Public Health Reports

Vol. 57 • NOVEMBER 13, 1942 • *No. 46*

THE ISOLATION OF *HAPLOSPORANGIUM PARVUM* N. SP. AND *COCCIDIOIDES IMMITIS* FROM WILD RODENTS. THEIR RELATIONSHIP TO COCCIDIOIDOMYCOSIS [1] [2]

By C. W. EMMONS, *Senior Mycologist*, and L. L. ASHBURN, *Passed Assistant Surgeon, United States Public Health Service*

The investigations of Dickson (*6*, *7*, *8*, *9*), Gifford (*9*, *12*), Smith (*5*, *15*, *16*, *17*), and many others, by correcting and clarifying earlier concepts, have made coccidioidomycosis well known as a mycotic disease of man, occurring frequently within endemic areas and probably only by importation elsewhere. Giltner (*13*), Beck (*3*, *4*), and Stiles and his associates (*19*, *20*) have reported the occurrence of the disease (in most cases in a localized and arrested form) in cattle and sheep, and Farness (*11*) has reported cases in the dog. It is reported (*19*) that Dr. E. W. Phillips observed a disease in sheep in the vicinity of Phoenix, Ariz., which resembled coccidioidomycosis, and from which he isolated a "fungus-like organism" tentatively identified as a variant of *C. immitis*. The paper by Stiles and Davis (*19*) contains a summary and bibliography of the reported cases in animals.[3]

It is generally believed that both man and animals become infected by inhalation of spore-laden dust. Direct transmission of the disease from one individual to another does not commonly occur. There must, therefore, be some reservoir, such as the soil in which the fungus, *Coccidioides immitis*, may grow saprophytically, or a living host, either plant or animal, which provides for the propagation and dissemination of the etiological fungus.

In a preliminary paper (*10*) the occurrence of coccidioidomycosis in wild rodents was reported for the first time. In a second paper (*2*) the histology of the granulomatous lesions found in these animals was described. The chronic nature of the lesions suggested that rodents may constitute a natural reservoir of coccidioidomycosis which

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

[2] This investigation was aided by the Office of Indian Affairs, Department of the Interior, as part of a cooperative study. The authors are particularly indebted to Dr. J. D. Aronson and Dr. R. D. Cunningham for aid in furnishing facilities for field studies.

[3] A report of coccidioidomycosis in a dog in Canada appears in the Canadian Journal of Comparative Medicine for May 1941. No culture was isolated and the diagnosis was made from sections. An examination of stained sections kindly sent by Dr. P. J. G. Plummer revealed very numerous budding cells entirely typical of *Blastomyces dermatitidis*—convincing evidence that this was actually a case of American blastomycosis.

influences the distribution and epidemiology of the disease in man. Because of certain confusing aspects of the disease in rodents, a complete analysis of the data was not made in the preliminary reports. Two additional series of rodents have now been collected in the same area but during other seasons of the year. An adequate explanation of all observed phenomena cannot yet be offered, but a more complete report can now be made.

The study was made on the San Carlos Indian Reservation, San Carlos, Ariz., which lies outside the previously recognized endemic areas of the disease. This area was chosen because the studies of Aronson et al. (*1*) had furnished evidence that coccidioidomycosis is prevalent there, although not recognized clinically. This evidence was based on the demonstration that 92 percent of Indian school children tested reacted to the intradermal injection of coccidioidin. If a reaction to coccidioidin is accepted as evidence of a previous infection, his studies indicated further that in this area coccidioidomycosis is predominately a disease of early childhood. The mildness of the disease in young children and the fact that comparatively few persons first enter the area as adults may account in part for the peculiar circumstances of a population in which most individuals have probably at some time been infected, but in which no clinically apparent case of the disease has yet been recognized and proved. Cases of the disease were not available for study, and in order to obtain an explanation of the evidence supplied by skin-testing with coccidioidin, the etiological agent itself was sought in other hosts and in soil.

The difficulty of isolating *C. immitis* from soil is generally recognized, but Stewart and Meyer (*18*) and Davis et al. (*5*) have reported its isolation from this substrate at two sites in the San Joaquin Valley of California. Using the methods described by Stewart and Meyer, a search for Coccidioides was begun in the vicinity of San Carlos in July 1941 (*10*). By inoculating guinea pigs with suspensions of soil, *C. immitis* was isolated from 5 of about 150 samples taken in three widely separated sites and representing three different types of soil. The diverse characters of these soils, the relative infrequency with which the fungus has been isolated from soil in the San Joaquin Valley as well as at San Carlos, and a consideration of the virulence of the fungus for animals suggested that it may be primarily a pathogen of animals and that its spores are perhaps to be found in soil only after it has been contaminated by infected animals. The small rodents were first investigated because they are very numerous on the desert, and some species were known to be susceptible to experimental infection. During December 1941, and March, April, and July, 1942, 303 wild rodents were trapped and examined (table 1). They were obtained from five sites in an area surrounding San Carlos Hospital,

within a maximum radius of about 7 miles. These sites [4] were chosen because of the evident presence of rodents in considerable numbers, and, in the case of sites Nos. 1, 3, and 4, because the fungus sought had already been isolated from soil at these places. The five sites had in common the presence of certain species of rodents. Animals infected by fungi were obtained from all sites, and, so far as numbers were sufficient for analysis, the percentage of infected animals of a given susceptible species was about the same in all.

TABLE 1.—*Animals with fungus infections as determined by observation of lesions or isolation of cultures, tabulated according to species and time of collection*

	December series		March series		July series		Total		
	Trapped	Infected	Trapped	Infected	Trapped	Infected	Trapped	Infected	Percent infected
Perognathus baileyi (pocket mouse)	12	9	10	10	6	6	28	25	89
Perognathus penicillatus, P. intermedius (pocket mice)	12	11	73	57	11	10	96	78	81
Dipodomys merriami (kangaroo rat)	7	3	19	9	3	0	29	12	41
Citellus harrisi (squirrel)	1	1	8	7	1	0	10	8	80
Onychomys torridus (grasshopper mouse)	10	0	10	2	7	1	27	3	11
Peromyscus eremicus (deer mouse)	59	2	48	0	6	0	113	2	1.7
All species	101	26	168	85	34	17	303	128	42

It was not surprising to find that soil-dwelling rodents, living in areas where coccidioidomycosis is endemic, were in some cases infected. They live in soil from which *C. immitis* has been isolated and in an area where spores of the fungus must be often present according to evidence supplied by skin-testing of human residents. If, as has been generally supposed, the fungus grows in soil, one would expect few susceptible animals living in burrows in infested soil to escape infection after such intimate exposure. However, a surprising and wholly unexpected fact which casts some doubt on this simple explanation immediately became apparent. Deer mice (*Peromyscus eremicus*) are susceptible to a quickly fatal disease which follows intraperitoneal injection of spores in the laboratory, but none of 113 animals of this species caught in the field were infected with *C. immitis* and this fungus was isolated from only 1 of 27 grasshopper mice (*Onychomys torridus*). On the other hand, 15 percent of 124 pocket mice (*Perognathus baileyi, P. penicillatus,* and *P. intermedius*) and 17 percent of 29 kangaroo rats (*Dipodomys merriami*) trapped were infected by *C. immitis* (table 2). It is obvious from field data that the course of

[4] The sites were: (1) points on both sides of the Ash Creek Ranch road between 4 and 5 miles from the San Carlos Hospital; (2) in Seven Mile Wash, 2 to 4 miles from the hospital; (3) beside the road toward the tufa quarries, 1 to 3 miles from the hospital; (4) beside the Globe Road, 1.5 to 3 miles from the hospital; and (5) to the left of the road toward Coolidge Dam, about 7 miles from the hospital.

the granulomatous process produced in these rodents is not rapid enough to exterminate the species. Indeed, it may be that it does not materially reduce the population. Information on the latter point is not available except that many of the infected animals caught in March and April were pregnant or lactating females. Based on the above evidence of the relationship of pocket mice and kangaroo rats to this disease, it seems that these rodents may serve as an animal reservoir of coccidioidomycosis. The relationship of other species to this disease is more obscure. It is suspected that there is a seasonal variation in the distribution of the disease in rodents which has not yet been observed. It would not be surprising to find coccidioidomycosis occurring in epidemic fatal form among deer mice, for example, at another season of the year.

TABLE 2.—*Species of fungus isolated from rodents with and without observed lesions*

	Total trapped	Lesions	Only *C. immitis* isolated	Only *H. parvum* isolated	*C. immitis* and *H. parvum* isolated	Total animals
Perognathus	124	Seen	11	18	5	34
		Not seen	1	60	2	63
Dipodomys	29	Seen	3	1	2	6
		Not seen		5		5
Citellus	10	Seen		5		5
		Not seen				
Onychomys	27	Seen	1			1
		Not seen		1		1
Peromyscus	113	Seen				
		Not seen		2		2
Total	303		16	92	9	*117

*Cultures from 11 additional animals were contaminated and the pathogens, if present, were lost.

Of the 303 wild rodents trapped, 128, or 42 percent, had fungus infections (table 1). This number represents a selection. It was found early in the study that a mycosis was frequently present in certain species of rodents, and in subsequent studies attempts were made to trap as many as possible of these species. Lesions were observed in 55 animals and fungi were isolated from 46 of these. Fungi were also isolated from 71 in which no lesions were found (tables 1 and 2). The fungus isolated was *C. immitis* in 25 cases and an apparently related new fungus described below in 101 cases (table 2). These numbers include 9 cases in which both fungi were isolated from the same animal. The strains of *C. immitis* isolated were typical of the species in morphology and virulence for laboratory animals and need not be considered further at this time. The second fungus requires a description, a recital of the circumstances surrounding its isolation, and a discussion of its probable relationship to the disease in rodents and to *C. immitis*. This fungus has the generic characters

of *Haplosporangium* [5] Thaxter, and the name *Haplosporangium parvum* is proposed, the specific name being descriptive of the small size of the fungus in comparison with other species of the genus. To comply with the International Rules of Botanical Nomenclature, a short Latin diagnosis, prepared with the assistance of Mrs. Hope F. Norris, is given in addition to the more detailed English description.

Haplosporangium parvum sp. nov.: Mycelio tenue, albido, demum fulvello; conidiophoris gracilibus, indivisis vel ramosis, 0.5 x 1-10μ; conidiis globosis, rugosis vel levibus, 3–3.5μ diam.

Mycelium delicate, matted, white, becoming slightly brownish with age, aerial hyphae tufted in some strains which produce unusually large numbers of spores, sparse in other strains; hyphae averaging about 1μ, but rarely reaching 4μ in diameter, with few septa and these not necessarily related spatially to branches; conidiophores slender, simple and only 0.5–4μ long, or branched and 0.5 x 1-10μ, in most cases not tapering toward the apex; conidia spherical or subspherical, minutely spiny, becoming smooth, 3–3.5μ in diameter, containing one or more hyaline bodies.

