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DOMESTIC WATER AND DENTAL CARIES [1] [2]

IV. Effect of Increasing the Fluoride Content of a Common Water Supply on the *Lactobacillus acidophilus* Counts of the Saliva

(Preliminary Report)

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During the past few years epidemiological studies have pointed out an inverse correlation between the amount of dental caries and the presence of fluorides in the domestic water supplies (*1*, *2*, *3*). Recently Dean, Jay, Arnold, and Elvove (*4*) presented evidence indicating that teeth calcified on a high fluoride water seemingly retained an increased resistance to dental caries even though they had been under the influence of a fluoride-free [3] water for 12 years immediately preceding the examination. These studies and others have indicated the possibility of partially controlling dental caries through the public water supply. This possibility was further enhanced by the findings in the study of 8 suburban Chicago communities (*3*). For instance, at Aurora (Ill.) the dental caries inhibitory factor, presumably present in the water and probably fluoride, was operative at levels (1.2 p. p. m.[4] of F) where mottled enamel per se was of minimal public health and no esthetic significance. At present, however,

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[1] From the Division of Infectious Diseases with the cooperation of the Division of Chemistry, National Institute of Health.

[2] Preceding papers in this series are:

Dean, H. T., Jay, P., Arnold, F. A., Jr., and Elvove, E.: Domestic water and dental caries. I. A dental caries study, including *L. acidophilus* estimations, of a population severely affected by mottled enamel and which for the past 12 years has used a fluoride-free water. Pub. Health Rep. 56:365-381 (1941).

Dean, H. T., Jay, P., Arnold, F. A., Jr., and Elvove, E.: Domestic water and dental caries. II. A study of 2,832 white children, aged 12-14 years, of 8 suburban Chicago communities, including *Lactobacillus acidophilus* studies of 1,761 children. Pub. Health Rep., 56:761-792 (1941).

McClure, F. J.: Domestic water and dental caries. III. Fluorine in human saliva. Am. J. Dis. Child., 62:512 (1941).

[3] The term "fluoride-free" is used in this report for waters containing less than 0.2 p. p. m. F—experimental error of F determination is 0.1 p. p. m.

[4] P. p m.=parts per million.

there is no direct evidence demonstrating whether or not the addition of fluorine to a fluoride-free common water supply will decrease the incidence of dental caries in the community. The epidemiological evidence seemingly justifies the assumption that dental caries activity in humans is markedly inhibited by drinking waters of fluoride concentrations no higher than about the minimal threshold of mottled enamel. Actual field experiments, however, are necessary to determine what age groups might thus benefit, how long ingestion must continue before dental caries activity decreases, and numerous other aspects of this phenomenon as it operates in human populations. An opportunity to study a few of these aspects recently presented itself. This paper is a preliminary report of what may be a long-term study.

Garrettsville, a small village in the northeastern section of Ohio, recently changed its public water supply. Prior to November 1939, the village water supply was obtained principally from three wells, the fluoride (F) content of which was about 0.1 p. p. m. In November 1939, a new well was put into service; the water from this well contains about 1.7 p. p. m. of fluoride (F). Mixture of the water from these wells resulted in a domestic water of about 0.7 p. p. m. F. An opportunity was therefore presented to study children whose domestic water was changed from one practically free of fluorides to a water containing this element in a range previously found (*3*) beneficial to children using such waters throughout life.

DESCRIPTION AND MINERAL COMPOSITION OF THE GARRETTSVILLE WATER SUPPLY

From 1924 to 1939 the water supply for the village was obtained from three drilled wells (Nos. 2, 3, 4), each 8 inches in diameter and approximately 50 feet deep, located about 1.5 miles northeast of the village. The well supply was supplemented during the spring months by water from a spring located in the same general area. This supply was abandoned in 1937. In November 1939 a new well (No. 1), 8 inches in diameter and 210 feet deep, was placed in service. Because of the fluoride content of the water from well No. 1, the water from this source is mixed with water from two of the other wells. On the average day, well No. 1 is pumped 8 hours and delivers approximately 65 gallons per minute and wells Nos. 2 and 3 are pumped 10 hours and deliver 45 to 50 gallons per minute. Since the beginning of the study, well No. 4 has been used only occasionally and at present is not used at all.

The water obtained from these wells is treated in an iron-removal plant. This plant comprises an aerator, a settling tank divided into four units operating in series, and two gravity type rapid sand filters. Water is pumped directly from the wells to the treatment plant

from where it flows by gravity to the clear well. High service pumping equipment pumps water directly from the clear well to the storage reservoir. All the water supplied to the town comes from this one storage reservoir. The present water consumption of the village ranges from 70,000 to 105,000 gallons daily.

TABLE 1.—*Analyses of the waters used at Garrettsville, Ohio*

[In parts per million [1]]

	Well No. 1	Well No. 2	Well No. 3	Domestic tap
Residue on evaporation	858.4	156.0	165.6	406.4
Loss on ignition	36.8	36.0	36.0	28.0
Fixed residue	821.6	120.0	129.6	378.4
Silica (SiO_3)	18.4	18.0	16.0	20.0
Iron (Fe)	.04	0	.04	.05
Aluminum (Al)	0	0	0	0
Calcium (Ca)	11.4	24.3	26.9	15.7
Magnesium (Mg)	1.4	7.4	8.9	7.4
Sodium and potassium (calculated as Na)	332.2	7.4	7.0	130.2
Carbonate (CO_3)			4.8	
Bicarbonate (HCO_3)	529.5	89.0	68.3	247.6
Sulfate (SO_4)	6.6	23.9	42.8	17.3
Nitrate (NO_3)	0	5.3	.5	2.1
Chloride (Cl)	220.5	2.5	2.0	85.0
Phosphate (PO_4)	0	0	0	0
Fluoride (F)	1.7	[2].1	[3].2	[3].8

[1] The samples from wells Nos. 1 and 2 were received Jan. 15, 1940; those from well No. 3 and the domestic tap were received Feb. 27, 1940. Assistant Chemist C. G. Remsburg carried out the determinations other than fluoride, using mostly the methods given in the Standard Methods of Water Analysis of the American Public Health Association. The fluoride determinations were made by Senior Chemist E. Elvove using the colorimetric Zirconium-Alizarin method (Pub. Health Rep., 48:1219 (1933)).

[2] 3 other monthly samples show 0.1 p. p. m. F.

[3] The mean fluoride (F) content of 12 monthly samples of the domestic tap was 0.7 p. p. m. (range 0.5–0.8).

PLAN OF STUDY

The present study group is composed of white public school children living within the corporate limits of the village and using the public water supply for all domestic purposes. Only children in the fourth to tenth grades were included in the study. Their ages ranged from 9 to 17 years at the beginning of the study, the average being 13 years. The original group included a total of 132 children, but due to absentees, migrations, and other uncontrollable factors, only 109 were followed for the complete year of study. All 109 children were living in Garrettsville prior to the change of water.

BACTERIOLOGICAL STUDY AND RESULTS

The technique followed in obtaining *L. acidophilus* counts was similar to that used in other studies in this series (*2*, *3*, *4*). Paraffin stimulated saliva samples were collected between 10:30 and 11:30 a. m. The time allowed for collection of saliva was approximately 5 minutes in all cases. The dilution used throughout was 1 cc. saliva to 4 cc. broth; 0.1 cc. of the broth and saliva mixture was placed on tomato juice agar, pH 5.0. All samples were collected at the school and were taken that same day to the Institute of Pathology,

Western Reserve University, Cleveland, Ohio, where they were plated and incubated. During the year 1940–41, eight separate saliva samples were collected from each child (two in May, three in November, three in April) and counts determined. The results of these eight examinations are shown in table 2.

TABLE 2.—*Summary of the distribution of oral L. acidophilus in 8 individual saliva samples from a group of 109 school children, Garrettsville, Ohio, during a 1-year period*

Date of collection	Distribution of children according to the estimated number of oral *L. acidophilus* per cubic centimeter of saliva								
	Negative	Less than 100	100 to 1,000	1,000 to 3,000	3,000 to 12,000	12,000 to 21,000	21,000 to 30,000	30,000 and over	Total
				NUMBER					
May 14, 1940	6	11	3	3	8	12	8	58	109
May 15, 1940	6	12	6	3	6	11	8	57	109
Nov. 12, 1940	11	4	10	2	9	16	4	52	108
Nov. 13, 1940	11	3	10	3	9	5	6	61	108
Nov. 14, 1940	13	4	5	4	6	5	5	63	105
April 14, 1941	8	7	2	0	8	7	5	68	105
April 15, 1941	9	3	1	1	13	4	3	72	106
April 16, 1941	11	1	1	2	10	6	4	71	106
				PERCENT					
May 14, 1940	5.5	10.1	2.8	2.8	7.3	11.0	7.3	53.2	
May 15, 1940	5.5	11.0	5.5	2.8	5.5	10.1	7.3	52.3	
Nov. 12, 1940	10.2	3.7	9.3	1.9	8.3	14.8	3.7	48.1	
Nov. 13, 1940	10.2	2.8	9.3	2.8	8.3	4.6	5.6	56.5	
Nov. 14, 1940	12.4	3.8	4.8	3.8	5.7	4.8	4.8	60.0	
April 14, 1941	7.6	6.7	1.9	.0	7.6	6.7	4.8	64.8	
April 15, 1941	8.5	2.8	.9	.9	12.3	3.8	2.8	67.9	
April 16, 1941	10.4	.9	.9	1.9	9.4	5.7	3.8	67.0	

Previous studies in this series (*2*, *3*, *4*) have indicated a correlation between the dental caries experience of the group and the *L. acidophilus* counts. In these studies group differences were found principally in the percentages of low counts (negative and less than 100) and high counts (30,000 and over). The results of one of these studies (*3*) present evidence that there is a group difference in *L. acidophilus* counts of saliva from children who were born and reared in a community where the water contained 0.5 p. p. m. fluorides (F) (Elgin, Ill.) when compared with counts from children who were continuously exposed to a fluoride-free water (Waukegan, Ill.). In order to compare the bacteriological results of the present study obtained at three different periods of the year with the results obtained at Elgin and Waukegan, Ill., the percentage of low and high *L. acidophilus* counts[5] are shown graphically in figure 1.

[5] In order to make the data comparable, the percentages shown for the low and high *L. acidophilus* counts at Garrettsville are those obtained on the first day of each study period.

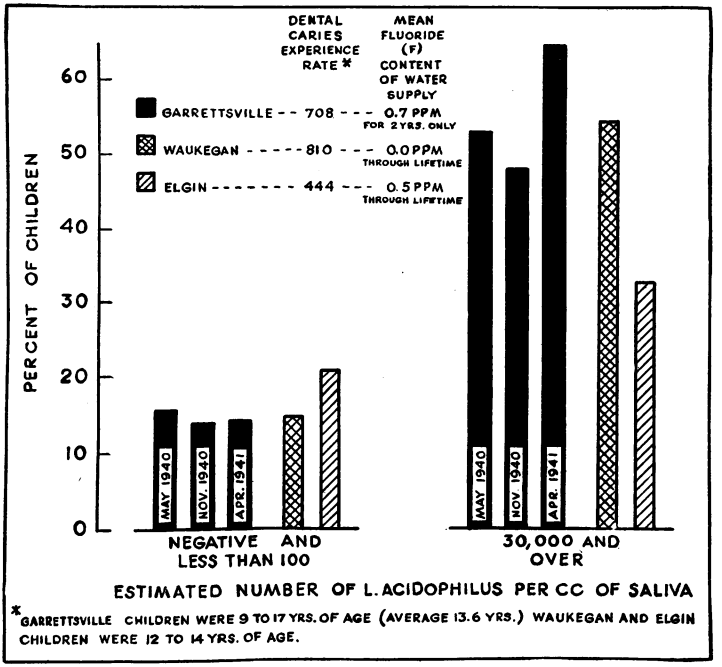

FIGURE 1.—A comparison of the low and high *L. acidophilus* counts obtained at Garrettsville, Ohio, with counts obtained at Waukegan and Elgin, Ill. The results shown for Garrettsville are those obtained on the first day of each visit during the study year.

