	12	12	2	32	32	1	16			2	24	
Alabama	13		9	10	1	11	12		12		1	4	11	2
Mississippi [a]	17		5	3	1	12	28		2		1		1	
WEST SOUTH CENTRAL														
Arkansas	3		13	9	1	11	34		3		9	4	19	2
Louisiana	6		1	1		18	6					5	1	
Oklahoma	1		9	2		11	86		1		1	3	11	3
Texas	15	1	299	26		147	65	1	13		3	14	80	16
MOUNTAIN														
Montana				7			13		2			1		
Idaho	1		2	2		2	28		[d] 3					
Wyoming				2	1	1	8				1			
Colorado		10	10	9	2	9	77		1			4	15	
New Mexico	1					9	7		1			3		
Arizona	5		6	8	1	4	6					1	11	
Utah [a]				3	1	1	12						3	
Nevada														
PACIFIC														
Washington				10	1		34		8				26	
Oregon			4	17		4	17		6			1	27	
California	5	2	3	42	3	21	121		[d] 16		1	9	72	1
Total	146	34	551	499	49	741	2,701	17	272	1	27	114	1,440	
Median, 1944–48	221	21	706	543	48		1,498	18	564	1	17	111	1,798	
Year to date, 36 weeks	4,732	479	79,205	589,017	2,470	58,039	[1] 26,457	500	59,210	42	869	2,602	42,340	
Median, 1944–48	7,527	390	193,473	552,229	4,616		10,972	461	88,476	275	666	2,886	70,100	
Seasonal low week ends	(27th) July 9		(30th) July 30	(35th) Sept. 3	(37th) Sept. 18		(11th) Mar. 19		(32d) Aug. 13	(35th) Sept. 3		(11th) Mar. 19	(39th) Oct. 2	
Since seasonal low week	964		3,338	499	3,314		[1] 25,541		950	1		2,142	52,373	
Median, 1944–45 to 1948–49 [b]	1,735		3,276	543	6,120		10,709		2,181	1		2,411	97,057	

[a] Period ended earlier than Saturday.
[b] The median of the 5 preceding corresponding periods; for meningitis and whooping cough, the corresponding periods are 1943–44 to 1947–48.
[c] New York City and Philadelphia only, respectively.
[d] Including cases reported as streptococcal infection and septic sore throat.
[e] Including paratyphoid fever; currently reported separately, as follows: Indiana 1, Georgia 2, Florida 1, Alabama 1, Louisiana 4, Oklahoma 1, Texas 3, Colorado 1, New Mexico 1, California 3. Cases reported as Salmonella infection, not included in the table, were as follows: Massachusetts 2, Pennsylvania 1.
[1] Poliomyelitis: Delayed reports, Maryland, July, and August onsets, 18 cases; deductions, Michigan, 1 case each, weeks ended August 20 and 27.
Alaska: Measles 7, pneumonia 3, streptococcal throat 2.
Hawaii Territory: Measles 2, lobar pneumonia 1, poliomyelitis 1.

FOREIGN REPORTS

AUSTRALIA

Victoria State and Melbourne City—Poliomyelitis.—According to information dated September 1, 1949, the State of Victoria, Australia, is experiencing the second worst epidemic of poliomyelitis in its history. From January 1–August 31, 1949, a total of 418 cases with 27 deaths is stated to have occurred. The total number of cases reported in the State of Victoria during the year 1948 was 32. A large number of the current cases are said to be in adults. Poliomyelitis has been declared epidemic in both the metropolitan area of Melbourne and the State of Victoria.