Present as spherical nonbudding cells reaching a diameter of 14μ in lung tissue of Perognathus, Dipodomys, Citellus, and other rodents trapped in the vicinity of San Carlos, Ariz.

Haplosporangium parvum grows more slowly than *C. immitis*. On acid dextrose agar the colony appears first as a glabrous colorless disc, the hyphae forming a rather firm surface mat with an even border. As growth proceeds, a small tuft of white aerial hyphae appears at the center of the colony, and delicate aerial hyphae gradually appear over the rest of the colony (figs. 2a, c, e). The strains isolated vary considerably in colony characteristics (figs. 2a–f). Some always retain glabrous zones. Most become entirely covered with densely matted but delicate white aerial hyphae, becoming brownish with age, which may form a cottony layer as much as 6 or 8 mm. deep. On agar slants this extends up onto the sides of the culture tube. In some strains the colony surface becomes tufted with a suggestion of chalkiness (fig. 2b) and is cream colored even while still young, a condition usually associated with increased spore production. Such strains approach *C. immitis* in appearance, but the hyphae are always more delicate, and the microscopic morphology is different.

Microscopic.—The hyphae are delicate, most falling within the limits of 0.5–2μ and averaging about 1μ in diameter. Occasional hyphae reach a diameter of 4μ. Branching is frequent and the location seems to be independent of the location of cross walls. Septa are infrequent or difficult to demonstrate except in old hyphae, where the

[5] The authors are indebted to Dr. David H. Linder, curator of the Farlow Herbarium, Harvard University, for an opportunity to examine type material of Haplosporangium, and to discuss the fungus with him.

protoplasm is sometimes divided by pseudosepta and vacuoles in a manner suggesting the Phycomycetes. The spores are borne singly, or rarely in chains of two at the ends of hyphae or at the tips of short lateral conidiophores (figs. 3a–d and fig. 1).

Conidiophores are simple (0.5 x 1-10μ) or complex. The latter vary from short branches bearing two or more spores to longer branches bearing many lateral spores. A type of conidiophore frequently seen is shown in figure 1a. A swollen hyphal tip which would appear to have the potentialities of a spore proliferates by the formation of two or more branches which are terminated by conidia. Conidia are almost spherical except for a short tubular projection or stalk at the base. Many young spores are slightly flattened, the point of attach-

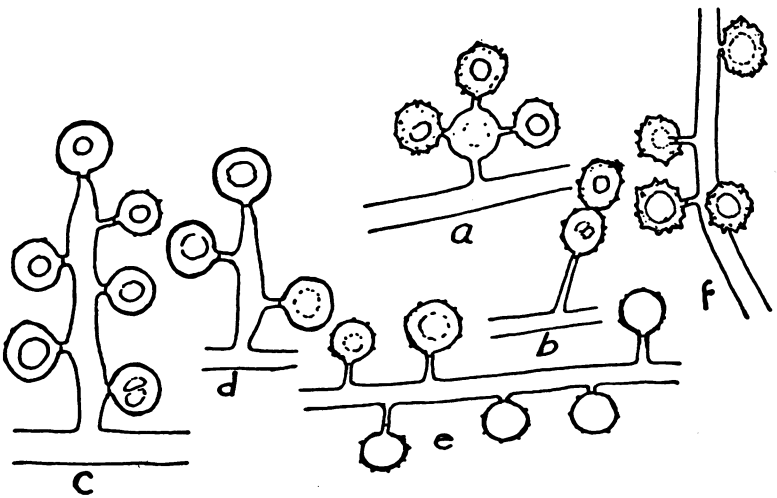

FIGURE 1.—Microscopic structure and production of conidia in *Haplosporangium parvum*.

ment to the conidiophore being on one flattened side. Spores average 3–3.5μ in diameter. Exceptional spores are clavate and much larger.

Spore formation is initiated by the development of a smooth spherical swelling at the tip of the conidiophore. As this swollen structure increases in size, rough markings, which under high magnification resemble spines, develop on the outer surface. In mature spores these markings are less conspicuous, and many old spores are quite smooth (fig. 1c). Formation of the spore is completed by the development of a wall across the conidiophore 0.25–0.5μ below the tip of the latter. The location of this wall well below the spherical portion of the spore produces the stipitate character of the spore already mentioned.

The manner of spore development in culture may be interpreted as an endogenous process within a sporangium. Shrinkage of the immature sporangium when placed in mounting fluid for examination

probably emphasizes the external markings. As the spore matures it increases in size, completely filling the space, and the sporangial wall disappears, or, more probably, adheres closely to the spore wall, the external markings being lost or partially obliterated. The structure can be defined as a monosporial sporangium or sporangiole, or more properly, a conidium. If this interpretation is correct, the fungus may be placed beside the three species of *Haplosporangium* Thaxter already described. Its spores also resemble the "stylospores" of Mortierella, furnishing additional evidence for a relationship to the Phycomycetes. It differs from the other species of Haplosporangium in size and shape of the conidiophore. *H. bisporale* Thaxter and *H. decipiens* Thaxter (*21*), isolated from dung, and *H. lignicola* Martin (*14*), isolated from rotten wood, are much larger and have tapering conidiophores.

Pathology.—The previous report (*2*) on the pathology of spontaneous coccidioidal granuloma was based on the lesions found in 9 of the first series of rodents trapped. Since then 121 animals from the second series have been examined histologically and of these nodular lesions were present in 20 pocket mice, 5 kangaroo rats, and 7 ground squirrels. In addition, the series included 7 animals with lesions which were not studied histologically.

From the first series the impression was gained that the lesions occurred more often in specific areas of the lungs. This impression, however, was not supported by the second and larger series of animals. In 25 mice and rats there were numerous lesions in the lungs of 8 (fig. 5g), many in 4, a few in 6, and from 1 to 3 lesions in the remaining 7 animals. When multiple nodules were present, they were diffusely distributed in all lobes. However, they were more often located peripherally than in the deeper part of the lung parenchyma.

Since the histology of the granulomas has been previously reported in detail (*2*), only their basic structure will be described here. In the mice and rats the lesions are formed of fusiform epithelioid cells irregularly disposed in central areas, but often showing concentric arrangement peripherally. In some nodules very large polygonal mononuclear cells form part or all of the central zones. This is particularly evident in the granulomas from the kangaroo rats. The nodules are well circumscribed and often have a peripheral zone of compactly disposed lymphocytes. Caseous or infrequently karyorrhectic necrosis was present in approximately 90 percent of the nodules examined. Fibrosis was noted in only two of the 20 lesions present in the first series, whereas in the second series 37 of 55 lesions showed slight to moderate fibrosis, or less often tufts or bands of sparsely cellular dense scar tissue. Calcification of the caseous material was observed in three lesions from the second series and

once in the first. In one of these nodules many of the fungus cells were also calcified.

Six early lesions were found in the lungs of two animals. These nodules were poorly circumscribed and formed of plump and short epithelioid cells growing within alveoli. They also showed many scattered neutrophils and a few minute to small foci of suppuration. In one of these lesions very large numbers of recently liberated endospores were seen. Groups of two or three were not infrequently present within phagocytes. One such early lesion was present in the first series.

The lesions of the seven ground squirrels were similar in all respects to the one described in the previous report. They were formed of large aggregations of mononuclear cells with ample cytoplasm and fewer irregularly scattered lymphocytes. In such lesions fungi were generally present in small numbers although in two, large clusters were present within giant cells, and in one a sporangium filled with faintly stained endospores was seen.

The fungus cells within granulomas showed considerable variation in number, size, depth of staining, and amount of cytoplasm. In some nodules fungi were present in very small numbers, whereas in a few 100 or more were counted in single 7μ sections.

As in the previous series endosporulating forms of the fungi were rarely observed, although it is obvious that the fungus present in at least some of the granulomas was *C. immitis*, since this fungus was isolated in culture from some of them. A belief that this suppression of maturation and endosporulation is a result of host influence is supported by the fact that when strains of *C. immitis* from these animals were inoculated into experimental animals endosporulating forms were regularly observed in the resultant lesions.

In the report describing the histology of the lesions, brief reference was made to the presence of fungus cells scattered in the lung parenchyma unrelated to granulomas as well as in lungs without granulomas. They have been found with equal regularity in animals of the second series. Although similar in some respects, most of these fungi show striking differences from those present in the nodules. These extragranulomatous fungi are generally spherical and have distinct, doubly contoured walls. Most of them have dense homogeneous deeply basophilic cytoplasm. In sections stained the proper depth for tissue nuclei, the cytoplasm of some of these fungi appears so dense that very little light is transmitted through them (figs. 5b and f). Many of these fungi show medium sized (one-third to one-half the diameter of the cell), central, less often multiple, small, fairly sharply marginated vacuoles. Even when such vacuoles are present, the surrounding or intervening cytoplasm is dense and homogeneous. It is believed that these deeply stained cells are those of *Haplosporangium*