CLINICAL EXAMINATION AND RESULTS

In order to have some measure of the prevalence of dental caries in this group of children, a complete dental examination was made in December 1940. This examination was made by use of mouth mirror and explorer in a well-lighted schoolroom with the child seated and facing a window. The criteria for making a diagnosis of dental caries were the same as used in previous studies (*2*, *3*). In computing the dental caries experience no tooth was counted more than once, even though it had a filling on one surface and an untreated lesion on another.[6] The principal findings of the clinical examination are shown in table 3.

DISCUSSION

The results of the single clinical examination given in table 3 record the dental caries experience of the permanent teeth of the study group up to the time of examination. Considering the average age of these children when examined (13.6 years) and their caries experience rate (708), it seems logical to assume that they have had a somewhat similar caries experience to that observed in children born and reared

[6] Any tooth showing evidence of a filling was classified as a filled tooth.

in communities in which the water is practically free of fluorides (3).[7] The low incidence of dental fluorosis (1.8 percent) is similar to the sporadic instances of "very mild" mottled enamel found in children who have calcified their teeth on water which is practically free of fluorine. In view of this evidence one might assume that up to the time of change in water supply this group of children at Garrettsville had an oral environment comparable to children born and reared in communities with practically fluoride-free waters.

TABLE 3.—*Summary of the clinical findings observed in an examination of 107 white school children, aged 9 to 17 years, at Garrettsville, Ohio*

Dental caries experience, permanent teeth						Macroscopic evidence of endemic dental fluorosis		
Teeth filled (past dental caries)	Teeth with untreated dental caries	Teeth, extraction indicated	Teeth, missing	Total	Proximal surfaces 4 superior incisors	Absent		Present
						Normal	Questionable	Very mild
(a)	(b)	(c)	(d)	(a+b+c+d)				
Number per 100 children examined					Number per 100 surfaces	Percent		Percent
361	284	16.8	46.7	708	12.7	83.2	15.0	1.8

In order to obtain direct evidence of a change in caries activity in a study of this nature, especially with such limited numbers, it will be necessary to make repeated clinical examinations. Such a plan necessarily requires sufficient time to elapse between examinations so that comparisons can be made on the basis of similar age groups with like exposure to risk of caries under the changed environment (new water supply). A presumptive indication of any significant change in caries activity of the study group might be gained by use of *L. acidophilus* counts as a measure of caries activity.[8] Previous studies in this series indicate that a negative correlation exists between the caries activity of a group population, as measured by the percentage distribution of *L. acidophilus* counts, and the presence of fluorides in the public water supplies. Attention may be called, however, to the fact that in the studies cited only children 12 to 14 years of age, *who had been continuously exposed to the respective waters throughout life,* were included in the study groups.

In view of the clinical results (as to the amount of dental caries)

[7] The dental caries experience rate of 1,008 children examined in Evanston, Oak Park, and Waukegan, Ill., in the study cited was 746. However, it should be noted that the range of ages in the present study (9 to 17 years) is different from that of the children referred to in these 3 cities.

[8] For more complete discussion of the relationship of *L. acidophilus* and dental caries activity, reference may be made to Dental Science and Dental Art, Chapter 10 (5).

in the present study group, and the bacteriological results in previous studies, one might expect that had there been an immediate beneficial change in caries activity between the date of change of water supply (November 1939) up to the beginning of the study (May 1940) the *L. acidophilus* counts would be changed. In other words, there would have been an increase in the number of low counts (0 or less than 100) and a decrease in the high counts (30,000 and over). However, the counts obtained in May 1940 indicate that no such change had occurred. The *L. acidophilus* counts at the beginning of the study would indicate that the caries activity of the group was similar to that found at Waukegan, Ill., (*3*) where the children studied had been continuously exposed to a fluoride-free water and had a similar high dental caries experience rate.

If a decrease in caries activity of the study group occurred during the year, the bacteriological results might be expected to have given some indication of this change. Had there been an increase in the number of low counts or a significant decrease in the number of high counts, a beneficial change in caries activity might have been assumed. This bacteriological change did not occur. If any change in caries activity might be inferred from the minor changes in the bacteriological counts, it would suggest a change toward a higher percentage of the children having active dental caries. According to the results shown in table 2 there was an average of 16.1 percent low counts in May 1940, and 12.3 percent low counts in April 1941; however, at present these changes cannot be considered significant.

The results obtained so far in this study might be interpreted as showing that increasing the fluoride content of a public water supply from 0.1 to 0.7 p. p. m. F will have little influence on dental caries activity for at least the first 2 years. In comparing the results of this study with previous studies in this series, one might infer that it is necessary that the individual be born and reared in a community having water of this approximate fluoride content in order to obtain any beneficial effects. It should be noted, however, that the group of children in Garrettsville have been using this new water supply for a comparatively short time and it is possible that the *L. acidophilus* counts obtained represent caries activity in lesions which started prior to the change in water.

SUMMARY

There are presented preliminary results obtained in a study of 109 public school children who have been exposed for about 2 years to a domestic water which has been increased in fluoride (F) content from about 0.1 to 0.7 p. p. m.

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A single clinical examination indicated that the dental caries experience rate of these children was similar to the rates reported in previous studies on children who were born and reared in communities where the domestic water is practically free of fluorides.

There is no indication according to the percentage distribution that the increased fluoride content of the water supply has as yet influenced the *L. acidophilus* counts. Such results might indicate that at least for the first 2 years increasing the fluoride (F) content of a public water supply from 0.1 p. p. m. to approximately 0.7 p. p. m. would result in little, if any, decrease in caries activity of children whose teeth had calcified and had been exposed for a number of years to a water practically free of fluorides. However, the relatively unchanged *L. acidophilus* counts in this group may be the result of dental caries activity in lesions which had started prior to the change of water and were still active, and subsequent clinical examinations may possibly show that fewer new carious lesions have developed since the introduction of the new water supply.

ACKNOWLEDGMENTS

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HOUSING OF HEALTH DEPARTMENTS [1]

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As chief of the States Relations Division of the United States Public Health Service, it is my duty as well as my privilege to visit many local health departments throughout the country. From these visits I derive valuable information regarding the status of health organization at the all-important local level. It is gratifying to be able to report that the zeal and devotion to duty frequently displayed by the guardians of community health cannot be praised too highly.

There is, however, one circumstance which never fails to astonish and depress me whenever I undertake a trip into the field—that is, the unsuitable, and often deplorable, condition of the quarters in which many local health departments are housed.

Upon entering a community, I proceed directly to the basement of the city hall, or to the basement of the courthouse if the visit is being made to a county health department. This is done with reasonable assurance that the health department will be found there. Occasionally my assumption will be wrong—the health department will be in the attic instead of in the basement. If it is not in either place I am at a loss because more likely than not it is situated in some out-of-the-way alley. Extended inquiries may be necessary to find someone who will attempt to direct me. Frequently I cannot follow the instructions, and am reduced to asking my informant if he will take me there personally.

Upon arriving at the health department, what do I find? If the quarters are rented, I descend—or climb—a stairway which is a hazard to life and limb, and enter a structure which meets no standard building code requirements. If the unit is in a courthouse or city hall, the stairway and building are probably substantial enough, but the entrance is apt to be equally forbidding. A motley fraternity of loafers sprawl or loiter persistently about the entrance, subjecting every client or visitor to idle but thorough scrutiny. In the corridors, inadequate lighting mercifully obscures the walls, floor, and woodwork which are spattered with tobacco juice and adorned with the jackknife carvings of a past generation.

Although the department's own quarters may present a more sanitary aspect, they were obviously not designed for a health unit. There is not enough space for desk work, to say nothing of a clinic and other essential accommodations. Ingenious partitioning devices and curtains may be employed to achieve a modicum of privacy. The furniture is a collection of "cast offs" assembled from heaven

[1] From the States Relations Division.

knows where. Being a person of some weight I often have trouble selecting a chair that looks substantial enough to hold me.

The foregoing is a harsh indictment. Lest I be considered guilty of exaggeration I will cite specific but anonymous cases. The following paragraphs contain authentic, and I may even say typical, descriptions of local health units which I have recently visited.

In one of the larger cities of the South the health unit is housed in an ancient structure formerly used as a school. The building is regarded as a fine old antique, and so, apparently, is the health department. When the school authorities abandoned the building because it was so old and dilapidated, it was placed at the disposal of the health department. No questions were asked regarding its suitability. Indeed, general opinion held that the department was now most bountifully provided for. It has an entire building for itself—an almost unheard of stroke of good fortune!

Lacking regular equipment, the health officer put his inventive talents to work and fashioned a collection of gadgets, deriving, it would seem, many of his concepts from the drawings of a well-known cartoonist. As he exhibited these makeshift wonders, which only he knew how to operate, he manifested great pride in his achievement.

In another southern community the health unit is lodged in the courthouse basement together with the county jail and the only public toilet facilities in the city. This town is now a haven for thousands of soldiers on leave and it is impossible to keep the basement orderly and clean. A dark and dreary clinic waiting room, fitted with rows of old theater seats, is flanked by the cells in which prisoners are kept.

Some years ago, while stationed in Missouri, I was instrumental in arranging for the location of two county health departments in buildings prior to their erection. One department was fairly well provided for in the county courthouse, the other in a county hospital. Here at last was evidence of enlightened policy, and I felt as if I were being granted a glimpse into the future. Recently, however, I visited these scenes of my former triumphs, only to be gravely disappointed. Both units had fared badly. In the case of one, the prosecuting attorney had decided that the rooms it occupied would exactly suit his purposes. Accordingly, the health department had been moved into the basement. It did not, however, stay there long; soon it was crowded out to allow space for the county records. The unit located in the county hospital had retained only one room of its original suite on the first floor, and most of its furniture had been strung out along the hallway leading to the basement where the clinics are now located.

Such successive pillar-to-post treatment has been the lot of many

local health units. There is a health department which uses the jury room of the courthouse. When a jury files in to deliberate, the health department simply stops whatever it is doing and gets out. An attempt is made to maintain a full schedule of field work during the time the jury is in session. I recall a Virginia city with more than 30,000 population where the health department has taken refuge on the mezzanine of the armory. Clinic patients use the sloping gallery as a waiting room. There is a unit in North Carolina which has been crowded out of the city hall proper and onto the porch. The clinic of this unit is in the basement where the city formerly locked up its drunks. Now the drunks are housed in new and better quarters, and the space has graciously been made available to the health department.

There seems to be an unremitting fate which consigns health officers and health workers to basements. They even go to school below ground level. One of the Nation's foremost schools of public health has recently been given space in a new and well constructed university building—in the basement. As usual the basement is crowded—much more so than the upper floors.

One health department visited in New Jersey is lodged in a weather-beaten shack set on poles. The wind whistles up through the cracks in the single layer of board flooring. There is no running water possible because pipes would freeze if they were installed. I am reasonably certain that the unit would be without toilet facilities if a public-spirited group of citizens had not supplied the materials, and WPA the labor for a pit privy in the backyard.

Another local department in a southeastern city with more than 50,000 population has its quarters in various parts of the municipal market center. In summer the odors emanating from the market stalls are particularly distressing. All day long the place is in a tumult with the shouting of hucksters and the cackling of poultry confined in crates piled high on the sidewalk.

A local health unit in a southwestern State has succeeded in getting quarters that are quite satisfactory when judged solely by the appearance of the interior. But the building entrance and the immediate surroundings are like something out of a bad dream. On one side is a combination "juke joint," grill, and pinball emporium; on the other a produce warehouse. From the left comes the odor of frying hamburger and onions; from the right, the stench of semi-decayed vegetable matter. A raucous and constant din issues from the juke box next door. Upon my first visit, as if the combination of odors and sounds was not enough to discourage me, a large rat scurried across my path when I approached the building. Incidentally, the town has been a focus of endemic typhus fever for several years.