CANADA

Provinces—Notifiable diseases—Week ended August 20, 1949.—Cases of certain notifiable diseases were reported by the Dominion Bureau of Statistics of Canada as follows:

Disease	Newfoundland	Prince Edward Island	Nova Scotia	New Brunswick	Quebec	Ontario	Manitoba	Saskatchewan	Alberta	British Columbia	Total
Chickenpox			5	2	12	18	3	15	16	27	98
Diphtheria				1	8	3					12
Dysentery:											
Amebic						1					1
Bacillary					4						4
Encephalitis, infectious						1		1	1		3
German measles					2	2		2	5	5	16
Influenza			31			4	1			1	37
Measles			15		40	18	22	40	46	69	250
Meningitis, meningococcal						1	1				2
Mumps			23		5	23	2	2	8	27	90
Poliomyelitis	1		2	7	86	128	4	5	10	9	252
Scarlet fever	1				11	5		2	5	2	26
Tuberculosis (all forms)			4	12	108	32	67	28	2	35	288
Typhoid and paratyphoid fever	3			1	6	1				3	14
Undulant fever					1	1					2
Venereal diseases:											
Gonorrhea	6		8	21	80	67	36	22	49	63	352
Syphilis			4	13	40	19	7	3	4	28	118
Whooping cough					70	28	3	4	8		113

CUBA

Habana—Notifiable diseases—4 weeks ended June 25, 1949.—Certain notifiable diseases were reported in Habana, Cuba, as follows:

Disease	Cases	Deaths	Disease	Cases	Deaths
Chickenpox	10		Smallpox	1	
Diphtheria	13	1	Tuberculosis	10	1
Malaria	1		Typhoid fever	12	1
Measles	13				

Provinces—Notifiable diseases—4 weeks ended June 25, 1949.—Cases of certain notifiable diseases were reported in the Provinces of Cuba as follows:

Disease	Pinar del Rio	Habana [1]	Matanzas	Santa Clara	Camaguey	Oriente	Total
Cancer	4	18	11	15	2	8	58
Chickenpox		11	3			4	18
Diphtheria		15	4		1		20
Leprosy		2				1	3
Malaria	4	4		1	1	7	17
Measles		20		5	6	5	36
Poliomyelitis					1	3	4
Scarlet fever			2				2
Smallpox		1					1
Tetanus				1			1
Tuberculosis	2	21	29	20	16	19	107
Typhoid fever	8	31	13	16	9	36	113
Undulant fever						3	3
Whooping cough		4	2				6

[1] Includes the city of Habana.

KOREA

Encephalitis, Japanese "B".—Information dated September 7, 1949, states that an outbreak of Japanese "B" encephalitis, not yet in epidemic form, has been reported by the health authorities in Korea. This outbreak is stated to have begun August 26, 1949, at Kaesong, and to have spread to Seoul and south of Seoul. As of September 5, cases and deaths had been reported as follows: Kaesong 110 cases, 51 deaths; other rural areas 46 cases, 17 deaths; Seoul 113 cases, 29 deaths.

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

From consular reports, international health organizations, medical officers of the Public Health Service, and other sources. The reports contained in the following tables must not be considered as complete or final as regards either the list of countries included or the figures for the particular countries for which reports are given.

CHOLERA

(Cases)

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

Place	January-June 1949	July 1949	August 1949—week ended—			
			6	13	20	27
ASIA						
Burma	229	11				
Bassein	164	11				
Moulmein	2					
Rangoon	2			[1] 1		
Ceylon:						
Trincomalee	2					
China:						
Amoy		[2] 1				
India	53,390	8,752	1,714	1,108	[3] 394	[3] 377
Allahabad	5			7		
Bombay	[1] 3	[1] 2			[1] 1	
Calcutta	[4] 4,190	321	42	43	47	63

CHOLERA—Continued

Place	January–June 1949	July 1949	August 1949—week ended—			
			6	13	20	27
ASIA—continued						
India—Continued						
Cawnpore	103	25	5	11	14	15
Cocanada		5	4	1	1	
Cuddalore	2					
Lucknow	25	7				
Madras	76	122	39	52	38	21
Masulipatam	1					
Nagpur		1	8	3		
Negapatam	26					
New Delhi	[4] 2	16				
Raj Samand	40					
Tuticorin	14					
India (French):						
Karikal	55					
Pondicherry	100					
Indochina (French):						
Annam	([5])					
Cambodia	42		3			
Cochinchina	10			1		
Pakistan	21, 893	818				
Chittagong	71	3	1			
Dacca	92				6	
Lahore	11			[1] 1	2	1
Siam (Thailand)	9					
Bangkok	8					

[1] Imported. [2] Suspected. [3] Preliminary figures. [4] Includes imported cases. [5] Correction: The 53 cases of cholera reported in Annam (Pub. Health Rep., May 27, 1949, p. 681, June 24, 1949, p. 812, July 29, 1949, p. 956) were erroneously recorded. These were cases of plague. No case of cholera has been reported in Annam this year. The plague figures have been corrected accordingly.