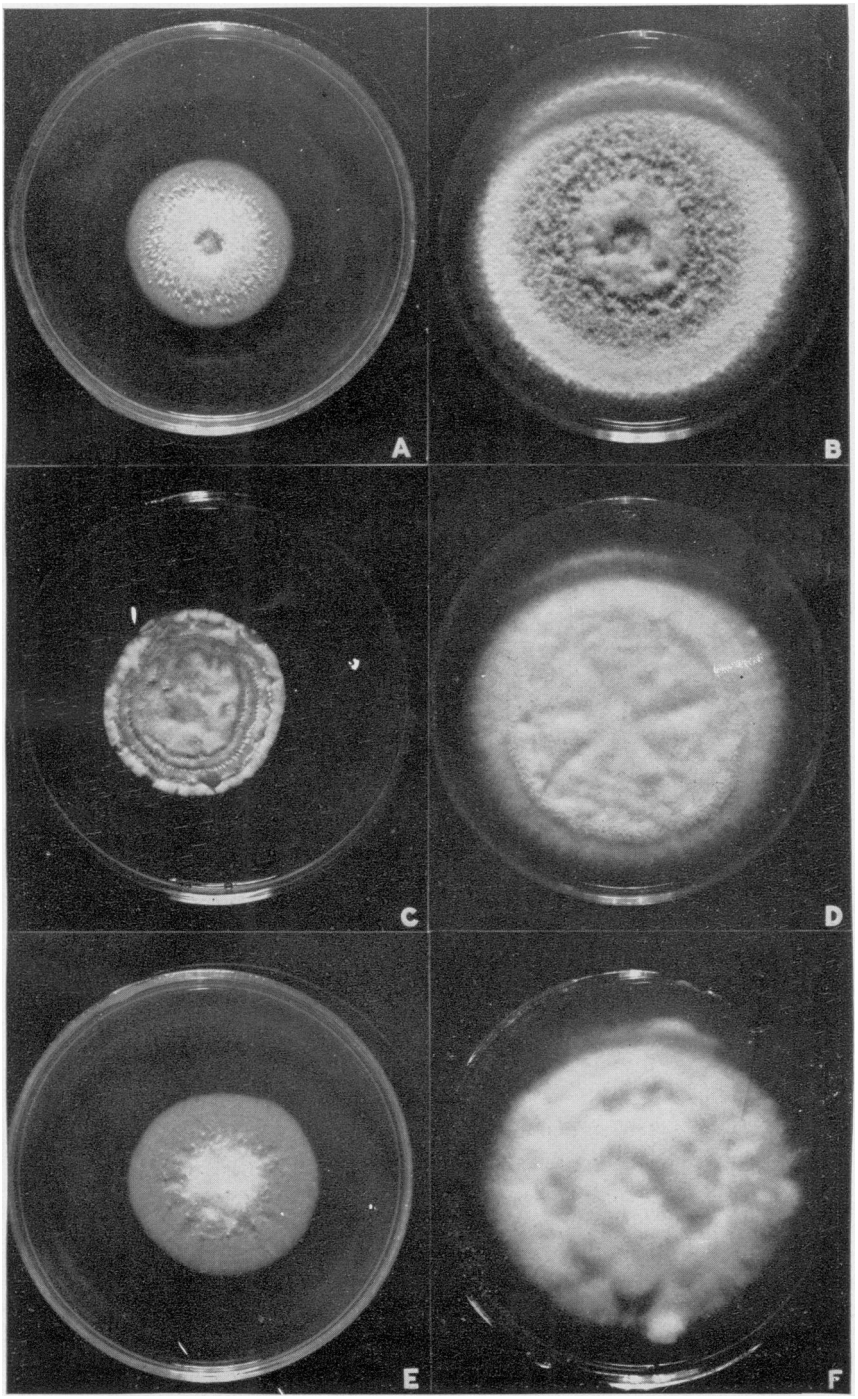

FIGURE 2.—Cultures of *Haplosporangium parvum* grown on acid dextrose agar at room temperature. A, C, and E, 2 weeks old; B, D, and F, 4 weeks old.

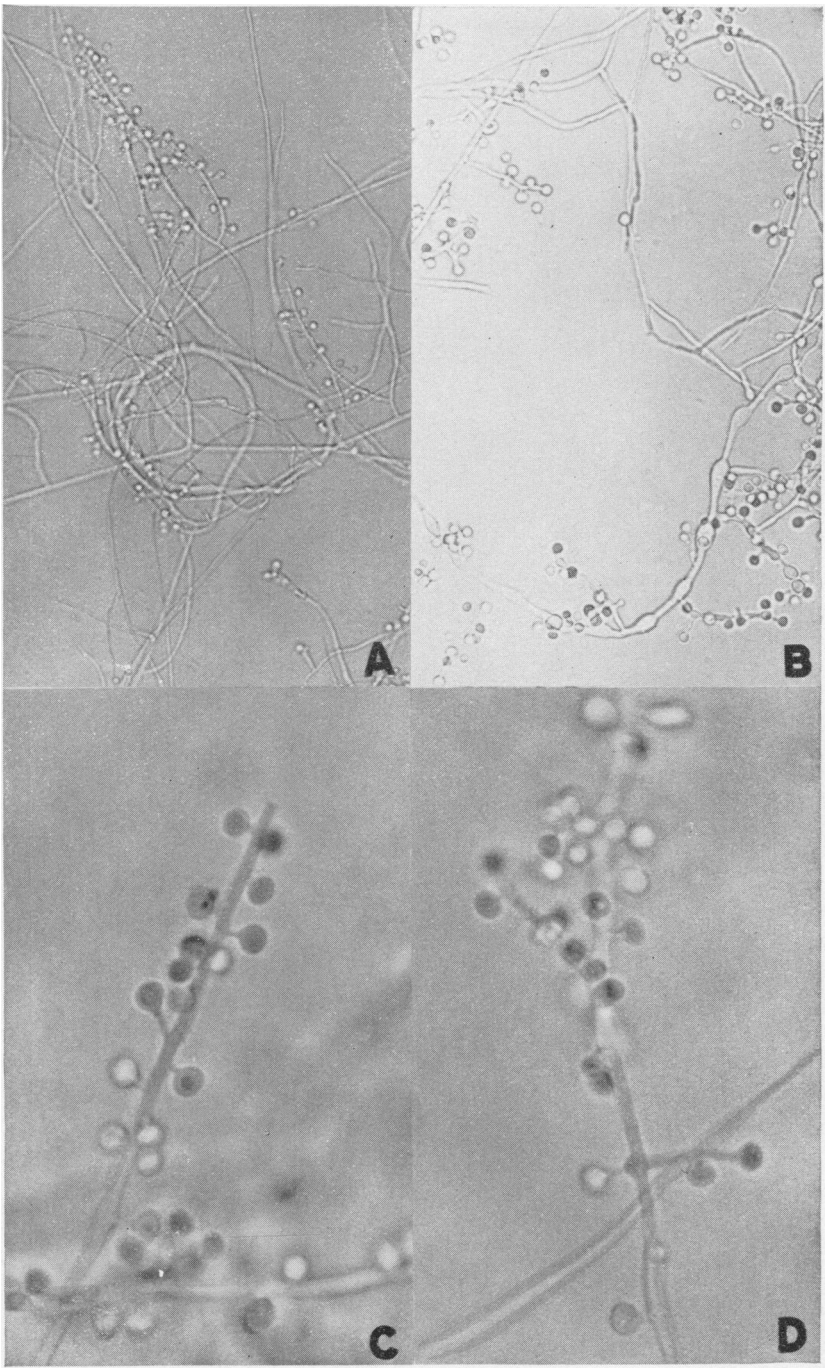

FIGURE 3.—Microscopic appearance of *H. parvum* in culture. A and B, ×350. C and D, ×1120.

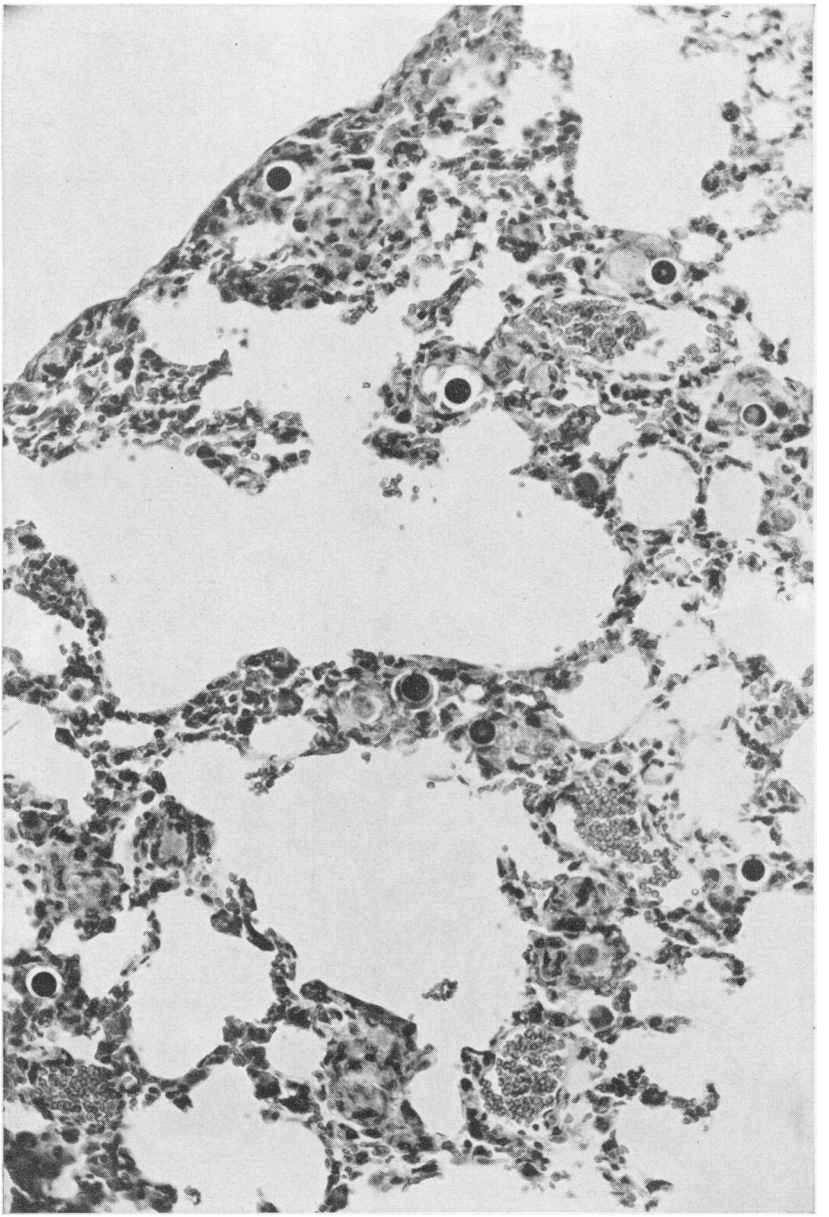

FIGURE 4.—Photomicrograph of mouse lung showing many fungus cells. Most are of the dense, intensely stained variety *(H. parvum)*. Some are enclosed in groups of large mononuclear cells. Romanowsky stain, ×300.