I have in mind another local unit which has been crowded into a temporary frame structure in a downtown alleyway. The windows look out at the overflowing garbage pails and miscellaneous rubbish discarded by a square block of restaurants and stores.

It is not pleasant to dwell at length on scenes such as these. But I have purposely done so to emphasize one fact: *The imagination can scarcely conceive of conditions worse than those found in the headquarters of the very agencies which ought to set an example of civic cleanliness and decency.* It should be pointed out that the health departments whose quarters are described above are full-time, professionally staffed organizations, not part-time units from which a certain amount of laxity might be expected.

What is needed to correct this disgraceful state of affairs? The answer usually given is, "money." Yet this is not the whole truth; there are certain fundamentals even closer to the heart of the matter. If local health departments have not been granted funds to operate according to twentieth-century standards, the fault lies partly with the departments themselves. A probability is that they have not asked for money, or that they have not been aggressive enough in pressing their claims; somehow health departments seem to derive great satisfaction out of being considered and treated as martyrs. Or, probably their programs do not meet community needs. No public agency can expect public support unless (1) it has convinced the community that it is doing a really important and worth while job, and unless (2) it commands respect for the way it is doing it.

Many local health departments have too narrow a conception of their functions and duties. They routinely perform the old-line tasks such as inspection, abatement of nuisances, enforcement of quarantine, and other activities handed down from the early days of public health organization. If this is all a health department is prepared to do, a little desk space is all it needs, and the location of such desk space is not a matter of great importance. On the other hand, if a health department is concerned with the total health problem of the community and is actually doing something about it—if it is operating clinics, maintaining nursing services, giving real protection with regard to water, milk, and food, assisting in civilian defense, and carrying on a health education program which reaches the people—then it has a right to expect consideration from the community. Moreover, it usually gets it.

Usually—but not always. This brings us to the second point—commanding the respect of the public. A health department may actually be making a heroic effort to provide a complete set of modern services. Nevertheless, if it is miserably housed and equipped, if it is relegated to a dark basement or drafty auditorium, then it is

not only hindered in the performance of its work but it fails to get the respect its efforts should command. Unstinting public support is engendered not only by recognition of services performed, but by the manner in which they are performed. Call this salesmanship, or even showmanship, but the truth is that it builds respect and recognition. The end result is that the health department is better able to discharge its obligation to the community.

Financial support for a local health department, however, need not come entirely from local sources. Since 1935, the Federal Government has encouraged the development and maintenance of local health agencies by means of substantial grants-in-aid to the States. Moreover, prior to the present emergency, the construction of health centers was possible through the Work Projects Administration and grants and loans administered by the Public Works Administration. Local health agencies largely neglected to take advantage of this aid, and now the opportunity has been lost. Here again the lack of prestige in the community was undoubtedly reflected. In making applications for grants, the city fathers or county officials were reluctant to consider the plight of that stepchild in the official family, the health department.

In the summer of 1941, Congress appropriated $150,000,000 for defense public works, including health centers. Applications for many more projects than could be undertaken with this sum were quickly submitted, but of the 3,725 applications received prior to January 31, 1942, only 157 were for health centers and clinics. As of February 2, 1942, 52 applications had been approved calling for the construction of centers and clinics involving a total estimated cost of approximately $3,000,000.

In January 1942, an additional sum of $150,000,000 in Federal funds was made available for the provision of community facilities vital to the war effort.

The construction of new buildings, except as defense projects, will undoubtedly be difficult or impossible for some time to come. Materials will be under priority restrictions and labor will be scarce. Nevertheless, a community which wants proper quarters for its health department need not wait until conditions permit the erection of an imposing new building. Quarters far more satisfactory than those now occupied by most health units can be rented, as they are for other municipal and county agencies. Minor repairs can be made when necessary, and decent furniture can be provided. At the very least, soap and water can be applied to remove accumulated grime and filth.

The next great opportunity for the construction of health centers on a large scale may come after the war is over. It is generally be-

lieved that a comprehensive public works program will be necessary in the post-war era to absorb the shock of anticipated unemployment. A "public works reserve" is being developed by the Federal Works Agency and the National Resources Planning Board which are now surveying communities and preparing lists of suitable projects. If local health departments wish to benefit from this contemplated program, they should begin now to consider their needs and problems, and to prepare the ground for suitable action.

Any health center constructed now or in the future should be planned so as to conform to the conception of what a modern health center should be. A well-planned health center requires more than a certain amount of ground space and four walls enclosing floor area partitioned into a number of rooms. If health centers could be planned in this way, it would only be necessary to install a prefabricated house, call the living room the clinic, the dining room the clerical office, and so on. Proper planning, in fact, requires specialized knowledge and consideration of many closely interrelated factors such as function, form, engineering, and local social and economic conditions.

For the best results, the services of specially trained architects should be utilized, at least in a consulting capacity.[2] Nevertheless, it is appropriate to outline here some of the outstanding and less technical points to be considered in judicious planning.

The selection of a suitable site is important. The health center should be situated away from the main business area, but in a place which is fairly accessible. Ground should be allowed for possible future expansion. Adequate vehicular parking space should be available. Preferably, the center should be in a *separate* building; it should not be located in a city hall, courthouse, school building, or welfare center. Those who argue for placing health departments in schools say that such an arrangement is ideal for the promotion of child health. Child health work, however, is only one of the necessary activities of a local health department. It must also carry on venereal disease control, tuberculosis work, and other activities for which a school building is certainly not a desirable location. City halls and courthouses are unsuitable because the work of the health department differs in all essential respects from that of other governmental units. Basic equipment bears no resemblance to that employed by other agencies, nor are techniques at all related. The same considerations rule out the welfare center. Moreover, the relief group constitutes only a small part of the clientele of a health department.

[2] The services of a hospital and health center architectural planning unit are now available to local health agencies upon request to the United States Public Health Service. This unit has prepared sketches and floor plans of suggested types of health centers for communities of various sizes. The plans are offered as suggestions only, and are not intended to supplant the working drawings of local architects who are familiar with local conditions and needs.

All these factors aside, sharing space with other agencies, as we have seen, usually means that the health department will eventually find itself in the basement.

An ideal arrangement is to establish the health center on the grounds of a publicly owned hospital; the next best is to locate it near a nonpublicly owned hospital organized to meet community needs. In this way the health department can utilize the hospital equipment and clinical staff, thus providing better service at lower cost. I would caution against a health center being placed in the hospital building itself unless the structural arrangement is such that it precludes subsequent reassignment of the space for hospital use.

Two main considerations govern the type of architecture. First, the health center should be planned on a functional basis, that is, its form and construction must be rigidly adapted to the uses to which it is to be put. Secondly, the building must be pleasing to the eye. A permanent, fireproof type of construction is preferable to a frame building and not much more expensive. When the expense of the necessary installations such as the water system, heating, lighting, and ventilating equipment—all of which are as necessary in a frame structure as in a fireproof one—is included, the difference in total cost becomes proportionately even less.

The interior of the building should be planned with efficient, coordinated function of all units in mind. Special attention should be given to the "front of the house," the entrance, foyer, reception and waiting room, and other parts of the building where there is contact with the public. All furniture and appointments should be attractive and designed for comfort and use. Adequate provision should be made for consultation rooms, clinics, X-ray and other equipment, laboratory diagnosis, and the maintenance of vital records. There should be an auditorium where lectures, motion pictures, and classes can be held, and space should be allowed for effective display of educational material. Appropriate landscaping and care of the lawn add to the attractiveness of both grounds and building.

An example of a health center in which these features are incorporated is shown in the accompanying illustrations. Figure 1 shows the exterior of a suggested type of health center for a town with 30,000–60,000 population. Figure 2 shows the floor plan of the same center.

A health department housed in such headquarters and properly administered will be a vital force in the community. It will establish itself in the people's hearts and minds as the focal point from which community health activities proceed. Under such circumstances, the health center becomes in fact what in theory it should be, a worthy monument to the science which combats human illness and promotes longer, happier, and more useful living.

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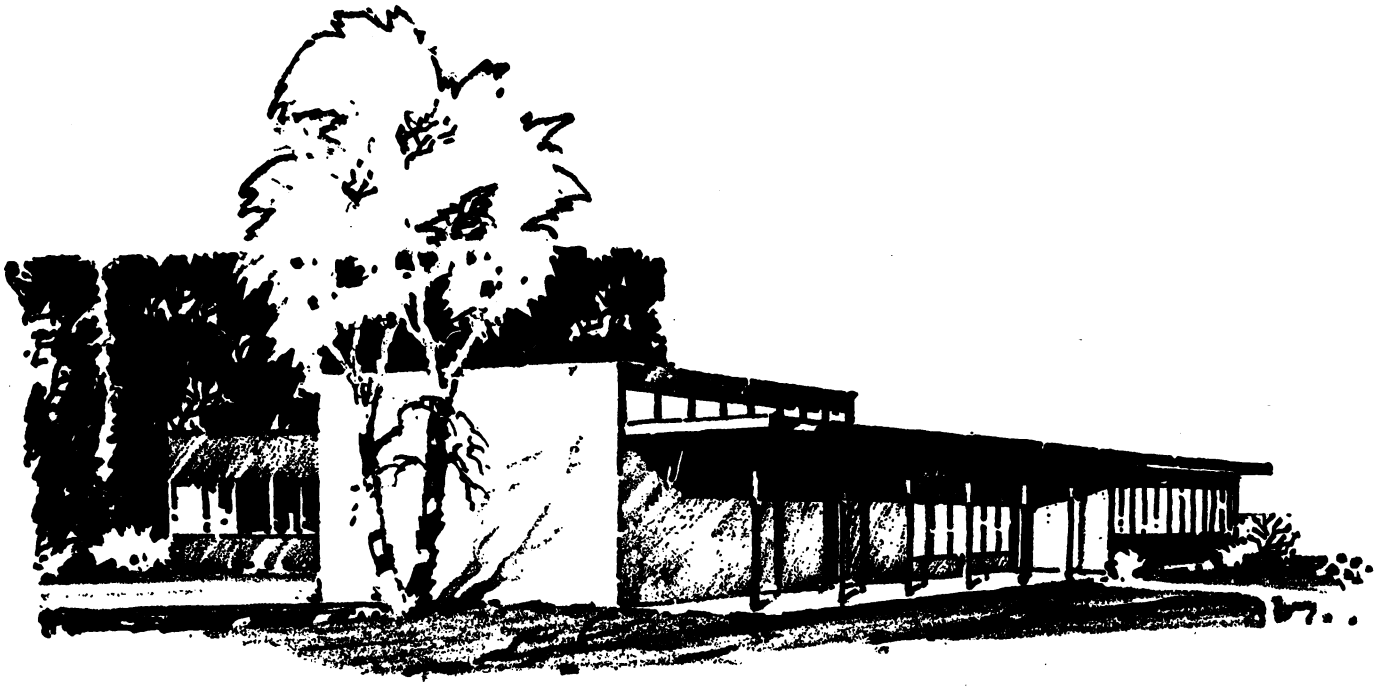

FIGURE 1.—Exterior drawing of a suggested type of health center for a community with 30,000–60,000 population.