PLAGUE [6]

(Cases)

AFRICA						
Basutoland	40	1				
Belgian Congo	7	[1] 5		1	1	
Costermansville Province	1	1			1	
Stanleyville Province	6	[1] 4		1		
British East Africa:						
Kenya	4	1				
Tanganyika	15					
Madagascar	66	4				
Tananarive	3					
Rhodesia, Northern	2					
Union of South Africa	[2] 36	[2] 26	2	[2] 3		
ASIA						
Burma	[3] 410	7		1		
Mandalay	1					
Moulmein	[3] 6					
Rangoon	[3] 6	1		1		
China:						
Chekiang Province	7					
Wenchow	7					
Fukien Province	20					
Kiangsi Province	9					
India	24, 680	779	154	163		
Indochina (French)	88			1	2	
Annam	[4] 63				2	1
Cambodia	20			1	1	
Cochinchina	31					1
Laos	3					
Java	31	2				
Siam (Thailand)	152			3		
EUROPE						
Portugal: Azores	4					
SOUTH AMERICA						
Peru:						
Lambayeque Department	7					
Lima Department	3					
Piura Department	6					

PLAGUE—Continued

Place	January–June 1949	July 1949	August 1949—week ended—			
			6	13	20	27
SOUTH AMERICA—continued						
Venezuela:						
Aragua State	2					
OCEANIA						
Hawaii Territory: Plague infected rats [5]						

*During the period July 23–Aug. 6, 1949, 2 cases of bubonic plague were reported in the State of New Mexico in the United States—1 case in Taos County and 1 case in Sandoval County.

[1] Includes 2 cases of pneumonic plague. [2] Includes suspected cases. [3] Includes imported cases. [4] Corrected figure. (See footnote 5 in Cholera table p. 1242). [5] Plague infection has been reported in Hawaii Territory as follows: On Mar. 12, 1949, in a mass inoculation of 2 pools of tissue from 10 rats (8 and 2), taken on Maui Island; on Mar. 16, 1949, in mass inoculation of 3 pools of 29 fleas (7, 12, and 10) from rats trapped on the Island of Hawaii; on Aug. 4, 1949 in mass inoculation of 15 fleas from rats trapped on the Island of Hawaii; on Aug. 18, 1949, in a pool of 31 fleas collected from rats trapped on the Island of Hawaii.

SMALLPOX

(Cases)

(P=present)

AFRICA						
Algeria	143	17				
Angola	[1] 327					
Belgian Congo	[1] 1,035	282	67			
British East Africa:						
Kenya	24		1			
Nyasaland	926	50				
Tanganyika	374					
Uganda	33					
Cameroon (British)	10	10				
Cameroon (French)	61	3				
Dahomey	270	27		[2] 18	[3] 15	
Egypt	3					
Eritrea	1					
Ethiopia	6					
French Equatorial Africa	69	18		[2] 40		
French Guiana	1					
French West Africa: Haute Volta	108	11				
Gambia	54	4				
Gold Coast	17					
Ivory Coast	211	40		[2] 1	[3] 1	
Morocco (French)	8					
Mozambique	127	44	1			
Nigeria	6,681	128				
Niger Territory	412	9		[2] 28		
Portuguese Guinea	1					
Rhodesia:						
Northern	5			1		
Southern	276	124				
Senegal	16	1				
Sierra Leone	107					
Sudan (Anglo-Egyptian)	[1, 4] 131	39	5	3	18	
Sudan (French)	152	2		[2] 1		
Togo (French)	104	28				
Union of South Africa	353	18	[5] 3	[5] 2	[5] 1	
ASIA						
Afghanistan	87	57				
Arabia	[4] 38	[6] 3				[6] 2
Bahrein Islands	[4] 46	8	1			
Burma	[4] 1,442	[4] 53	3	12		10
Ceylon	[6] 1		[6] 1			
China	889	22				
India	54,147	4,831	656	325	[7] 107	[7] 107
India (French): **Yanaon**	1					
India (Portuguese)	205	11				
Indochina (French)	2,235	93	3	2	5	5
Iran	224	14				
Iraq	[4] 360	48	2	4	[4] 10	3
Israel	5					
Japan	117	6				