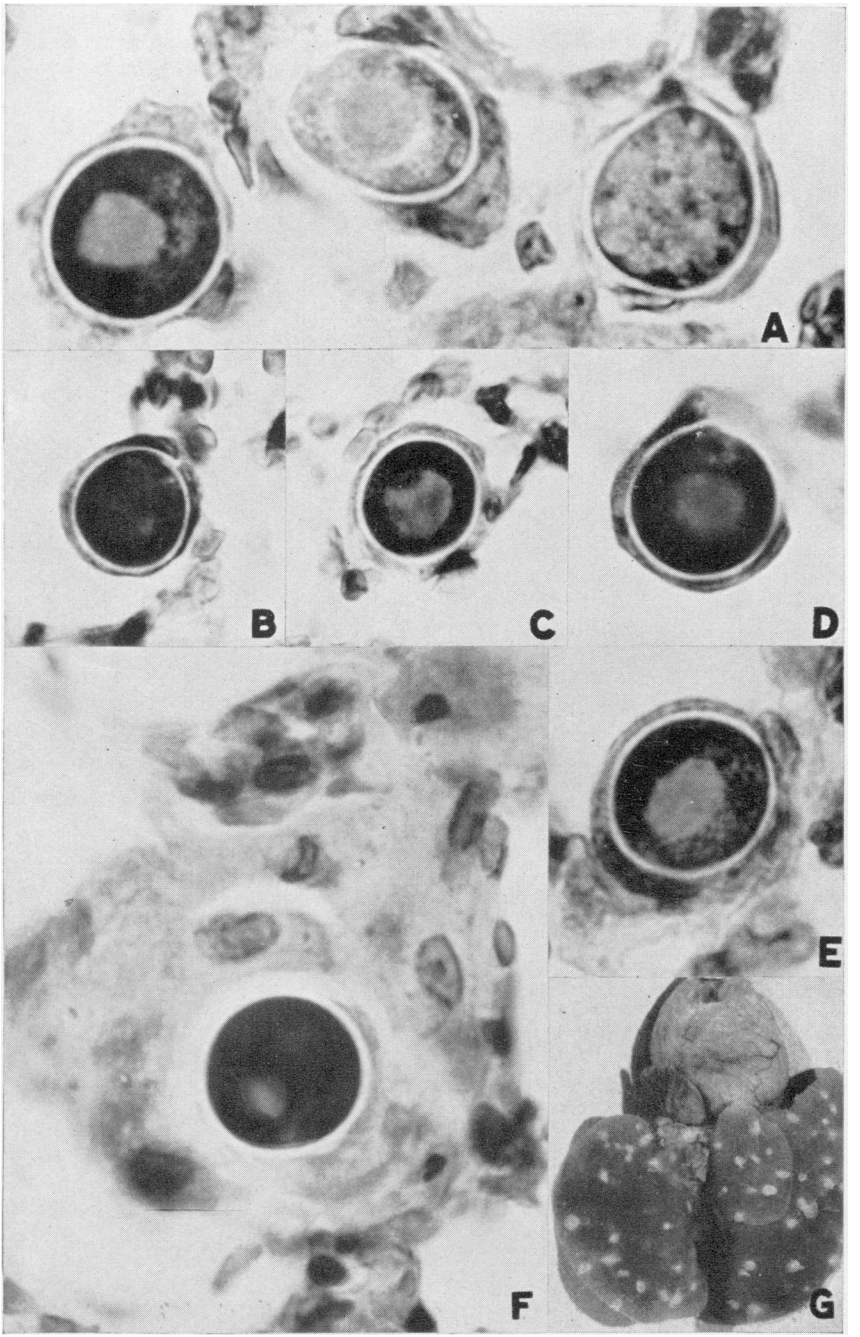

FIGURE 5.—A to F. Higher magnification of fungus cells shown in figure 4. Note the variation in density and depth of staining, and, in some, a central vacuole. These fungi measure from 10 to 14 microns in diameter. There is practically no reaction to the presence of the fungi except in F. G. Photograph of mouse lung showing numerous nodular lesions. Romanowsky stain.

parvum and they shall be referred to as such. The cytoplasm of a very small percentage of these fungus cells is much less deeply stained. These individuals cannot be differentiated from *C. immitis* and are not unlike some of the fungi present within granulomas.

In contrast to the wide range in size of the intra-granulomatous fungi, *H. parvum* usually varied only between 10 and 13μ in diameter. This was true for the fungi from the same as well as those from different animals. In most lungs an occasional fungus cell measured less than 10μ, and in an occasional animal such cells represented approximately one-third of the total number seen. Fungi larger than 14μ were rarely observed.

The lack of or relatively slight reaction to the presence of *H. parvum* was a striking and surprising finding. Most fungi were within or surrounded by from 1 to 3 or 4 large angular or polygonal mononuclear cells. Usually the fungus cells were enclosed in a narrow rim of cytoplasm in which from 1 to 3 long curved nuclei were visible (figs. 5b to e). Such enclosed fungi were fixed to alveolar walls and made knobby protrusions into alveolar spaces. In some cases, particularly in lungs in which fungi were numerous, the mononuclear cell aggregations measured up to 50μ in diameter. Not infrequently more than one fungus cell was present in such a nodule and rarely a giant cell of the fusion type was present. One, rarely more, of these cells was seen in the walls of a very few granulomas.

The frequency with which *H. parvum* occurred in a given area of lung varied considerably in different animals. In a few, only one fungus cell was found after study of two cross sections of both lungs, whereas in one animal 60 were counted in a triangular lung section measuring 3 mm. on a side and 7μ thick (fig. 4). *H. parvum* was found in the sectioned lung of 74 animals. Taking the above sized section of lung (approximate) for comparison, there were 11 animals which showed 6 or more fungi per section, 9 animals with 3 to 5 fungi, and in 54 cases there were 2 or fewer fungi per section. These fungi were no more numerous (average) in lungs showing granulomas than in lungs without such lesions.

It is clear that *H. parvum* is not a contaminant. Between 24 and 48 hours after pieces of infected lung are placed on acid dextrose agar slants one can see with a 10X lens that a fungus has begun to grow. This is manifested by minute, barely visible hyphae which appear over all or most of the surface of the lung tissue. Under the same circumstances, a contaminant, which is usually present in or on the fresh lung tissue as one or a few spores, first appears as a single colony which makes a considerable growth before invading the rest of the inoculum. Evidence on this point is not valid unless a very careful examination is made with a good lens before growth is visible to the naked eye, and it may be misleading if Mucor is present as a

contaminant. Other fungi, including species of Penicillium, Aspergillus, and Actinomyces, appeared in some cultures but were readily recognized as contaminants. Further, *H. parvum* was frequently isolated from certain species of rodents and only rarely from other animals. An analysis of the figures which follow supply further evidence that *H. parvum* is not a contaminant.[6] This fungus was isolated from 63 of 74 animals in which *H. parvum* was seen in tissue sections. Of 61 animals in which no fungi were seen microscopically, *H. parvum* was isolated from only 11. In view of the fact that a much greater bulk of lung was planted on culture medium than was studied histologically, it is not surprising that certain cases were negative by histological examination but positive by culture.

Finally, although conspicuous progressive lesions have not yet been produced experimentally in animals, the fungus has been recovered in culture from white mice several months after intranasal inoculation, and it produces in these animals the type of microscopic lesion with which it is associated in wild rodents trapped in the field. Koch's postulates have therefore been fulfilled with respect to the microscopic lesions. The granulomatous lesions have not yet been experimentally reproduced. The relationship of *H. parvum* to granulomas is not yet clear. Evidence for and against an etiological relationship is discussed in the following paragraphs.

Can the isolation of this fungus be explained by postulating the existence of a widespread new fungus infection of rodents unrelated to coccidioidomycosis? Some observations suggest this possibility. *H. parvum* appears to be associated most frequently with the type of fungus cell which is found outside grossly visible lesions, as described above (table 2), and in this respect differs from *C. immitis*. In most of the individual animals from which both *C. immitis* and *H. parvum* were isolated in culture, *C. immitis* was obtained from a nodular lesion and *H. parvum* from grossly normal lung tissue.

On the other hand, in 24 of the 117 animals (table 2) *H. parvum* only was isolated from nodules. The isolation of *H. parvum* in cultures made from macroscopic lesions freed so far as possible from adjacent lung tissue strongly suggests that *H. parvum* may be related etiologically to granulomas. In these cases when the lesion was crushed and spread over the surface of the agar slant *H. parvum* came up in pure culture at very numerous points on the inoculated agar surface. These nodules were grossly indistinguishable from those yielding *C. immitis*, and a careful histological study of pathological material did not permit a separation of granulomas into two categories based on etiology. In making cultures, if both fungi had been present in the inoculum, one would expect *C. immitis* to be isolated

[6] *C. immitis* was also isolated from a number of these animals.

invariably and *H. parvum* to be lost unless special care was taken to separate and save it. The former grows more rapidly and produces more spores than the latter, thus increasing the relative abundance of separate viable elements of *C. immitis* in a sample of mixed inoculum. In cases where a mixture of the two fungi was actually known to be present in a culture, great difficulty was experienced in isolating *H. parvum* from the mixture, while *C. immitis* was readily isolated. Further, in considering the species distribution it seems improbable that two separate mycoses occur commonly in this area in pocket mice and kangaroo rats and very rarely in deer mice which live in precisely the same terrain and occupy adjacent burrows.

Additional evidence indicating a probable relationship of *H. parvum* to coccidioidomycosis is furnished by the use of a skin test. A skin testing antigen (C. 2) was prepared from *H. parvum* following the procedures used in making coccidioidin. With the assistance of Mrs. Mabel C. Head, field nurse in the Indian Service, this material was used in skin testing Indian school children at San Carlos and at Fort Apache, an adjacent area in which only about 15 percent of the school children react to coccidioidin (*1*). The results of this testing are shown in table 3. The percentage of coccidioidin reactions observed in the San Carlos area during this study is lower than reported by Aronson et al. (*1*), because the second test with a larger dose of coccidioidin was not made. The fact that reactions to the two testing materials did not run exactly parallel is probably due in part to a lower potency of C. 2. This lot of material was prepared from cultures which grew only 6 weeks before being processed. In spite of the discrepancies appearing in table 3, the fact that 29 of 33 reactions to C. 2 occurred in individuals who also reacted to coccidioidin probably indicates an antigenic relationship, although the possibility of a dual sensitivity must be considered. This probable antigenic relationship, the circumstances under which *H. parvum* was isolated from rodents, resemblances of the two fungi in lung tissue, and the results of laboratory investigations lead to the conclusion that *H. parvum* is genetically related to *C. immitis* and in some manner to coccidioidomycosis.

TABLE 3.—*Reactions of school children in two contrasting areas, injected intradermally on one arm with lot No. 9 of coccidioidin (Dr. C. E. Smith), and on the other arm with C.2*

	125 children tested at San Carlos		123 children tested at Fort Apache	
	Positive to coccidioidin 1:1000	Negative to coccidioidin 1:1000	Positive to coccidioidin 1:1000	Negative to coccidioidin 1:1000
Positive to C.2 1:100	27	2	2	2
Negative to C.2 1:100	65	31	5	114

The preliminary skin testing studies indicate a necessity for further investigation of a question which was constantly in mind throughout the studies made in Arizona; viz, can the prevalence of skin hypersensitivity to coccidioidin in a population in which clinically apparent coccidioidomycosis is relatively infrequent be due to exposure to a fungus less virulent than *C. immitis*, but possessing an antigen in common with it? *H. parvum* seems to fulfill some of the specifications of such a fungus. It is relatively avirulent for experimental animals; it is present in an animal reservoir in at least one area in which Coccidioides also is known to be present, and residents of that area react to the intradermal injection of an antigen prepared from it. Skin hypersensitivity to coccidioidin has not yet been experimentally produced by *H. parvum*, however. Variability of fungi appearing either spontaneously or in response to some external stimulus such as radiation is a frequently observed phenomenon. It is possible that *H. parvum* may be a mutant of *C. immitis* which arises under desert conditions or is induced when spores of *C. immitis* are inhaled by certain relatively resistant animal hosts such as pocket mice.