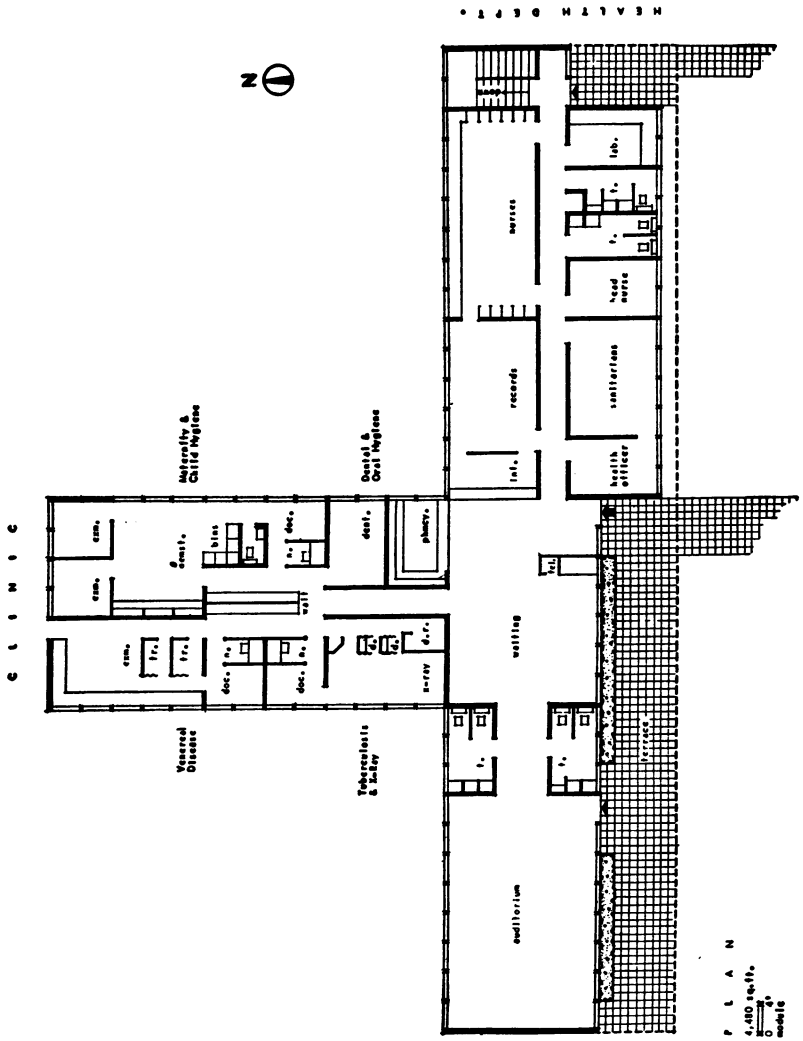

FIGURE 2.—Floor plan of the health center shown in figure 1.

THE HISTOPATHOLOGY OF EXPERIMENTAL "Q" FEVER IN MICE [1]

By THEODORE L. PERRIN, *Passed Assistant Surgeon*, and IDA A. BENGTSON, *Senior Bacteriologist, United States Public Health Service*

"Q" fever, a new disease entity, was described by Derrick (*1*) in 1937. Extensive studies on the clinical, epidemiological, bacteriologic, and serologic aspects have been published. However, only two reports dealing with the histopathology of the disease have been published.[2] Burnet and Freeman (*2*) briefly described the histologic appearance of livers and spleens from infected mice, and Lillie, Perrin, and Armstrong (*3*) reported on the histopathology of pneumonitis due to the virus of "Q" fever in man and rhesus monkeys.

Since there is little published data on the microscopic pathology of "Q" fever, a detailed study of the histopathology of experimentally produced "Q" fever in mice is reported here.

MATERIAL AND METHODS

The virus used in this experiment was the "X" strain which was isolated and fully identified in this laboratory (*4*). For the purpose of inoculation, the virus was obtained either from rickettsiae growing on yolk sac membrane, or from the spleens of infected mice; in either case, rickettsiae were demonstrated by smears before preparing the inoculum. The infected yolk sac or spleen was made up into a 10 percent suspension in normal saline.

A total of 91 young Swiss mice were used. Under light ether anesthesia 54 were inoculated intranasally with a suspension of infected yolk sac, while 20 controls were inoculated in the same manner with a 10 percent suspension of normal yolk sac. The remaining 17 were inoculated with a suspension of infected spleen, 12 by the intranasal route, and 5 intraperitoneally. Two-tenths of a cubic centimeter was given to each mouse inoculated intranasally, while 0.5 cc. was used for intraperitoneal injection.

The mice were killed by chloroform inhalation at intervals varying from 1 to 21 days after inoculation. The organs were removed at once and placed in Orth's solution for fixation. Sections for routine study were stained with a modified Romanowsky (*5*) and Van Gieson's stains.

INTRANASAL INOCULATION WITH INFECTED AND NORMAL YOLK SAC

Lungs.—Pneumonia was found in 52 of the 54 mice which were inoculated with infected yolk sac. One day after inoculation the

[1] From the Division of Pathology and the Division of Infectious Diseases, National Institute of Health.

[2] Subsequent to the submission of this paper for publication in May 1941, Findlay (*3*) has reported on the pathology of pneumonitis in mice infected intranasally with "Q" fever.

involvement was confined to a few small peribronchial areas. The inflammatory process was exudative at this stage and the cells of the exudate were largely polymorphonuclear neutrophils.

In mice examined 2 and 3 days after inoculation, the pneumonic process was more extensive. The exudative changes were prominent, but proliferative changes were seen and in some animals were equally prominent. Purulent exudate in small amounts was present within most bronchi. In the alveolar exudate, serum was present in some areas, and the cells were about half polymorphonuclear and half mononuclear. The latter were chiefly large and either of the macrophage type or of spindle to polyhedral shape with large leptochromatic nuclei and scanty cytoplasm. The proliferative changes were characterized by adventitial cell proliferation around vessels and bronchi, and by the presence of large, actively multiplying mononuclear cells in alveoli, and on or within thickened interalveolar septa.

In animals killed from the fourth through the eleventh day after inoculation, the pneumonic process was moderately extensive. It was chiefly patchy and peribronchial in distribution, although it was diffuse in some lobes. The proliferative phase nearly always overshadowed the exudative phase, and large mononuclear cells and lymphocytes outnumbered the polymorphonuclears. A little purulent exudate was seen in some bronchi and focally the bronchial epithelial cells were swollen and slightly heaped. Small to moderate numbers of lymphocytes and fewer polymorphonuclears were seen in the connective tissue around bronchi and vessels, and lymphocytes in small numbers were infiltrating interalveolar septa. Nodular accumulations of coherent spindle-shaped mononuclear cells were occasionally seen within alveoli or in septa; the mononuclear cells often tended to be arranged concentrically, and a few polymorphonuclears and lymphocytes were at times intermingled.

In animals killed on the fourteenth and twenty-first days after inoculation, the pneumonic areas were small and the scanty exudate was usually composed of macrophages. Nodular accumulations of spindle-shaped mononuclear cells similar to those described above were present, and generally they were more conspicuous, owing to less prominent pneumonic changes; in each of two nodules seen in one lung, a multinucleate giant cell was observed. Proliferative changes in lung tissue were similar to but less marked than those seen in mice killed before the fourteenth day. Perivascular, peribronchial, and septal lymphocyte infiltration was also less marked, and focal heaping of bronchial epithelium was seldom seen.

Pneumonia was observed in 8 of the 20 control mice. The first observations were made on the fourth day after inoculation, and the pneumonic process was slightly less extensive than that seen in "Q"

infected mice killed on the same day. In the controls it tended to be more exudative and polymorphonuclears usually outnumbered the mononuclear cells in the exudate.

Six and 7 days after inoculation, 3 of 6 controls had no pneumonia, and 1 had only very slight involvement. In the remaining 2, pneumonia was moderately extensive; the proliferative and exudative changes were about equal, as were the cell types in the exudate. Otherwise, the inflammatory process was essentially similar to that described in "Q" infected mice, except that nodular accumulations of concentrically arranged mononuclear cells were not seen.

Only 1 of 10 control mice killed on the fourteenth and eighteenth days had pneumonic lesions.

Spleen.—The most striking changes produced by the inoculation of the virus were found in the spleen. The lesions were nodular or patchy, granulomatous in nature, and nearly always located in the red pulp. They were composed chiefly of large spindle to polyhedral shaped mononuclear cells with oval or elongated leptochromatic nuclei and moderately abundant cytoplasm which was lightly oxyphilic with Romanowsky staining. The cells were at times concentrically arranged. Small to moderate numbers of polymorphonuclears were often found in the lesions, and less often lymphocytes were seen; not infrequently a few degenerating cells with pyknotic and karyorrhectic nuclei were included.

The lesions were first observed on the fourth day after inoculation in 3 of the 6 animals examined. After the fourth day, they were present in all but 2 of the 36 mice studied. The lesions varied in number from spleen to spleen in mice killed on the same day; only an occasional lesion was seen in some spleens, while in others the red pulp was almost completely replaced. There was no definite relation between the time of observation and the number of lesions present after the fourth day; a mouse killed on the fourth day could have as few or as many lesions as one killed on the tenth or twenty-first day.

The lesions varied in size, but the majority were about 100 to 200μ in diameter. There was no clear-cut difference in size between the first lesions seen in mice killed on the fourth day, and in those observed at later intervals. The mononuclear cells were a little larger in the latter, and polymorphonuclears were at times replaced by lymphocytes.

Collagenization was not seen in the nodules at any stage, and in several sections stained for reticulum, reticulum fibers were few or absent.

In the 20 control mice, no lesions similar to those described above were seen.

Other changes were observed in both the infected and the control mice. Follicle hyperplasia was seen more often in control than in

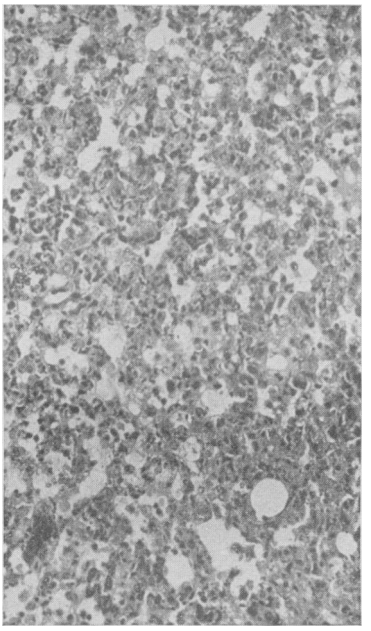

FIGURE 1.—Lung, 6 days. Thickening of interalveolar septa. Serocellular exudate in alveoli. (×150)

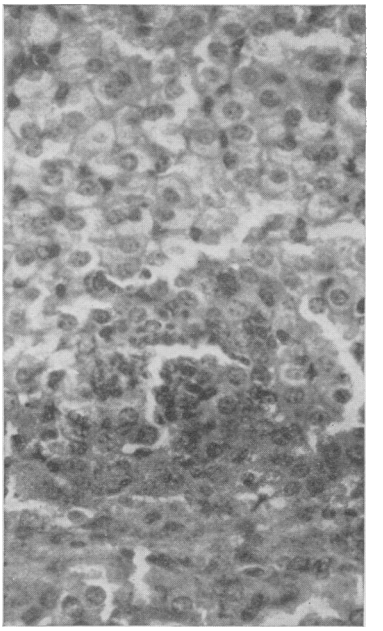

FIGURE 2.—Adrenal, 6 days. Granulomatous lesion at corticomedullary junction. Cortex above. (×400)

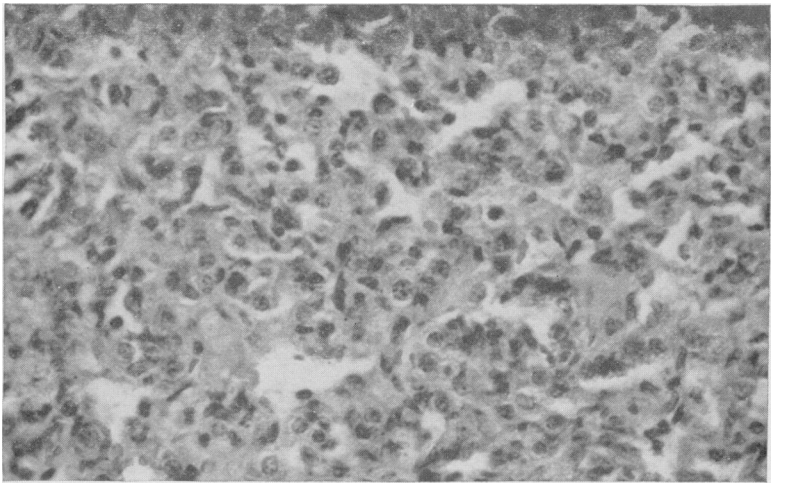

FIGURE 3.—Lung, 9 days. Thickening of interalveolar septa and proliferation of mononuclear cells. (×400)