SMALLPOX—Continued

Place	January-June 1949	July 1949	August 1949—week ended—			
			6	13	20	27
ASIA—continued						
Korea (Southern)	544					
Lebanon	[4] 139					
Malay States (Federated)	43					
Manchuria		9				
Netherlands Indies:						
Java	5,523	1,774	326	415	222	
Riouw Archipelago	2					
Sumatra	[4] 94	[4] 33	9	10	8	11
Pakistan	3,201	215				
Philippine Islands:						
Mindoro Island	11					
Romblon Island	[6] 4					
Tablas Island	2					
Portuguese Timor	4					
Siam (Thailand)	37		6			2
Straits Settlements: **Singapore**	[4] 2					2
Syria	357	50	5	3	14	14
Transjordan	184	7	2			
Turkey. (See Turkey in Europe)						
EUROPE						
Belgium	1					
Germany		1				
Great Britain: England and Wales	[4] 20					
Italy	[8] 98					
Portugal	5	2				
Spain	2					
Canary Islands	6					
Turkey	89	3				
NORTH AMERICA						
Cuba: **Habana**	[4] 6					
Guatemala	4					
Mexico	44	[9] 1				
SOUTH AMERICA						
Argentina	[1] 72	[1] 28	[1] 26	[9] 1	[9] 8	[9] 20
Bolivia	35					
Brazil	[1] 73	[9] 6	[9] 2	[9] 3	[9] 4	[9] 2
Chile	[6] 2					
Colombia	[1] 1,646	[9] 159				
Ecuador	[1] 501	[1] 37				
Paraguay	[9] 2					
Peru	1,151					
Venezuela	[1] 1,306	[1] 23				
OCEANIA						
Guam	2					

[1] Includes alastrim. [2] Aug. 1-10, 1949. [3] Aug. 11-20, 1949. [4] Includes imported cases. [5] In Johannesburg. [6] Imported. [7] Preliminary figures. [8] Includes 95 cases of varioloid reported in Rome Jan. 1-June 10, 1949. [9] Alastrim.

TYPHUS FEVER*

(Cases)

(P=present)

Place	January-June 1949	July 1949	6	13	20	27
AFRICA						
Algeria	47	9				
Basutoland	7	2				
Belgian Congo	[1] 41					
British East Africa:						
Kenya	68					
Nyasaland	4					
Tanganyika		1				
Egypt	171	3	1			
Eritrea	50	12				
Ethiopia	404					
Gold Coast	1					
Libya	187	10	6			2
Madagascar: **Tananarive**	[1] 10					