If despite great morphological differences in culture, *H. parvum* is genetically related to *C. immitis*, it provides for the latter a hitherto unrecognized point of attachment to the Phycomycetes. It may be pointed out further that there is a remarkable resemblance in culture between *H. parvum* and two other pathogenic fungi, *Blastomyces dermatitidis* and *Histoplasma capsulatum*. The discovery of this fungus therefore supplies evidence which may indicate a closer relationship than has been previously recognized between the etiological agents of coccidioidomycosis, blastomycosis, and histoplasmosis, and between these Fungi Imperfecti and the Phycomycetes. A consideration of these points will be presented in a later paper.

SUMMARY

Typical strains of *Coccidioides immitis* were isolated from the lung in 15 percent of pocket mice and 17 percent of kangaroo rats trapped in the vicinity of San Carlos, Ariz. The histology of the lung lesions found in these rodents and the fungi present in them are briefly described. The frequency of the infection and the character of the lesions support a suggestion that rodents may constitute a reservoir of coccidioidomycosis important in the distribution and epidemiology of the disease in man. *Haplosporangium parvum* n. sp. was isolated from 101 of 128 rodents with or without grossly visible lung lesions and *C. immitis* was isolated from 25. These numbers include 9 animals from which both fungi were isolated. *H. parvum* was most often found in minute pulmonary lesions reaching 50μ in diameter and consisting of aggregations of large coherent mononuclear cells. This type of lesion was reproduced in white mice inoculated intranasally,

and *H. parvum* was isolated from these animals several months after inoculation. Evidence is presented which suggests that this fungus is also sometimes etiologically related to granulomas indistinguishable from those produced in rodents by *C. immitis*.

REFERENCES

(*1*) Aronson, J. D., Saylor, R. M., and Parr, E. O.: Relationship of coccidioidomycosis to calcified pulmonary nodules. Arch. Path., **34**: 31–48 (1942).

(*2*) Ashburn, L. L., and Emmons, C. W.: Pathology of spontaneous coccidioidal granuloma in wild rodents. Arch. Path. In press.

(*3*) Beck, M. D.: Occurrence of *Coccidioides immitis* in lesions of slaughtered animals. Proc. Soc. Exper. Biol. Med., **26**: 534–536 (1929).

(*4*) Beck, M. D., Traum, J., and Harrington, E. S.: Coccidioidal granuloma: occurrence in animals. J. Am. Vet. Med. Assoc., **78**: 490–499 (1931).

(*5*) Davis, B. L., Smith, R. T., and Smith, C. E.: An epidemic of coccidioidal infection (coccidioidomycosis). J. Am. Med. Assoc., **118**: 1182–1186 (1942).

(*6*) Dickson, E. C.: Coccidioides infection. I. Arch. Int. Med., **59**: 1029–1044 (1937).

(*7*) ——— "Valley fever" of the San Joaquin Valley and fungus Coccidioides. Calif. Western Med., **47**: 151–155 (1937).

(*8*) ——— Primary coccidioidomycosis. Am. Rev. Tuberc., **38**: 722–729 (1938).

(*9*) ——— and Gifford, M. A.: Coccidioides infection (coccidioidomycosis). II. The primary type of infection. Arch. Int. Med., **62**: 853–871 (1938).

(*10*) Emmons, C. W.: Isolation of Coccidioides from soil and rodents. Pub. Health Rep., **57**: 109–111 (1942).

(*11*) Farness, O. J.: Coccidioidal infection in a dog. J. Am. Vet. Med. Assoc., **97**: 263–264 (1940).

(*12*) Gifford, M. A.: Coccidioidomycosis. Annual Report, Kern County Dept. Public Health, 1938–1939.

(*13*) Giltner, L. T.: Occurrence of coccidioidal granuloma (oidiomycosis) in cattle. J. Agric. Res., **14**: 533–541 (1918).

(*14*) Martin, G. W.: New or noteworthy fungi from Panama and Columbia. I. Mycologia, **29**: 618–625 (1937).

(*15*) Smith, C. E.: Epidemiology of acute coccidioidomycosis with erythema nodosum. Am. J. Pub. Health, **30**: 600–611 (1940).

(*16*) ——— Parallelism of coccidioidal and tuberculous infections. Radiology, **38**: 643–648 (1942).

(*17*) ——— and Baker, E. E.: A summary of the present status of coccidioidal infection. Weekly Bull. Calif. Dept. of Public Health, **20**: 113–115, 117–118 (1941).

(*18*) Stewart, R. A., and Meyer, K. F.: Isolation of *Coccidioides immitis* (Stiles) from the soil. Proc. Soc. Exp. Biol. Med., **29**: 937–938 (1932).

(*19*) Stiles, G. W., and Davis, C. L.: Coccidioidal granuloma (coccidioidomycosis). J. Am. Med. Assoc., **119**: 765–769 (1942).

(*20*) Stiles, G. W., Shahan, M. S., and Davis, C. L.: Coccidioidal granuloma in cattle in Colorado. J. Am. Vet. Med. Assoc., **82**: 928–930 (1933).

(*21*) Thaxter, Roland: New or peculiar Zygomycetes. No. 3. Blakeslea, Dissophora, and Haplosporangium, nova genera. Bot. Gaz., **58**: 353–366 (1914).

CHAULMOOGRA OIL IN THE TREATMENT OF LEPROSY

By G. W. McCoy, *Medical Director (Retired), United States Public Health Service*

There is a widespread belief among the members of the medical profession that chaulmoogra oil and its derivatives are valuable—specifically curative agents—in the treatment of leprosy. This is in marked contrast with the views expressed by many experienced

students of the disease especially when the subject is discussed privately. My own observations have led me to the conclusion that the oil and its derivatives are of little or no curative value, and that the unpleasant side effects probably outweigh any advantage to the patient that might possibly accrue from their use. I am not prepared, however, to go so far as Lara (*1*) who expressed the opinion that unless cases are carefully selected great harm may be done and even that death may be hastened by unwise treatment. Lara, whose experience has been wide, believes that the oil is of great value in suitable cases. Of course, many therapeutic agents are potentially harmful if improperly used but this alone is not enough to condemn them.

I can no better illustrate the faith some teachers of medicine or therapeutics must have in the value of chaulmoogra oil and its derivatives than to relate the experience I have had with young medical graduates who have taken the examinations of the National Board of Medical Examiners. In response to a question involving a comparison of certain features of tuberculosis and leprosy, occasionally a candidate would state in effect that as a last resort in a case of doubtful diagnosis a therapeutic test might be tried using chaulmoogra oil. If the patient recovered, the diagnosis of leprosy was established.

The confidence some physicians have in the efficacy of the oil is demonstrated by the following experience. I was studying the records on leprosy in a hospital, in an endemic focus where the disease is encountered perhaps two or three times each year. One patient had been under observation for over 2 years without a definite diagnosis. In February 1937 an ulnar nerve was reported as being enlarged and then suspicion of leprosy was aroused. The following note in the history is found under a May 1937 date, "evidence of leprosy sufficient to warrant a therapeutic test with chaulmoogra oil." This note was made by, or at the direction of, a physician who had had considerable experience with the diagnosis and treatment of leprosy.

A recently published work (*2*) would make treatment of leprosy with chaulmoogra oil or its derivatives an important feature in a campaign for the suppression of the disease. This would be logical provided the drug exerted a definitely curative effect—as, for example, the arsphenamine compounds do in syphilis.

Chaulmoogra oil is so well established that in addition to being favorably referred to in practically all textbooks dealing with the treatment of the disease, we find it in the U. S. Pharmacopoeia XI.

It is understood that it is the general policy of pharmacopoeial authorities to recognize not only remedial agents of established value but also those in extensive use.

There are two publications of the American Medical Association

designed to aid the physician in the selection of therapeutic agents—New and Non-Official Remedies (1941) and Useful Drugs (1940). The former states (p. 188) that the bulk of the evidence indicates that chaulmoogra oil is of value though not having specific, curative properties. The latter, under the heading of "Actions and Uses" of chaulmoogra oil, states (p. 73), "It is of some value in affording relief of symptoms, but it does not effect a cure"—a statement at variance with the views of those who advocate the use of the oil and of those who regard it as valueless.

To illustrate the attitude of certain authorities, Stanley (*3*) and his associates introduce a chemical and a bacteriological study with the statement, "Chaulmoogra oil and related compounds have been used as specifics against leprosy for several centuries." These authors quote from a paper published by the League of Nations on the prophylaxis of leprosy (*4*) which includes this statement, "Treatment by chaulmoogra oil and its derivatives is efficacious, however we may explain its action."

The published reports on the use of the oil are so nearly unanimously in its favor that it seems unnecessary to cite many individual authorities other than those already mentioned. I will present evidence contrary to what has been the generally accepted view, not only of clinical observers but also of some research workers. I will make no attempt to present a full review of the literature, but will cite what seem to me to be the most significant reports.

According to many authors chaulmoogra oil has been used in leprosy for centuries. My own somewhat cursory review of the literature has gone back no further than publications in the present century with a single exception of the work of Hansen and Looft (*5*) who treated five cases (three nodular and two anaesthetic) for from 8 months to 1 year and sum up their experience with the words, "Results were as with other remedies, nil." It is recognized that this opinion is seriously, possibly fatally, defective as evidence because of the small number of cases, but it is thought worth presenting on account of the standing of the authors who were scientifically second to none among the students of the disease of almost half a century ago.

McCoy and Hollmann (*6*) reported on the use of the oil in 16 cases, 10 of which were recorded as improved, only 1 to the extent that acid fast bacilli could not be found. These authors called attention to some unpleasant consequences that occurred in their small series but concluded that the oil is helpful in many cases—perhaps the majority.

A review of this experience by the senior author after 25 years have elapsed leaves him with the opinion that not enough consideration

was given to the natural evolution of leprosy, i. e., the tendency of many cases to improve spontaneously, and to the extreme meagerness of the data, which were insufficient for even the modest conclusion that was drawn. Indeed, the observations of the quarter of a century that has passed have left the senior author with the very definite impression that chaulmoogra oil and its derivatives are of doubtful value in the treatment of leprosy.