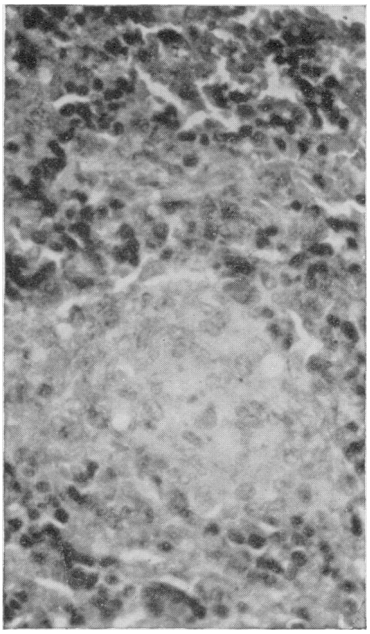

FIGURE 4.—Spleen, 14 days. Granulomatous lesion in red pulp. (×400)

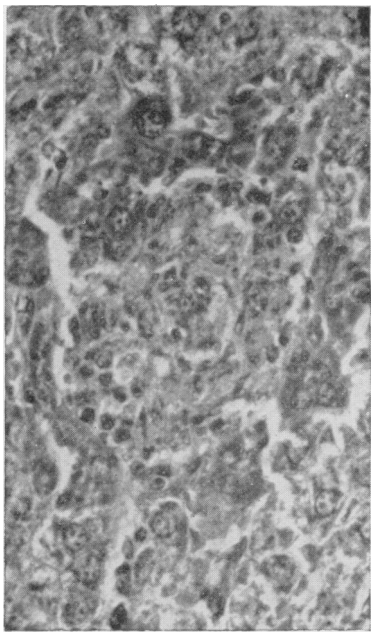

FIGURE 5.—Liver, 4 days. Granulomatous lesion. (×400)

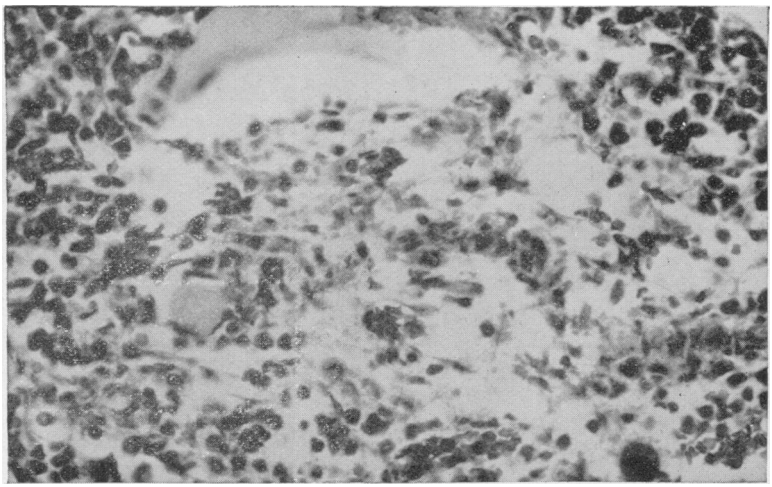

FIGURE 6.—Bone marrow, 9 days. Aplastic lesion. (×400)

infected mice, while varying degrees of phagocytosis of nuclear fragments by reticulum cells of the follicles was observed with equal frequency in both. Increased numbers of lymphoid and myeloid cells in the red pulp were often seen in control mice and in infected mice which had few nodular lesions; however, when lesions were numerous, lymphoid and myeloid cells were largely replaced by the granulomatous lesions.

Liver.—Examination of the liver in mice inoculated with infected yolk sac revealed nodular granulomatous lesions quite similar to those described in the spleen; they were usually not as numerous and they were not found with such regularity. The changes were first seen on the fourth day, corresponding with the appearance of splenic lesions.

The lesions occurred in various parts of the liver lobules and were approximately of the same size as those seen in the spleen. However, they were often not as compact, and the tendency toward concentric arrangement of the mononuclear cells was not as prominent.

A very few shrunken and degenerating liver cells were occasionally seen in or adjacent to lesions, and marginating liver cells usually had a more homogeneous and deeply staining cytoplasm than those seen elsewhere.

The lesions were found in 31 of the 42 infected mice killed on the fourth day or after. When lesions were present, there was no apparent relation between the time of observation and the number or size of the lesions found.

Three of the 20 control mice showed a few nodular lesions similar to those described above.

In a few of the infected mice, there was a slight increase in size of liver cells and a small to moderate number of mitoses were observed. Other changes in the liver were seen with about equal frequency in infected and control mice. These changes included congestion, slight to moderate focal paravascular and portal lymphocyte infiltration, and a slight to moderate increase in the number of lymphoid cells in the sinusoids; the increase in lymphoid cells was at times associated with proliferation of sinusoidal endothelium.

Bone marrow (vertebral).—The bone marrow was negative in infected mice examined prior to the sixth day after inoculation. On and after the sixth day lesions were observed, but they were different in appearance from those seen in the liver and spleen. They appeared as small to moderate sized, nodular or patchy areas of degeneration and aplasia. Occasionally, moderate numbers of adult polymorphonuclears were seen in the early lesions, but for the most part there was almost complete absence of recognizable myeloid cells and only a loose network of stromal cells remained. Usually a small number

of shrunken cells with pyknotic and karyorrhectic nuclei were seen in the fibrillar network connecting the stromal cells.

The lesions first seen on the sixth and seventh days were of smaller size than those observed in mice killed later on. There was slight stromal cell proliferation in the later stages.

The lesions were present in 2 of the 5 infected mice studied on the sixth day, and were found in 15 of the 18 mice in which bone marrow was examined thereafter.

The bone marrow examined in control mice was normal.

Adrenals.—Histopathologic changes were observed in the adrenals in 20 of 32 infected mice killed on and after the sixth day. The pathologic changes were of two types, appearing either as nodular granulomatous lesions similar to those noted in the liver and spleen, or as foci of lymphocyte infiltration. The granulomatous lesions were smaller than those seen in either the liver or spleen and were few in number. The lesions were most often cortical in location, and while they were occasionally seen together in one adrenal, one type was usually present without the other. Granulomatous lesions were present in 8 mice and focal lymphocyte infiltration in 15.

The adrenals examined in control mice were normal.

Kidneys.—Small nodular or patchy areas of interstitial cell proliferation, some of which were granulomatous in appearance, were seen in the kidneys of infected mice killed on and after the sixth day; there was an associated lymphocyte infiltration in some lesions, and at times a few polymorphonuclears and degenerating cells were seen. The lesions were found in 16 of the 35 infected mice examined after the fifth day.

Focal interstitial and perivascular lymphocyte infiltration of slight to moderate degree, not associated with interstitial cell proliferation, occurred with equal frequency in the infected and control mice.

Heart.—Slight to moderate focal lymphocyte infiltration of the myocardium and epicardium was seen in infected and control mice.

Mediastinal and retroperitoneal lymph nodes.—Varying degrees of sinus dilatation, with or without proliferation of sinus endothelium, and slight to moderate phagocytosis of nuclear fragments by reticulum cells of the follicles was observed in approximately the same proportion of control and infected mice.

Thymus.—In a few of the infected and control mice, small cortical cells were reduced in number and partially replaced by large phagocytic mononuclear cells. This process, probably involutional in type, has been observed in mice in other experimentally produced infections (unpublished data). Nelson and Oliphant (*6*) described apparently similar changes in mice experimentally infected with influenza virus.

Stomach, small intestine, and colon.—Foci of lymphocyte and plasma cell infiltration seen in the mucosa or submucosa and less often in the

serosa were not significantly more frequent in the infected than in the control animals.

Testicle.—In most of the infected and control mice, a few small foci of epithelial cell degeneration were seen in seminiferous tubules.

Brain and meninges.—The brain and meninges were examined in 52 infected mice. Lymphocytic meningitis of moderate degree was observed in 2. Three other mice showed a few scattered microglial nodules and small perivascular lymphocyte foci in the thalamus, basal ganglia, and cerebral cortex.

Similar lesions were found in 1 of the 16 control mice in which the brain and meninges were examined.

Other organs.—The thyroid, esophagus, pancreas, spinal cord, and spinal ganglia were examined in a majority of the infected and control mice. No lesions were found.

INTRANASAL INOCULATION WITH A SUSPENSION OF INFECTED SPLEEN

Twelve mice were inoculated intranasally with a suspension of infected spleen. In these mice histopathologic lesions essentially similar to these described in mice inoculated with infected yolk sac were found in the lungs, liver, spleen, bone marrow, adrenals, and kidneys. In addition, a few small nodular or patchy granulomatous areas were seen in the pulp of mediastinal and retroperitoneal lymph nodes. In some of these lesions, nuclear fragments were at times found, and occasionally a multinucleate giant cell was observed.

INTRAPERITONEAL INOCULATION WITH INFECTED SPLEEN

With the exception of the lungs, the 5 mice inoculated intraperitoneally with infected spleen showed similar lesions to those inoculated intranasally with the spleen suspension. The lungs in the mice inoculated intraperitoneally were essentially negative.

DEMONSTRATION OF RICKETTSIAE

The demonstration of the rickettsiae of "Q" fever in smears from infected mouse spleen and liver is usually best accomplished after several passages of infected material, since rickettsiae are often present in increasing numbers in successive transfers.

This experiment was originally planned for the study of histopathologic lesions without regard to the possible demonstration of rickettsiae, and the majority of mice studied represented first passage of the virus from yolk sac. Liver and spleen smears from several of the mice, made at the time of autopsy, showed a very few or no rickettsiae. Correspondingly, in the routine sections stained with Romanowsky stain (*5*), only a very few mice showed an occasional small accumulation of rickettsiae in spleen, liver, and lung.

Since Burnet and Freeman (*2*) described numerous rickettsiae in the liver and spleen in mice experimentally infected with the virus of "Q" fever, it was decided to transfer the virus in small groups of mice until numerous rickettsiae could be demonstrated by smear, and to examine histologic sections from the same tissues for the purpose of observing rickettsiae in stained tissue.

In a group of 4 mice, representing the third passage from yolk sac, numerous rickettsiae were seen in smears from the spleen, and a smaller number in smears from the liver. Histologically, the tissues were similar in appearance to those described above, with numerous granulomatous lesions present in splenic tissue, and a smaller number observed in the liver.

Rickettsiae were found in the sections of both spleen and liver, but they were much more numerous in the former. They usually appeared in sharply circumscribed, round to oval accumulations, both intra and extra-cellular in location. The accumulations varied from about 3μ to 25μ in diameter. The individual organisms within the accumulations were minute coccoid or rod-shaped bodies of rather indistinct outline. They were pale blue in color with Romanowsky stain and were usually set in a very pale blue matrix. The bodies were much paler than mast cell granules or nuclear chromatin and were gram negative. They were not acid fast and did not react as ferric iron with ferrocyanide.

In the spleen, rickettsiae were found only in the red pulp where they were irregularly distributed; they were found both within and apart from the granulomatous lesions. In the spleen the intracellular accumulations were found in large mononuclear cells, and in the liver they were found in the Kupffer cells.