TYPHUS FEVER—Continued

Place	January-June 1949	July 1949	August 1949—week ended—			
			6	13	20	27
AFRICA—continued						
Morocco (French)	14	2				
Morocco (Spanish)	2					
Sierra Leone	[1] 1					
Tunisia	57	4				
Union of South Africa	[2] 62	P	P			
ASIA						
Afghanistan	1,477	(3)				
Arabia: **Aden**	[4] 2					
Burma	4					
Ceylon: **Colombo**	[1] 4	[1] 1				
China	25	2				
India	[4] 225	4				
India (Portuguese)	19	1				
Indochina (French)	10					1
Iran	140	4				
Iraq	29	12	1	4	4	
Japan	84	9				
Korea	142					
Lebanon	[1] 1		[1] 1			
Pakistan	589					
Palestine	[5] 100					
Philippine Islands: **Manila**	[1] 1					
Straits Settlements: **Singapore**	[6] 2					
Syria	20	1	1			
Transjordan	53	5			1	
Turkey. (See Turkey in Europe.)						
EUROPE						
Belgium	[4] 5					
Bulgaria	345	19				
Czechoslovakia	20			24		
France	4					
Great Britain: Island of Malta	[1] 4	[1] 1		[1] 1		
Greece	[2] 31	[2] 2		[1] 2		
Hungary	20					
Italy	[2] 29		4			
Sicily	13					
Poland	228	15	9			
Portugal	5					
Rumania	417					
Spain	3					
Turkey	122	14	4	2	2	5
Yugoslavia	156	3	5			
NORTH AMERICA						
Bahama Islands: **Nassau**	[1] 1					
Costa Rica [1]	22	1			2	2
Cuba [1]	3					
Guatemala	27					
Jamaica [1]	10	6		1		
Mexico [2]	91	40	7	12	2	4
Panama Canal Zone [1]	6					
Puerto Rico [1]	20	7	1	3		
SOUTH AMERICA						
Argentina [1]	1					
Bolivia	53					
Brazil	2					
Chile [2]	124	18	3	7	1	
Colombia [2]	1,444	223				
Curacao [1]	5					
Ecuador [2]	170	43				
Peru	663					
Venezuela [1]	42	17				
OCEANIA						
Australia [1]	82	6	2			
Hawaii Territory [1]	4	1	1			

*Reports from some areas are probably murine type, while others include both murine and louse-borne types.

[1] Murine type. [2] Includes murine type. [3] An epidemic of louse-borne typhus fever was reported in Afghanistan on July 22, 1949. [4] Includes imported cases. [5] Approximate number reported in outbreak in villages in Hebron and Bethlehem districts in February 1949. [6] One case type unspecified, 1 case murine type.

YELLOW FEVER

(C=cases; D=deaths)

Place		January—June 1949	July 1949	August 1949—week—ended— 6	13	20	27
AFRICA							
Belgian Congo:							
Stanleyville Province	D	5					
French Equatorial Africa:							
Bangui	D					1	
Gold Coast	C	[1] 4	[1] 10	[2] 1	[2] 2	2	[1] 3
Akwatia	C		4		[2] 1		
Brim District	C	[2] 2	1				
Komenda Village [3]	D	1					
Nkwanta Dunkwa Area	D		1				
Oda Area:							
Bawdua	C		[2] 1	[2] 1			
Esuboni	C		[2] 1				[2] 1
Oseikrome Village	D	1					
Winneba Area:							
Apam	D					1	
Akukuom	D					1	
Nyakrom	C		[4] 2		[2, 4] 1		[5] 2
Nigeria:							
Lagos	D	2					
NORTH AMERICA							
Panama:							
Colon Province	D				1		[6] 1
Pacora	C	[7] 8					
SOUTH AMERICA							
Brazil:							
Amazonas State	D	1					
Para State	D	3					
Ecuador:							
Napo Pastaza Province	D	1					
Peru:							
Cuzco Department	D	2					
San Martin Department	D	1					

[1] Includes suspected cases. [2] Suspected. [3] Near seaport of Sekondi. [4] Fatal. [5] 1 suspected case, 1 fatal confirmed case. [6] Case contracted in same jungle area of the Province of Colon as the one reported on August 7, 1949 (see Public Health Reports for September 2, 1949, p. 1132). Death occurred on August 21, 1949, in Saint Tomas Hospital in Panama City. [7] Reported Jan. 15, 1949. Date of occurrence Nov. 11–Dec. 30, 1948. 5 cases, all fatal, confirmed; 3 suspected cases.

DEATHS DURING WEEK ENDED SEPT. 3, 1949

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

	Week ended Sept. 3, 1949	Corresponding week, 1948
Data for 94 large cities of the United States:		
Total deaths	8,470	10,579
Median for 3 prior years	7,965	
Total deaths, first 35 weeks of year	324,344	327,305
Deaths under 1 year of age	672	741
Median for 3 prior years	682	
Deaths under 1 year of age, first 35 weeks of year	22,955	23,574
Data from industrial insurance companies:		
Policies in force	70,196,573	70,926,141
Number of death claims	11,630	11,333
Death claims for 1,000 policies in force, annual rate	8.6	8.4
Death claims per 1,000 policies, first 35 weeks of year, annual rate	9.3	9.5