Renewed interest in the treatment of leprosy with chaulmoogra oil came in 1920 with the report from Hawaii of McDonald and Dean (*7*). These authors appear to have been well impressed with various therapeutic agents in the treatment of the disease since they say, "Of the long list of drugs and remedies used against leprosy by various authorities we have nothing derogatory to state" and assert that they have great faith in strychnine. They also say that arsenous acid and Gurjun oil and several other remedies have maintained their good reputation. These authors used the derivatives of the oil called ethyl esters, chiefly by hypodermic injection. These preparations had been studied about 20 years earlier from the chemical point of view. They report on their clinical experience over a period of 18 months, ended December 31, 1919, during which time over 25 percent of the cases treated apparently became arrested or cured—in the authors' words, "apparently clinically and bacteriologically free from the disease."

In a later paper (May 1921) (*8*) even better results were reported by the same observers, 50 percent of the patients having improved and been paroled. Up to the time of publication no relapse had been reported. So favorably impressed were these authors with their results that in the same paper they reported that the use of the remedy at the Leprosy Receiving Station in Honolulu had been changed from a voluntary basis to one in which the patient was automatically placed on the treatment as soon as he was admitted to this institution.

The very brief summary of the work of McDonald and Dean is referred to not solely to contrast with the results of others working at the same institution but to illustrate how misleading may be conclusions based on meager data and short periods of observation.

Probably a clue to the reason for reporting favorable results during a certain period in Hawaii is to be found in the observations of the late Surgeon M. H. Neill of the United States Public Health Service (*9*) who also worked at the Leprosy Receiving Station in Honolulu during a period when the mixed ethyl esters of chaulmoogra oil were used for routine treatment. A very large proportion, 70.97 percent of cases bacteriologically negative on admission, were paroled, while but 15.23 percent of cases microscopically positive on admission were paroled. All students of the disease recognize the more favorable

outlook for the bacteriologically negative cases, without respect to treatment.

The highly favorable experiences of McDonald and Dean were not duplicated by another investigator working at the same institution from which these excellent results were reported a few years earlier.

Wayson (*10*), at the conclusion of a particularly careful study of the influence of treatment in Hawaii, has this to say: "The use of chaulmoogra oil and its derivatives in Hawaii for 10 years has not been attended by results which indicate that they have any specific therapeutic value, and any effect they may have remains to be determined."

Morrow and his associates at San Francisco (*11*) point out that the esters were patented in Germany in 1909 by Ludwig Taub of Elberfeld and put on the market as antileprol. These observers treated 21 cases from 3 to 18 months over an average period of 8 months. The results were as follows: 2 died, 3 became worse, 9 showed no improvement, 2 were markedly improved, 3 slightly improved, and 2 absconded; none became bacteriologically negative. Obviously these results are what might be expected in any group of lepers without reference to special treatment.

Tomb (*12*) quotes in part the report of the Leprosy Commission, League of Nations Health Organization (1930), "There is no proof that general dietetic treatment, plus chaulmoogra oil, yields better results than general dietetic treatment alone. No conclusive evidence exists of the efficiency of chaulmoogra oil as such."

In the International Journal of Leprosy Dr. H. P. Lie (*13*), the distinguished Norwegian authority on leprosy and worthy successor of Danielssen and Hansen, published a paper on the curability of leprosy. This article is valuable from several points of view but the only one in which I am interested now is Dr. Lie's review of Danielssen's experience with chaulmoogra oil which is put in the following words, "This in the form of ol. gynocardia odoratae seemed to have no healing qualities; in fact Danielssen found it detrimental." This appraisal is what might have been expected of a master clinical observer, an observer who had the ability to give us about the middle of the last century descriptions of leprosy and a clinical classification valuable to this day. Danielssen's experience covered approximately the latter half of the last century (1849–95) and I am sure that objection will be made that he dealt only with advanced cases unfavorable from the therapeutic point of view. Dr. Lie furnishes some data which have a bearing on this by showing that 11.8 percent of all cases resulted in complete improvement and a further 9.1 percent resulted in incomplete improvement, a total of 20.9 percent that may be regarded as having improved. But there is further evidence that the cases dealt with by Danielssen were not too unfavorable—of 93 regarded as cures but 14 returned as relapsed. To illustrate further that the clinical types of that day

responded much as they do today, it was recorded that 3.5 percent of nodular cases could be regarded as cured while of the more favorable maculo-anaesthetic type 29.4 percent achieved that happy termination The summary of necropsy findings as presented by Dr. Lie supports the view that when the Norwegian medical authorities recorded a case as cured it was cured and usually remained so. It is to be kept in mind that most of the cases dealt with in those days had well-marked clinical manifestations, since much of the experience was before the days of microscopic diagnosis.

Recently I had occasion to restudy the subject with special reference to ethyl chaulmoograte. Being somewhat familiar with the published evidence and knowing well the generally favorable nature of such evidence I sought the opinions of four special students of the disease. These physicians have had from 8 to nearly 20 years of experience in the treatment of leprosy, each having had continually under observation from approximately 100 to 400 patients. It goes without saying that they are clinicians in whose judgment and open-mindedness I have great confidence and who probably would not feel that their time and energies would be profitably employed in preparing their present views for publication. The following quotations express the opinions of the four leprologists referred to above.

"I really feel that chaulmoogra oil, or its derivatives, is of little or no value in the treatment of leprosy. I should like to qualify that by saying that in lieu of anything better to offer, we deem it necessary to use it, although I do not encourage anyone to take it, especially by mouth, as it, I am sure, does a great deal of harm to many who take it orally due to its being such a gastric irritant."

"In reply to your direct question concerning chaulmoogra oil, as appended to your letter of January 16, my opinion is that chaulmoogra oil or its derivatives have not been shown to be of specific value in the treatment of leprosy.

"I make the above statement with full knowledge that many patients seem to have improved while under the treatment with this drug. However, others have not shown improvement and additional patients have shown equal improvement under the administration of other oils or esters, and under a regimen of personal and institutional hygiene without the administration of any oil."

"In my experience, chaulmoogra oil has been disappointing when viewed from results obtained over a long period. In a small percentage of cases it seems to have been of value. In most cases its value is uncertain, especially if the case is followed over a period of 5 to 10 years. It certainly has no specific action in leprosy, nor has its value, in my experience, even approached that which is usually accorded it by lay individuals and by some scientific workers."

"I have never noted any unquestionable evidence that ethyl chaulmoograte is of any value in the treatment of leprosy. During the four years that I was in charge of leprosy investigations in Hawaii, none was employed. During this period just as many patients became arrested and were removed from segregation as during previous periods when the ethyl esters were employed extensively, between twenty-five and thirty percent of all admissions becoming arrested."

REFERENCES

(1) Lara, C. B.: Progress of leprosy treatment at Culion Leper Colony. J. Philippine Islands Med. Assoc., **10**: 469–480 (1930).
(2) Rogers, Sir Leonard, and Muir, Ernest: Leprosy. 2d ed. rev. Williams and Wilkins, Baltimore, Md., (1940).
(3) Stanley, W. M., and others: Bacteriological action of certain synthetic organic acids towards *Mycobacterium leprae* and other acid-fast bacteria. J. Pharmacol. and Exper. Therap., **45**: 121–162 (1932).
(4) The Principles of the Prophylaxis of Leprosy. First general report of the Leprosy Commission, Geneva, April 1931. Series of League of Nations Publications III. Health. 1931. III. 2.
(5) Hansen, G. A., and Looft, C.: Leprosy in its Clinical and Pathological Aspects. Translated by Norman Walker. J. Wright & Co., Bristol, 1895.
(6) McCoy, G. W., and Hollmann, H. T.: Studies upon leprosy. XXVIII. The chaulmoogra oil treatment of leprosy. Pub. Health Bull. No. 75, 1916. Pp. 3–10.
(7) McDonald, J. T., and Dean, A. L.: The treatment of leprosy with especial reference to some new chaulmoogra oil derivatives. Pub. Health Rep., **35**: 1959–1974 (1920).
(8) McDonald, J. T., and Dean, A. L.: Constituents of chaulmoogra oil effective in leprosy. J. Am. Med. Assoc., **76**: 1470 (1921).
(9) Annual Report of the Surgeon General, U. S. Public Health Service, 1927. Pp. 36–39.
(10) Wayson, N. E.: Observations on the treatment of leprosy in Hawaii. Pub. Health Rep., **44**: 3095–3110 (1929).
(11) Morrow, H., Walker, E. L., and Miller, H. E.: Chaulmoogra oil derivatives in leprosy. J. Am. Med. Assoc., **79**: 434–440 (1922).
(12) Tomb, J. W.: Chaulmoogra oil and its derivatives in treatment of leprosy. J. Trop. Med., **36**: 201 (1933).
(13) Lie, H. P.: Curability of leprosy. Internat. J. Leprosy, **3**: 1–22 (1935).

ANTRICOLA NEW GENUS, *AMBLYOMMA GERTSCHI* NEW SPECIES, AND NOTES ON *IXODES SPINIPALPIS* (ACARINA: IXODOIDEA) [1]

By R. A. Cooley, *Senior Entomologist*, and Glen M. Kohls, *Associate Entomologist, United States Public Health Service*

In recent studies of the Argasidae we have found that *Ornithodoros coprophilus* McIntosh and *O. marginatus* Banks require the erection of a new genus.

The two species included are known only from bats or bat retreats.

Antricola, new genus

Argasidae having dorsal body wall flattened and marginated; below the flattened dorsum the body convex and deep. Integument semi-translucent and the surface smooth, shining, and with tubercles. Discs absent on the venter. Mouth parts adapted for quick feeding and not for clinging to the host; hypostome convex ventrally, concave dorsally, and lacking effective denticles; chelicerae large and effective. Anal ring large. Eyes absent. Eggs small and the larvae small with bulbous pulvillae in place of claws.

[1] Contribution from the Rocky Mountain Laboratory of the Division of Infectious Diseases, National Institute of Health.

Genotype, *Ornithodoros coprophilus* McIntosh, 1935

In reviewing several lots of ticks recently sent from the American Museum of Natural History, New York, by Dr. W. J. Gertsch, associate curator of spiders, a single male specimen was found which represents a previously undescribed species and is here named for Dr. Gertsch.

Amblyomma gertschi n. sp.

MALE

Body.—Length 5.75 mm.; width 4.4 mm. Widest behind.

Scutum.—With marginal groove absent, punctations numerous, unequal, absent in four small isolated areas on each side and in a median posterior patch, which is mildly elevated giving an irregular surface. Punctations present also on the festoons which are longer than broad. Cervical grooves deep, short, divergent anteriorly. Ornamentation in scattered pattern as shown in the figure. Eyes flat and not easily seen.