DISCUSSION

The only reference in the literature to the histopathologic findings in mice infected with the virus of "Q" fever is in a report by Burnet and Freeman (*2*). They described only the liver and spleen, and stated that in the latter the only feature calling for comment was the character and distribution of rickettsiae. In the liver they found a diffuse infiltration with cells which appeared to be largely of vascular endothelial origin, variable numbers of small inflammatory necrotic foci, and rickettsiae in interstitial (Kupffer) cells.

In our experiment, striking lesions were present in the liver and spleen, as well as in other organs; the brief description of the liver findings by Burnet and Freeman does not permit an accurate comparison of the changes in this organ. Rickettsiae were demonstrated satisfactorily in tissue sections only after successive transfers of infected material in order to increase the number of rickettsiae.

The "X" strain of the "Q" fever virus used in this experiment has been proved immunologically identical to a strain of Australian "Q" fever virus supplied to this laboratory by Burnet. Lillie (*7*), working with the "X" strain, the Australian strain referred to above, and a third strain ("M") of "Q" fever virus in guinea pigs, has found lesions which are more widespread than those found in mice in this study. The lesions in guinea pigs included both granulomatous lesions and focal lymphocyte exudation, and in the granulomatous lesions multinucleate giant cells were more frequently encountered than in the mice described in this report.

The pneumonic reaction found in the lungs of mice in this experiment is similar to that described by Lillie, Perrin, and Armstrong (*3*) in the lungs of monkeys inoculated with the virus of "Q" fever, and, with exceptions, to the pneumonic process in a fatal human case described by the same authors. In the human case fibrin was abundant in the alveolar exudate, while it was not seen in the pneumonia in mice.

SUMMARY

Histopathologic observations have been made on mice killed at intervals from 1 to 21 days after intranasal and intraperitoneal inoculation with a suspension of "Q" fever virus. The virus suspension was prepared from rickettsiae growing on yolk sac and from the spleens of infected mice. In mice inoculated by both routes and using both sources of the virus, nodular and patchy granulomatous lesions composed chiefly of large mononuclear cells were found in the spleen, liver, kidneys, and adrenals. Nodular and patchy areas of aplasia and degeneration were found in the bone marrow. The lesions were first seen on the fourth day in the spleen and liver, and on the sixth day in the kidneys, adrenals, and bone marrow.

A pneumonic reaction characterized by early and prominent proliferative changes, and by the predominance of large and small mononuclear cells in the exudate, was seen only in intranasally inoculated mice.

Granulomatous lesions in mediastinal and retroperitoneal lymph nodes were similar to those seen in the other organs noted above, but they were found only in mice inoculated with a suspension of infected spleen.

Control mice were inoculated intranasally with a suspension of normal yolk sac. Pneumonia was found in 8 of 20 (40 percent), as compared to 52 of 54 (96 percent), infected mice. The pneumonia seen in the control mice tended to be more exudative than proliferative and polymorphonuclears predominated in the exudate.

Nodular granulomatous lesions were seen in the liver in 3 (15 percent) of the control mice, while they were found in 31 of the 42

(74 percent) infected mice killed after the third day. There were no comparable lesions found in the spleen, bone marrow, kidneys, adrenals, and lymph nodes in control mice.

In the infected mice, no significant lesions were found in the heart, thyroid, esophagus, stomach, small intestine, colon, pancreas, testicles, brain, meninges, spinal cord, or spinal ganglia.

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SILICOSIS AMONG METAL MINE WORKERS [1]

A Review

Public Health Bulletin No. 277, which was recently issued, reports the presence of silicosis among a group of metal mine workers in Utah. Previous investigations in this country by the United States Bureau of Mines in cooperation with the United States Public Health Service had indicated the existence of a serious problem of pulmonary disease among metal mine workers near Joplin, Mo. The present study is part of an investigation of the health and working environment of industrial workers in Utah, made with the cooperation of agencies such as the State Industrial Commission, the State Board of Health, industrial organizations, and labor groups.

Sixty-six cases of silicosis were found by medical and X-ray examination of 727 workers whose only experience in dusty trades had been in nonferrous metal mines. Quantitative evaluation of the working environment was made. Correlation of these findings

[1] Health and working environment of nonferrous metal mine workers. By W. C. Dreessen, R. T. Page, J. W. Hough, V. M. Trasko, J. L. Jones, and R.W. Franks. With a chapter on the physiological response of peritoneal tissue by J. W. Miller. Public Health Bulletin No. 277. Government Printing Office, 1942. For sale by the Superintendent of Documents, Washington, D. C. Price 20 cents per copy.

indicated that the incidence of silicosis increased regularly with severity and duration of dust exposure.

When the principal occupational experience of metal mine workers was taken into consideration, it was evident that the incidence of silicosis was concentrated among workers at the face (i. e., drillers, miners, and muckers). Among the group of face workers employed in metal mines for 10 years or more, 29.5 percent were found to have silicosis, while all other workers had an incidence of 7.5 percent.

Lead poisoning ranked next to silicosis in importance as an occupational disease of workers in the mines studied.

On the basis of the data presented, if the atmospheric dust in these and similar metal mining operations is kept below 10 million particles per cubic foot, no disabling silicosis should occur and morbidity from lead may also be expected to decrease.

Nonindustrial diseases such as tuberculosis, syphilis, and heart disease did not differ appreciably in prevalence from that observed in other industrial workers.

Recommendations for elimination or control of environmental health hazards were made.

DEATHS DURING WEEK ENDED MAY 9, 1942

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

	Week ended May 9, 1942	Corresponding week, 1941
Data from 86 large cities of the United States:		
Total deaths	8,184	8,161
Average for 3 prior years	8,385	
Total deaths, first 18 weeks of year	159,961	168,133
Deaths per 1,000 population, first 18 weeks of year, annual rate	12.5	12.8
Deaths under 1 year of age	541	468
Average for 3 prior years	494	
Deaths under 1 year of age, first 18 weeks of year	10,045	9,310
Data from industrial insurance companies:		
Policies in force	64,975,585	64,517,124
Number of death claims	11,858	12,394
Death claims per 1,000 policies in force, annual rate	9.5	10.0
Death claims per 1,000 policies, first 18 weeks of year, annual rate	10.1	10.7

PREVALENCE OF DISEASE

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

UNITED STATES

REPORTS FROM STATES FOR WEEK ENDED MAY 16, 1942

Summary

The incidence of both measles and meningococcus meningitis for the current week and the cumulative totals to date (first 19 weeks of the year) continue above the 5-year (1937–41) median. The highest incidence rates for measles are in the Pacific, Mountain, and New England States, which areas reported approximately one-half of the total of 22,632 cases for the current week, and the highest incidence of meningococcus meningitis is reported from the New England, Pacific, South Atlantic, and West South Central areas. The total number of reported cases of meningococcus meningitis declined from 89 for the preceding week to 86 for the current period. The largest numbers of cases were reported in New York State (14), Texas (7), and California (7).

A total of 14 cases of poliomyelitis was reported, as compared with 19 for the preceding week, while smallpox cases increased from 18 to 24, of which 8 cases were reported in Tennessee, 4 in Arkansas, and 3 in Missouri. The current incidence of diphtheria, poliomyelitis, scarlet fever, smallpox, and whooping cough is below that for any corresponding week of record.

The onset of the Rocky Mountain spotted fever season in the East is indicated by the occurrence in the Eastern States of 7 of the 17 cases reported for the week. Montana, Idaho, and Wyoming reported the remaining 10 cases.

Other reports include 19 cases of amebic, 69 cases of bacillary, and 64 cases of unspecified dysentery; 27 cases of tularemia, and 24 cases of endemic typhus fever (of which all but 1 case (in California) occurred in the Southern States).

The death rate for 88 large cities in the United States for the current week is 11.5 per 1,000 population, as compared with 11.6 for the preceding weeks and a 3-year (1939–41) average of 11.9. The cumulative rate to date (first 19 weeks) this year is 12.5, as compared with 12.7 for the same period last year.

Telegraphic morbidity reports from State health officers for the week ended May 16, 1942, and comparison with corresponding week of 1941 and 5-year median

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

Division and State	Diphtheria			Influenza			Measles			Meningitis, meningococcus		
	Week ended		Median 1937-41	Week ended		Median 1937-41	Week ended		Median 1937-41	Week ended		Median 1937-41
	May 16, 1942	May 17, 1941		May 16, 1942	May 17, 1941		May 16, 1942	May 17, 1941		May 16, 1942	May 17, 1941	
NEW ENG.												
Maine	0	0	1	------	------	------	127	141	140	3	0	0
New Hampshire	0	0	0	------	------	------	38	40	23	1	0	0
Vermont	1	0	0	------	------	------	147	45	45	0	0	0
Massachusetts	7	2	2	------	------	------	1,280	1,053	766	6	0	1
Rhode Island	1	0	0	------	------	------	275	4	74	0	0	0
Connecticut	0	0	3	2	------	1	407	467	333	0	0	0
MID. ATL.												
New York	15	16	26	[1] 5	4	7	855	4,134	2,320	14	5	5
New Jersey	3	5	9	3	5	5	726	2,499	934	4	0	0
Pennsylvania	10	16	16	------	------	------	1,329	5,921	1,530	3	1	4
E. NO. CEN.												
Ohio	10	4	9	5	11	11	497	3,001	1,801	0	1	2
Indiana	1	4	7	1	9	8	219	1,097	609	1	1	0
Illinois	14	8	32	3	13	13	445	1,704	296	0	0	2
Michigan [2]	4	5	8	0	2	2	570	3,035	802	0	1	1
Wisconsin	0	1	2	5	15	32	1,401	2,021	1,065	1	3	0
W. NO. CEN.												
Minnesota	4	23	3	0	1	1	862	24	89	0	1	1
Iowa	3	2	2	1	4	3	313	205	205	0	0	0
Missouri	4	2	5	0	1	2	251	590	39	3	1	1
North Dakota	1	1	1	9	------	2	17	41	41	0	0	0
South Dakota	4	0	1	0	------	------	89	17	1	0	0	0
Nebraska	1	0	4	27	------	------	402	21	21	0	0	0
Kansas	4	1	3	4	15	2	597	771	453	0	0	0
SO. ATL.												
Delaware	0	0	0	0	------	------	8	137	27	0	0	0
Maryland [2]	3	6	6	3	3	4	423	400	241	5	3	2
Dist. of Col	0	0	2	0	------	------	106	251	104	4	0	0
Virginia	5	9	9	114	52	52	167	1,149	496	5	2	3
West Virginia	0	7	4	8	4	20	34	619	39	1	1	1
North Carolina	4	9	9	8	6	6	706	1,622	356	2	0	2
South Carolina	2	5	6	161	327	179	100	751	74	1	0	0
Georgia	2	2	5	46	23	23	217	550	109	0	0	0
Florida	6	5	5	1	45	4	219	357	137	0	1	1
E. SO. CEN.												
Kentucky	2	3	4	0	------	5	68	1,057	286	2	2	2
Tennessee	2	2	4	27	37	45	154	425	134	1	3	3
Alabama	4	1	4	20	49	49	143	400	149	0	1	2
Mississippi [2]	6	3	3	------	------	------	------	------	------	1	4	1
W. SO. CEN.												
Arkansas	[3] 4	3	5	[3] 20	8	34	193	371	55	2	0	0
Louisiana	7	2	8	2	4	7	223	52	14	4	0	1
Oklahoma	2	1	2	19	22	25	153	74	74	0	0	0
Texas	24	18	27	301	442	230	991	1,146	758	7	0	3
MOUNTAIN												
Montana	2	0	0	0	1	1	207	36	42	0	0	0
Idaho	1	0	0	1	------	1	156	12	22	0	0	0
Wyoming	0	0	0	64	1	------	93	30	28	0	0	0
Colorado	7	7	7	35	14	4	260	641	299	2	0	0
New Mexico	6	1	1	3	------	2	27	212	72	0	0	0
Arizona	2	2	2	71	65	52	144	125	66	2	0	0
Utah [2]	0	1	0	3	12	------	1,269	63	86	0	0	0
Nevada	------	0	------	------	------	------	4	0	------	0	0	------
PACIFIC												
Washington	1	1	1	------	------	------	547	21	62	4	0	0
Oregon	0	2	4	12	8	18	185	197	67	0	0	0
California	6	13	16	24	72	53	4,988	450	450	7	1	2
Total	185	193	288	1,008	1,275	1,014	22,632	37,979	15,800	86	32	48
19 weeks	[3] 5,255	5,145	8,468	[3] 73,372	477,010	153,546	351,766	654,845	258,610	1,486	942	942

See footnotes at end of table.