Capitulum.—Broad, punctate, and with the cornua long; width, 0.84 mm. Palpi moderate in length, broader distally and with a few curved hairs; postero-dorsal point on article 2 faint, article 2 twice as long as 1; length, 0.84 mm. Hypostome with dentition arranged 3/3, limited to the terminal half.

Legs.—All tarsi with deep, dorsal, subapical grooves (to receive the stalks of the pulvillus when retracted). Tarsus I broader distally, with ventral spurs absent; all other tarsi with two ventral, subapical spurs.

Coxae.—Coxae I with two moderately long, broad subequal spurs. Coxae II and III each with a short external spur; coxa IV with a moderately long internal spur.

Genital aperture.—Placed between coxae II.

Spiracular plate.—Large, comma-shaped.

Female unknown.

Holotype, 18828, from 3-toed sloth, Barro Colorado Island, Canal Zone, March 30, 1940, and deposited in the collections of the Rocky Mountain Laboratory.

Ixodes spinipalpis Nuttall, 1916

In his Notes on Ticks IV (Parasitology, *8*: 294 (1916)), Nuttall states that in 1911 Dr. Hadwen sent him specimens with a short description and sketchy figures and labeled "*Ixodes dentatus* var. *spinipalpis* Hadwen and Nuttall 1915, n. var." Nuttall considered the specimens to be more or less closely related to *I. fuscipes* Koch, *I. dentatus* Marx 1899, and *I. boliviensis* Neumann 1904. Nuttall forwarded a female specimen to Nathan Banks, then in Washington,

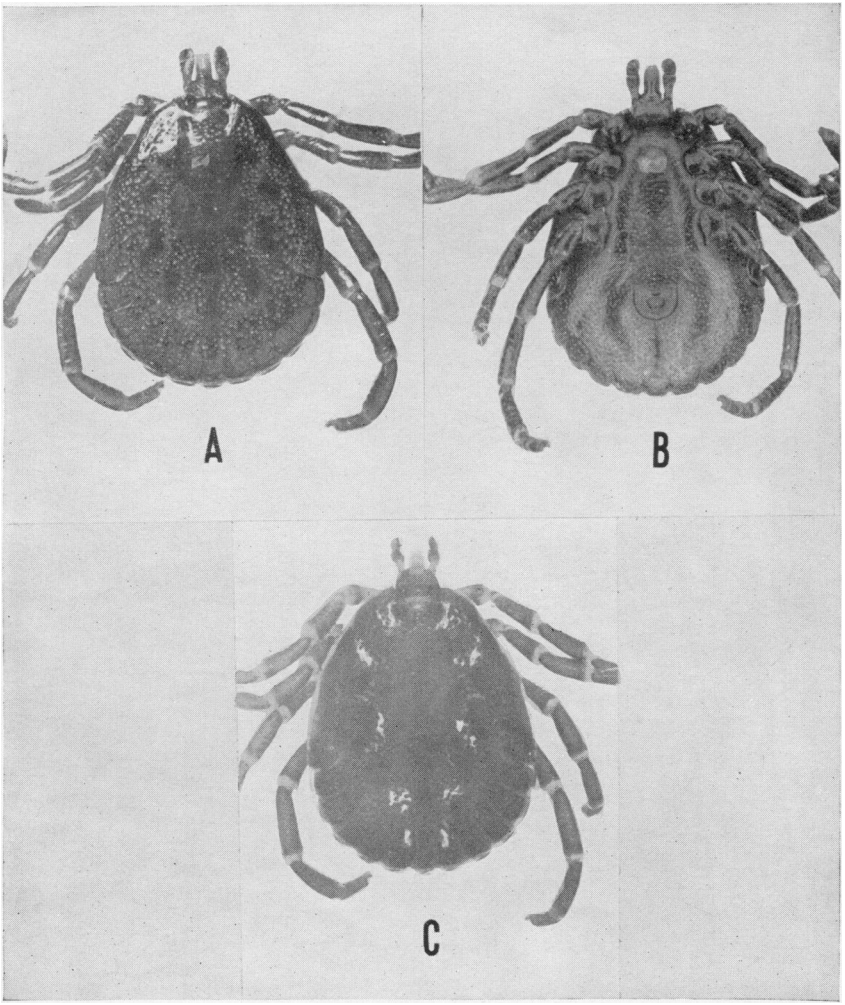

FIGURE 1.—Male of *Amblyomma gertschi* n. sp. A. Dorsal view. B. Ventral view. C. Dorsal view photographed when immersed to show the color pattern.

who compared it with the type of *I. dentatus* and his report stated that the specimen differed from *dentatus* in various particulars. On Banks' suggestion, the form was described as originally labeled by Hadwen.

The present authors find this tick described by Nuttall to be a distinct species which is not more closely related to *dentatus* than to

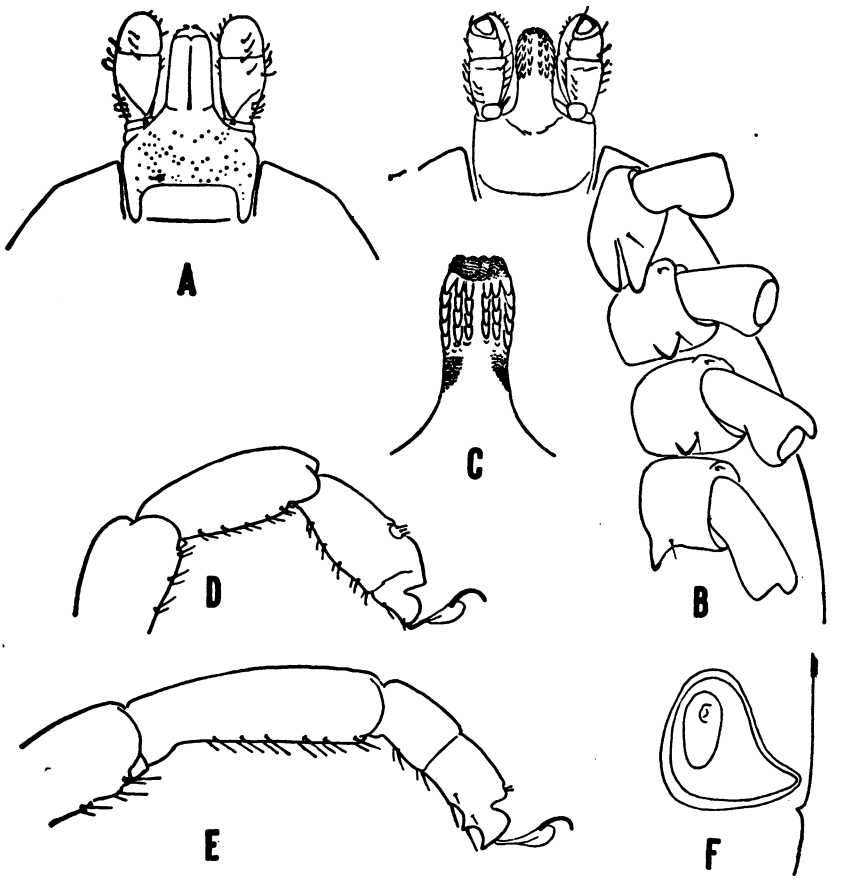

FIGURE 2.—Male of *Amblyomma gertschi* n. sp. A. Basis capituli, dorsal view. B. Basis capituli, ventral view, and coxae. C. Hypostome. D. Tarsus and metatarsus of leg I. E. Tarsus and metatarsus of leg IV. F. Spiracular plate.

other forms in the same group which are recognized as good species. We have seen specimens from the locality from which *spinipalpis*, was described and we recognize the tick to be the same as another species which had been identified as *I. diversifossus* Neumann 1899 of which we have numerous collections from Washington, Oregon, California, Idaho, and Montana. Nuttall stated, "The variety here described occurs in Western Canada on *Lepus americanus* and *Sciurus hudsonicus*." In addition to Nuttall's two hosts, the records of the

Rocky Mountain Laboratory include cottontail rabbit, wood rat, and *Peromyscus* sp.

Neumann described *I. diversifossus* from two specimens taken on raccoon, *Procyon lotor*, in New Mexico. One type is in the United States National Museum and has been studied by the senior author; the second type specimen appears to be missing. Host and locality records of the types indicate that *diversifossus* is a southern form.

The type of *diversifossus* differs from *spinipalpis* in various particulars, including the following:

Length and width of scutum in *diversifossus*, 1.38 mm. by 1.26 mm.; in *spinipalpis*, 1.28 mm. by 0.99 mm. Thus, in *diversifossus* the scutum is nearly circular and evenly rounded behind, while in *spinipalpis* it is distinctly longer than wide, has the postero-lateral margins flattened, and is well rounded behind.

Nuttall has adequately described and figured *spinipalpis* in his paper referred to above. This species should be known as *Ixodes spinipalpis* Nuttall 1916, not *Ixodes dentatus* var. *spinipalpis* Hadwen and Nuttall 1915.

DEATHS DURING WEEK ENDED OCTOBER 31, 1942

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

	Week ended Oct. 31, 1942	Corresponding week 1941
Data from 87 cities of the United States:		
Total deaths	8,495	7,912
Average for 3 prior years	7,843	
Total deaths, first 43 weeks of year	355,821	355,286
Deaths per 1,000 population, first 43 weeks of year, annual rate	11.7	11.6
Deaths under 1 year of age	697	562
Average for 3 prior years	519	
Deaths under 1 year of age, first 43 weeks of year	24,561	22,469
Data from industrial insurance companies:		
Policies in force	65,198,406	64,581,852
Number of death claims	11,834	10,738
Death claims per 1,000 policies in force, annual rate	9.5	8.7
Death claims per 1,000 policies, first 43 weeks of year, annual rate	9.2	9.5

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 NOVEMBER 7, 1942

Summary

Of the nine communicable diseases included in the following table and for which comparable weekly figures are available for prior years, the incidence of only two—influenza and meningococcus meningitis—is above the 5-year (1937–41) median expectancy for the current week.

A total of 59 cases of meningococcus meningitis was reported as compared with 68 for the preceding week. The highest incidence rate was recorded for the eastern and western States. No cases were reported in the West South Central and Mountain States. The total number of cases reported to date this year exceeds that for the corresponding period of any other year since 1937.

Of a total of 1,576 cases of influenza, 68 percent were reported in three States—Texas 602, South Carolina 285, and Virginia 187.

Of 19 cases of infectious encephalitis, 9 cases were reported in California, and of 100 cases of endemic typhus fever, 36 occurred in Georgia and 32 in Texas. A total of 3,114 cases of endemic typhus fever has been reported this year to date—a larger number than has been reported in the United States for any full year.

The death rate for the current week for 88 large cities in the United States is 11.6 per 1,000 population, as compared with 12.0 for the preceding week and 11.1 for the 3-year (1939–41) average.