Telegraphic morbidity reports from State health officers for the week ended May 16, 1942, and comparison with corresponding week of 1941 and 5-year median—Con.

Division and State	Poliomyelitis			Scarlet fever			Smallpox			Typhoid and paratyphoid fever		
	Week ended		Median 1937–41	Week ended		Median 1937–41	Week ended		Median 1937–41	Week ended		Median 1937–41
	May 16, 1942	May 17, 1941		May 16, 1942	May 17, 1941		May 16, 1942	May 17, 1941		May 16, 1942	May 17, 1941	
NEW ENG.												
Maine	0	0	0	4	12	11	0	0	0	1	0	0
New Hampshire	0	0	0	7	1	5	1	0	0	0	0	0
Vermont	0	0	0	12	9	9	0	0	0	1	0	0
Massachusetts	0	0	0	284	214	214	0	0	0	1	11	2
Rhode Island	0	0	0	14	20	13	0	0	0	0	0	0
Connecticut	0	0	0	20	41	107	0	0	0	2	0	1
MID. ATL.												
New York	3	0	0	366	488	727	0	0	0	4	9	8
New Jersey	0	1	0	158	274	261	0	0	0	1	2	2
Pennsylvania	0	1	1	406	388	388	0	0	0	9	5	5
E. NO. CEN.												
Ohio	0	0	0	269	228	351	0	0	0	4	4	4
Indiana	0	0	0	56	82	85	0	1	21	1	0	2
Illinois	0	1	1	128	298	420	0	6	11	2	4	4
Michigan [3]	0	0	0	188	255	385	0	0	1	0	5	2
Wisconsin	1	0	0	112	127	131	0	2	2	2	0	0
W. NO. CEN.												
Minnesota	0	0	0	45	55	70	1	0	4	0	1	1
Iowa	0	0	0	23	29	75	1	8	20	2	1	1
Missouri	0	0	0	58	160	64	3	3	3	2	0	2
North Dakota	0	0	0	5	2	12	0	0	1	0	0	0
South Dakota	0	1	0	12	14	16	0	21	5	0	0	0
Nebraska	0	0	0	26	8	23	0	1	5	0	0	0
Kansas	[3] 0	0	0	50	19	54	0	1	4	2	1	1
SO. ATL.												
Delaware	0	0	0	30	12	4	0	0	0	0	0	0
Maryland [3]	1	0	0	54	33	33	0	0	0	2	4	2
Dist. of Col	0	0	0	6	11	12	0	0	0	0	1	0
Virginia	0	1	0	15	19	19	0	0	0	1	4	4
West Virginia	0	0	0	26	38	38	0	0	0	1	2	3
North Carolina	1	0	1	16	16	22	1	1	0	3	2	2
South Carolina	1	0	0	3	12	2	0	0	0	2	1	1
Georgia	0	0	0	10	16	9	0	4	1	10	6	5
Florida	2	1	1	4	2	6	0	0	0	7	4	4
E. SO. CEN.												
Kentucky	1	0	0	44	115	48	0	9	3	5	6	5
Tennessee	0	0	0	25	43	43	8	0	1	2	5	5
Alabama	2	1	1	8	13	7	1	0	0	0	1	5
Mississippi [3]	0	1	1	0	0	5	1	0	0	4	0	2
W. SO. CEN.												
Arkansas	1	1	0	6	3	6	4	1	1	2	1	2
Louisiana	0	1	0	12	3	10	0	0	0	11	6	9
Oklahoma	0	1	0	2	14	18	2	0	1	2	3	3
Texas	0	1	1	48	33	37	1	1	5	7	3	7
MOUNTAIN												
Montana	0	0	0	16	15	17	0	0	2	0	0	1
Idaho	0	0	0	7	1	9	0	0	0	0	0	0
Wyoming	1	0	0	19	9	5	0	0	0	0	0	0
Colorado	0	0	0	22	23	38	0	2	5	3	1	2
New Mexico	0	0	0	0	0	13	0	0	0	0	3	1
Arizona	0	3	0	4	3	7	0	0	0	0	1	1
Utah [3]	0	0	0	20	9	20	0	0	0	0	0	0
Nevada	0	0	-----	0	0	-----	0	0	-----	0	0	-----
PACIFIC												
Washington	0	1	1	18	24	25	0	1	1	2	0	1
Oregon	0	0	1	13	7	18	0	1	19	0	0	1
California	0	2	3	71	105	147	0	0	6	4	1	8
Total	14	18	19	2,742	3,303	4,284	24	63	250	102	98	129
19 weeks	[3] 391	417	395	72,081	70,865	94,223	419	882	5,987	1,507	1,506	2,119

See footnotes at end of table.

Telegraphic morbidity reports from State health officers for the week ended May 16, 1942—Continued

Division and State	Whooping cough		Week ended May 16, 1942								
	Week ended			Dysentery							
	May 16, 1942	May 17, 1941	Anthrax	Amebic	Bacillary	Unspecified	Encephalitis, infectious	Leprosy	Rocky Mountain spotted fever	Tularemia	Typhus fever
NEW ENG.											
Maine	26	36	0	0	0	0	0	0	0	0	0
New Hampshire	2	13	0	0	0	0	0	0	1	0	0
Vermont	31	28	0	0	0	0	0	0	0	0	0
Massachusetts	239	257	0	0	0	0	1	0	0	0	0
Rhode Island	36	29	0	0	0	0	0	0	0	0	0
Connecticut	100	93	0	1	0	0	0	0	0	0	0
MID. ATL.											
New York	437	257	0	5	2	0	2	0	1	0	0
New Jersey	313	134	0	0	0	0	2	0	0	0	0
Pennsylvania	219	392	0	0	0	0	0	0	0	0	0
E. NO. CEN.											
Ohio	189	452	0	0	0	1	2	0	0	0	0
Indiana	58	35	0	0	0	0	0	0	0	0	0
Illinois	243	108	0	1	1	0	2	0	0	2	0
Michigan [1]	187	389	0	2	0	0	0	0	0	0	0
Wisconsin	246	125	0	0	0	0	1	0	0	0	0
W. NO. CEN.											
Minnesota	48	101	0	0	0	0	0	0	0	0	0
Iowa	18	50	0	0	0	0	0	0	0	2	0
Missouri	7	78	0	0	0	0	0	0	0	1	0
North Dakota	7	29	0	0	0	0	1	0	2	0	0
South Dakota	5	19	0	0	0	0	0	0	0	0	0
Nebraska	1	18	0	0	0	0	0	0	0	0	0
Kansas	42	169	0	0	0	0	0	0	0	0	0
SO. ATL.											
Delaware	0	5	0	0	0	0	0	0	0	0	0
Maryland [2]	39	77	0	0	0	0	0	0	2	0	0
Dist. of Col	19	23	0	0	0	0	0	0	0	0	0
Virginia	55	140	0	0	0	27	0	0	0	3	0
West Virginia	1	89	0	0	0	0	0	0	1	0	0
North Carolina	100	276	0	0	0	0	0	0	0	0	1
South Carolina	56	163	0	0	0	0	0	0	0	1	3
Georgia	62	58	0	0	1	0	0	0	0	1	7
Florida	12	21	0	1	0	0	0	0	0	0	1
E. SO. CEN.											
Kentucky	63	67	0	0	0	0	0	0	0	0	0
Tennessee	41	99	0	1	0	0	0	0	0	2	3
Alabama	83	51	0	0	0	0	0	0	0	0	2
Mississippi [2]	------	------	0	0	0	0	0	0	0	1	0
WEST SOUTH CENTRAL											
Arkansas	9	50	0	0	1	0	0	0	0	3	0
Louisiana	8	14	0	3	0	0	0	0	0	2	2
Oklahoma	8	26	0	0	0	0	0	0	0	0	0
Texas	136	309	0	2	63	0	0	0	0	0	4
MOUNTAIN											
Montana	14	24	0	0	0	0	0	0	7	6	0
Idaho	10	22	0	0	0	0	0	0	1	0	0
Wyoming	8	6	0	0	0	0	1	0	2	0	0
Colorado	27	249	0	0	0	0	0	0	0	0	0
New Mexico	14	27	0	1	0	0	0	0	0	0	0
Arizona	36	43	0	0	0	36	0	0	0	0	0
Utah [2]	32	100	0	0	0	0	0	0	0	2	0
Nevada	10	1	0	0	0	0	0	0	0	0	0
PACIFIC											
Washington	75	177	0	0	0	0	0	0	0	0	0
Oregon	21	42	0	0	0	0	0	0	0	0	0
California	265	774	0	2	1	0	1	0	0	1	1
Total	3,658	5,745	0	19	69	64	13	0	17	27	24
19 weeks	73,019	87,979	------	------	------	------	------	------	------	------	------

[1] New York City only.
[2] Period ended earlier than Saturday.
[3] Correction.—Week ended May 9, 1942: Arkansas, diphtheria, 7 cases, influenza, 47; Kansas, poliomyelitis, 1 case.

WEEKLY REPORTS FROM CITIES

City reports for week ended May 2, 1942

This table lists the reports from 89 cities of more than 10,000 population distributed throughout the United States, and represents a cross section of the current urban incidence of the diseases included in the table.

	Diphtheria cases	Encephalitis, infectious, cases	Influenza		Measles cases	Meningitis, meningococcus, cases	Pneumonia deaths	Poliomyelitis cases	Scarlet fever cases	Smallpox cases	Typhoid and paratyphoid fever cases	Whooping cough cases
			Cases	Deaths								
Atlanta, Ga	0	0	6	1	4	0	2	0	6	0	2	3
Baltimore, Md	0	0	1	0	309	4	14	0	37	0	0	33
Barre, Vt	0	0	----	0	0	0	0	0	0	0	0	3
Billings, Mont	0	0	----	0	9	0	0	0	0	0	0	0
Birmingham, Ala	0	0	3	0	5	0	6	0	4	1	0	2
Boise, Idaho	0	0	----	0	----	0	0	0	----	0	0	0
Boston, Mass	0	0	----	0	401	0	14	0	110	0	0	44
Bridgeport, Conn	0	0	----	0	11	1	0	0	5	0	0	0
Brunswick, Ga	0	0	----	0	9	0	0	0	0	0	0	0
Buffalo, N. Y	0	0	----	1	14	0	4	0	12	0	0	7
Camden, N. J	0	0	----	0	3	0	1	0	17	0	0	0
Charleston, S. C	0	0	4	0	6	0	2	0	0	0	1	7
Charleston, W. Va	0	0	----	0	0	0	0	0	0	0	0	0
Chicago, Ill	14	0	----	1	93	0	25	0	70	0	0	98
Cincinnati, Ohio	0	0	----	0	0	0	3	0	25	0	0	21
Cleveland, Ohio	0	0	2	1	9	1	3	0	76	0	0	39
Columbus, Ohio	3	0	1	1	32	0	5	0	6	0	0	13
Concord, N. H	0	0	----	1	0	0	1	0	0	0	0	0
Cumberland, Md	0	0	----	0	2	0	0	0	2	0	0	0
Dallas, Tex	0	0	----	0	100	0	2	0	3	0	1	5
Denver, Colo	4	0	10	0	153	0	5	0	6	0	0	6
Detroit, Mich	0	0	----	2	16	1	11	0	119	0	1	85
Duluth, Minn	0	0	----	0	3	0	2	0	7	0	0	0
Fall River, Mass	0	0	----	0	61	0	1	0	28	0	0	0
Fargo, N. Dak	0	0	----	0	0	0	0	0	0	0	0	0
Flint, Mich	0	0	----	0	4	0	1	0	1	0	0	9
Fort Wayne, Ind	0	0	----	0	0	0	5	0	0	0	0	1
Frederick, Md	0	0	----	0	0	0	0	0	0	0	0	0
Galveston, Tex	0	0	----	0	7	0	0	0	2	0	0	0
Grand Rapids, Mich	0	0	----	1	5	0	0	0	2	0	0	3
Great Falls, Mont	0	0	----	0	44	0	0	0	1	0	0	5
Hartford, Conn	0	0	----	0	33	0	1	0	4	0	0	11
Helena, Mont	0	0	----	0	0	0	0	0	0	0	0	1
Houston, Tex	0	0	----	0	69	0	6	0	2	0	0	1
Indianapolis, Ind	0	0	----	1	75	0	7	0	34	0	0	40
Kansas City, Mo	1	0	----	0	150	0	5	0	42	0	0	3
Kenosha, Wis	0	0	----	0	14	0	0	0	1	0	0	18
Little Rock, Ark	0	0	----	0	7	2	3	0	0	0	0	0
Los Angeles, Calif	2	0	11	1	725	0	18	1	13	0	0	25
Lynchburg, Va	0	0	----	0	0	0	2	0	0	0	0	30
Memphis, Tenn	0	0	3	1	32	0	2	0	3	2	0	12
Milwaukee, Wis	0	0	2	2	133	1	7	0	28	0	0	50
Minneapolis, Minn	0	0	----	0	477	0	3	0	11	0	1	3
Missoula, Mont	0	0	----	0	0	0	0	0	3	0	0	0
Mobile, Ala	1	0	1	1	0	0	1	1	0	0	0	0
Nashville, Tenn	0	0	----	0	----	0	0	0	1	0	0	11
Newark, N. J	0	1	1	0	330	0	3	0	17	0	0	38
New Haven, Conn	0	0	----	1	170	0	1	0	3	0	0	7
New Orleans, La	0	0	1	0	76	1	8	0	3	0	1	5
New York, N. Y	18	0	7	1	93	14	52	0	283	0	5	284
Omaha, Nebr	0	0	----	0	222	0	1	0	5	0	0	1
Philadelphia, Pa	1	1	2	2	56	1	24	0	246	0	0	109
Pittsburgh, Pa	1	0	----	0	10	0	9	0	22	0	2	32
Portland, Maine	0	0	----	0	6	4	1	0	0	0	0	0
Providence, R. I	1	0	----	0	208	1	2	0	10	0	0	31

City reports for week ended May 2, 1942—Continued

	Diphtheria cases	Encephalitis, infectious, cases	Influenza		Measles cases	Meningitis, meningococcus, cases	Pneumonia deaths	Poliomyelitis cases	Scarlet fever cases	Smallpox cases	Typhoid and paratyphoid fever cases	Whooping cough cases
			Cases	Deaths								
Pueblo, Colo	0	0	------	0	5	0	1	0	0	0	0	2
Racine, Wis	0	0	------	0	422	0	1	0	2	0	0	38
Reading, Pa	0	0	------	0	10	0	0	0	1	0	0	6
Richmond, Va	0	1	1	0	6	0	1	0	0	0	0	1
Roanoke, Va	0	0	------	0	0	0	0	0	0	0	0	0
Rochester, N. Y	0	0	------	0	7	0	1	0	0	0	0	77
Sacramento, Calif	0	0	------	0	97	1	7	0	0	0	1	31
Saint Joseph, Mo	0	0	------	0	2	0	0	0	1	0	0	0
Saint Louis, Mo	0	0	3	1	150	1	16	0	27	0	0	4
Saint Paul, Minn	0	0	------	0	206	0	5	0	7	0	0	20
Salt Lake City, Utah	0	0	------	0	99	0	2	0	4	0	0	7
San Antonio, Tex	0	0	1	0	30	0	12	0	0	0	0	1
San Francisco Calif	0	1	3	0	355	1	11	0	7	0	1	14
Savannah, Ga	0	0	1	1	1	0	2	0	0	0	0	-
Seattle, Wash	0	0	------	1	72	1	3	0	1	0	0	24
Shreveport, La	0	0	------	0	3	0	2	0	0	0	0	0
South Bend, Ind	0	0	------	0	2	0	0	0	8	0	0	0
Spokane, Wash	0	0	------	0	45	0	2	0	5	0	0	7
Springfield, Ill	0	0	------	0	50	0	0	0	4	0	0	0
Springfield, Mass	0	0	1	0	93	0	4	0	14	0	0	6
Superior, Wis	0	0	------	0	1	0	0	0	4	0	0	1
Syracuse, N. Y	0	0	------	0	209	0	2	0	1	0	0	49
Tacoma, Wash	0	0	------	0	8	0	0	0	1	0	0	1
Tampa, Fla	0	0	2	2	139	0	3	0	0	0	1	2
Terre Haute, Ind	0	0	------	0	7	0	2	0	0	0	0	0
Topeka, Kans	0	0	------	0	40	0	4	0	1	0	0	4
Trenton, N. J	0	0	------	0	4	0	6	0	10	0	0	9
Washington, D. C	0	0	------	0	84	4	10	0	13	0	1	27
Wheeling, W. Va	0	0	------	0	4	0	1	0	2	0	0	2
Wichita, Kans	0	0	------	0	108	0	2	0	1	0	0	4
Wilmington, Del	0	0	------	0	9	0	4	0	3	0	0	0
Wilmington, N. C	0	0	------	0	7	0	1	0	0	0	0	3
Winston-Salem, N. C	0	0	------	0	37	0	2	0	2	0	0	0
Worcester, Mass	0	0	------	0	5	1	10	0	9	0	0	62

Dysentery, amebic.—Cases: Dallas, 1; New York, 2.
Dysentery, bacillary.—Cases: Chicago, 4; Los Angeles, 1; Rochester, N. Y., 7; New York, 3.
Typhus fever.—Cases: New York, 2; Tampa, 1.

Rates (annual basis) per 100,000 population for the group of 89 selected cities in the preceding table (estimated population, 1942, 34, 042, 779)

Period	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Typhoid and paratyphoid fever cases	Whooping cough cases
		Cases	Deaths						
Week ended May 2, 1942	7.05	10.26	3.68	996.06	58.20	214.13	0.46	2.76	219.80
Average for week 1937-41	14.84	20.56	6.34	[1] 706.50	78.38	283.07	2.47	3.09	192.32

[1] Median.

TERRITORIES AND POSSESSIONS

Hawaii Territory

Plague (rodent).—According to information dated April 20, 1942, a rat found near Makawao Post Office 9.6 miles from the port of Kahului, Island of Maui, T. H., has been proved positive for plague.

FOREIGN REPORTS

BERMUDA

Communicable diseases—1941.—During the year 1941, cases of certain communicable diseases were reported in Bermuda as follows:

Disease	Cases	Disease	Cases
Chickenpox	136	Ophthalmia neonatorum	2
Dengue	1,401	Scarlet fever	8
Diphtheria	19	Tetanus	4
German measles	9	Tuberculosis (all forms)	13
Influenza	182	Typhoid and paratyphoid fever	20
Measles	326	Undulant fever	15
Mumps	15	Whooping cough	2

Vital statistics—1941.—Following are vital statistics for Bermuda for the year 1941:

	Number	Rate per 1,000 population		Number	Rate per 1,000 population
Marriages	295		Deaths from—continued		
Live births	734	23.5	Heart disease	89	
Deaths, all causes	400	12.33	Nephritis	36	
Deaths under 1 year of age	32	[1] 43.59	Pneumonia	22	
Deaths from—			Puerperal septicemia	1	
Appendicitis	4		Senility	7	
Cancer	39		Suicide	1	
Cerebral hemorrhage, embolism, and thrombosis	57		Syphilis	2	
Diabetes mellitus	9		Tetanus	3	
Diphtheria	2		Tuberculosis (all forms)	12	
Enteritis (under 2 years of age)	1		Typhoid fever	2	

[1] Per 1,000 live births.

NOTE.—The estimated civil population for 1941 is 32,451.

CANADA

Provinces—Communicable diseases—Week ended April 18, 1942.—During the week ended April 18, 1942, 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 [1]	British Columbia	Total
Cerebrospinal meningitis		3	1	1	10		1		5	21
Chickenpox		14		203	296	28	38		151	730
Diphtheria	2	12	5	7	7	5	1		1	40
Dysentery				6						6
German measles			7	26	60	8	23		59	183
Influenza		24				3			48	75
Measles			7	431	229	236	6		20	929
Mumps		7	1	326	545	129	247		494	1,749
Pneumonia		11			17	2	1		15	46
Poliomyelitis			2			1				3
Scarlet fever	5	20	17	81	234	39	26		25	447
Tuberculosis	1	1	5	87	38	[2] 47	24		23	226
Typhoid and paratyphoid fever				13	2		6		1	22
Undulant fever				1						1
Whooping cough		24	4	114	64	6	2		43	257
Other communicable diseases	1	5		7	251	33	2		1	300

[1] No reports were received from Alberta for this period.
[2] For the 4-week period ended Apr. 22, 1942.

EGYPT

Notifiable diseases—First 3 quarters of 1941.—During the first 3 quarters of the year 1941, certain notifiable diseases were reported in Egypt as follows:

Disease	Cases	Deaths	Disease	Cases	Deaths
Cerebrospinal fever	142	82	Pneumonia	4,562	3,946
Chickenpox	1,773	15	Poliomyelitis	14	9
Diphtheria	2,839	1,354	Puerperal septicemia	375	288
Dysentery	2,425	360	Tetanus	333	243
Influenza	9,153	141	Tuberculosis (all forms)	4,926	2,590
Leprosy	397	57	Typhoid fever	4,507	924
Malaria	6,789	61	Typhus fever	8,684	1,607
Measles	8,854	2,579	Whooping cough	2,596	158
Plague	14	6			

SWEDEN

Notifiable diseases—February 1942.—During the month of February 1942, cases of certain notifiable diseases were reported in Sweden as follows:

Disease	Cases	Disease	Cases
Cerebrospinal meningitis	3	Poliomyelitis	13
Diphtheria	77	Scarlet fever	1,499
Dysentery	48	Syphilis	25
Epidemic encephalitis	1	Undulant fever	5
Gonorrhea	777	Weil's disease	1
Paratyphoid fever	6		

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

NOTE.—Except in cases of unusual prevalence, only those places are included which had not previously reported any of the above-mentioned diseases, except yellow fever, during the current year. All reports of yellow fever are published currently.

A cumulative table showing the reported prevalence of these diseases for the year to date is published in the PUBLIC HEALTH REPORTS for the last Friday in each month.

Plague

Indochina—Laos.—During the period April 21–30, 1942, 3 deaths from plague were reported in Laos, Indochina.

Peru.—During the month of March 1942, plague was reported in Peru, by Departments, as follows: Lambayeque, 1 case; Libertad, 3 cases; Lima, 8 cases, 4 deaths; Piura, 3 cases, 1 death.

Typhus Fever

France—Vichy.—During the week ended May 2, 1942, 1 case (imported) of typhus fever was reported in Vichy, France.

Hungary.—During the week ended April 25, 1942, 11 cases of typhus fever were reported in Hungary.

Morocco.—During the week ended April 25, 1942, 1,386 cases of typhus fever were reported in Morocco.

Rumania.—During the week ended May 2, 1942, 140 cases of typhus fever were reported in Rumania.

Tunisia.—During the week ended April 18, 1942, 676 cases (71 in Tunis and 14 in Sousse) of typhus fever were reported in Tunisia. For the week ended April 11, 1942, 583 cases (51 in Tunis and 7 in Sfax) were reported.