For the first 9 months of 1942 the provisional birth and death rates for the United States are 20.1 and 10.3, respectively, per 1,000 population, as compared with 18.7 and 10.7 for the corresponding period of last year.

Telegraphic morbidity reports from State health officers for the week ended November 7, 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
	Nov. 7, 1942	Nov. 8, 1941		Nov. 7, 1942	Nov. 8, 1941		Nov. 7, 1942	Nov. 8, 1941		Nov. 7, 1942	Nov. 8, 1941	
NEW ENG.												
Maine	1	0	1				0	54	37	2	0	0
New Hampshire	0	1	0	2			21	1	1	0	0	0
Vermont	0	0	0				104	0	8	0	0	0
Massachusetts	2	3	3				208	79	80	4	2	0
Rhode Island	1	3	1				0	6	2	1	0	0
Connecticut	0	0	2	3	1	2	49	51	7	1	0	0
MID. ATL.												
New York	13	26	18	[1] 9	[1] 2	[1] 6	126	116	128	13	4	4
New Jersey	5	5	11	10	4	6	19	18	18	2	3	1
Pennsylvania	13	9	33	3			190	237	237	3	2	2
E. NO. CEN.												
Ohio	31	19	43	11	11	7	26	36	34	2	2	0
Indiana	8	20	31	17	13	13	16	5	7	0	0	1
Illinois	40	25	34	8	20	12	38	47	47	1	1	1
Michigan [2]	7	11	11				152	29	59	1	0	1
Wisconsin	2	5	2	27	16	21	54	95	50	1	0	0
W. NO. CEN.												
Minnesota	6	1	4	3	1	1	3	1	28	2	0	1
Iowa	1	6	5	2			25	20	20	0	0	0
Missouri	5	4	13		6	6	9	8	13	1	0	0
North Dakota	3	2	4		4		1	91	1	1	1	0
South Dakota	9	5	3		1		0	1	2	1	0	0
Nebraska	14	7	6	1		1	47	2	2	0	0	0
Kansas	4	2	6	2	9	4	0	53	22	0	1	1
SO. ATL.												
Delaware	0	0	0				0	1	1	0	0	0
Maryland [2]	6	22	16	5	1	3	18	28	6	7	2	0
Dist. of Col	1	0	2	1	2		0	1	0	1	0	0
Virginia	25	36	63	187	157	74	2	62	28	2	1	1
West Virginia	32	16	21	20	9	9	7	179	14	0	1	1
North Carolina	65	94	117		1	2	4	53	101	0	1	2
South Carolina	50	23	23	285	221	221	2	22	9	0	0	0
Georgia	25	32	42	38	36	31	1	14	4	1	0	0
Florida	8	5	11	3	7	2	2	5	14	0	0	0
E. SO. CEN.												
Kentucky	16	22	24	5	4	7	14	24	24	2	1	2
Tennessee	8	7	21	17	7	25	18	4	6	1	0	1
Alabama	39	34	34	42	49	49	3	12	9	0	1	2
Mississippi [2]	17	16	17							1	2	0
W. SO. CEN.												
Arkansas	19	15	19	31	42	42	2	9	4	0	0	0
Louisiana	14	9	12	2	13	3	0	0	1	0	2	0
Oklahoma	15	14	22	22	35	33	1	31	1	0	0	0
Texas	64	79	58	602	1,392	218	18	44	28	0	1	1
MOUNTAIN												
Montana	0	6	1	4	5	5	11	15	15	0	1	0
Idaho	0	0	0				28	3	3	0	0	0
Wyoming	0	1	0	45	7		4	2	3	0	0	0
Colorado	11	23	7	88	21	7	12	21	21	0	0	0
New Mexico	1	2	2			1	1	5	14	0	0	0
Arizona	4	5	4	26	76	57	4	54	3	0	0	0
Utah [2]	0	0	0	3	14	2	191	9	10	0	0	0
Nevada	0	0		5			6	0		0	0	
PACIFIC												
Washington	6	0	1	3	3		205	1	20	3	0	0
Oregon	1	12	4	17	10	12	89	15	10	3	0	0
California	27	14	18	27	108	22	40	228	149	2	4	1
Total	619	641	857	1,576	2,308	996	1,771	1,792	1,792	59	33	32
44 weeks	12,408	13,000	18,657	91,272	501,817	176,684	476,152	835,413	356,340	2,970	1,737	1,737

See footnotes at end of table.

Telegraphic morbidity reports from State health officers for the week ended November 7, 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
	Nov. 7, 1942	Nov. 8, 1941		Nov. 7, 1942	Nov. 8, 1941		Nov. 7, 1942	Nov. 8, 1941		Nov. 7, 1942	Nov. 8, 1941	
NEW ENG.												
Maine	0	0	0	8	7	10	0	0	0	0	1	2
New Hampshire	2	0	0	12	9	6	0	0	0	0	0	0
Vermont	1	2	0	2	1	3	0	0	0	0	0	0
Massachusetts	0	4	1	182	100	95	0	0	0	7	1	1
Rhode Island	0	1	0	1	5	6	0	0	0	1	0	0
Connecticut	3	1	0	30	17	30	0	0	0	0	1	1
MID. ATL.												
New York	10	39	10	181	151	173	0	0	0	7	7	12
New Jersey	4	8	5	62	65	61	0	0	0	0	0	2
Pennsylvania	1	6	6	166	133	216	0	0	0	9	9	12
E. NO. CEN.												
Ohio	3	15	10	233	219	227	0	0	0	6	9	12
Indiana	2	0	2	42	36	109	2	1	3	2	1	4
Illinois	20	15	15	186	155	213	0	1	2	2	2	6
Michigan [2]	0	6	6	66	94	109	0	1	1	3	2	3
Wisconsin	4	12	7	179	115	119	0	0	2	0	0	0
W. NO. CEN.												
Minnesota	1	10	10	69	49	68	0	0	3	3	0	0
Iowa	2	2	12	55	45	54	1	0	3	0	0	1
Missouri	4	0	1	48	39	70	1	0	1	5	4	5
North Dakota	0	1	0	10	4	17	0	0	0	0	0	0
South Dakota	2	1	1	38	12	18	0	0	1	0	0	1
Nebraska	5	0	4	15	11	12	0	0	0	0	0	0
Kansas	4	1	2	49	71	71		0	1	0	2	2
SO. ATL.												
Delaware	0	0	0	6	2	6	0	0	0	0	1	1
Maryland [2]	0	2	0	56	38	38	0	0	0	5	7	7
Dist. of Col.	0	2	0	14	13	10	0	0	0	0	0	1
Virginia	1	8	1	88	70	49	0	0	0	1	12	9
West Virginia	0	1	1	49	62	74	0	0	0	3	1	9
North Carolina	1	1	1	135	80	88	0	0	0	6	2	5
South Carolina	0	1	0	12	16	21	0	0	0	2	3	3
Georgia	0	2	1	41	31	31	0	1	0	1	3	7
Florida	1	0	0	7	1	11	0	0	0	3	1	1
E. SO. CEN.												
Kentucky	2	5	5	64	53	53	0	0	0	5	7	12
Tennessee	0	14	1	59	26	49	0	0	0	1	1	7
Alabama	2	6	1	39	22	24	0	0	0	2	0	4
Mississippi [2]	2	4	2	24	6	12	0	0	0	1	2	2
W. SO. CEN.												
Arkansas	3	0	1	11	6	14	0	0	1	2	8	8
Louisiana	2	0	1	9	4	11	0	0	0	4	5	7
Oklahoma	------	3	2	35	17	23	1	0	1	12	1	9
Texas	10	4	4	38	47	47	1	0	2	6	17	17
MOUNTAIN												
Montana	0	3	0	9	26	26	0	0	0	1	0	1
Idaho	0	2	1	4	8	10	0	1	1	0	1	2
Wyoming	0	0	0	0	17	8	0	0	0	0	1	1
Colorado	2	0	0	31	13	35	0	0	0	1	1	2
New Mexico	0	0	0	3	5	8	0	0	0	3	0	5
Arizona	0	0	0	2	1	6	0	0	0	2	0	2
Utah [2]	0	3	3	6	6	17	0	0	0	1	0	1
Nevada	0	0	------	1	3	------	0	0	------	0	0	------
PACIFIC												
Washington	3	1	1	39	52	25	0	0	1	0	0	3
Oregon	0	0	0	6	10	17	0	0	1	1	0	0
California	8	5	5	134	83	130	0	0	0	3	3	7
Total	105	191	191	2,556	2,146	2,659	6	5	55	111	116	259
44 weeks	3,624	8,356	8,356	105,407	104,796	135,828	689	1,220	8,861	6,112	7,621	11,530

See footnotes at end of table.

Telegraphic morbidity reports from State health officers for the week ended November 7, 1942—Continued

Division and State	Whooping cough		Week ended November 7, 1942								
	Week ended			Dysentery							
	Nov. 7, 1942	Nov. 8, 1941	Anthrax	Amebic	Bacillary	Unspecified	Encephalitis	Leprosy	Rocky Mountain spotted fever	Tularemia	Typhus fever
NEW ENG.											
Maine	50	16	0	0	0	0	0	0	0	0	0
New Hampshire	0	13	0	0	0	0	0	0	0	0	0
Vermont	22	17	0	0	0	0	0	0	0	0	0
Massachusetts	170	172	0	0	11	0	1	0	0	0	0
Rhode Island	24	18	0	0	0	0	0	0	0	0	0
Connecticut	80	66	0	0	3	0	0	0	0	0	0
MID. ATL.											
New York	405	459	0	1	10	0	1	0	0	0	0
New Jersey	158	173	0	2	0	0	0	0	1	0	0
Pennsylvania	334	250	0	0	0	0	1	0	1	0	1
E. NO. CEN.											
Ohio	124	222	0	0	3	0	0	0	0	0	0
Indiana	29	9	0	0	0	0	0	0	0	0	0
Illinois	217	215	0	0	0	0	1	0	0	0	0
Michigan [2]	155	257	0	4	0	0	0	0	0	0	0
Wisconsin	151	252	0	0	0	0	0	0	0	0	0
W. NO. CEN.											
Minnesota	40	45	0	0	1	0	0	0	0	0	0
Iowa	11	27	0	0	0	0	0	0	0	0	0
Missouri	9	3	0	0	0	1	1	0	0	0	0
North Dakota	15	9	0	0	0	0	0	0	0	0	0
South Dakota	9	26	0	0	0	0	0	0	0	0	0
Nebraska	3	6	0	0	0	0	0	0	0	0	0
Kansas	30	56	0	0	0	0	0	0	0	0	0
SO. ATL.											
Delaware	7	0	1	0	0	0	0	0	0	0	0
Maryland [2]	81	45	0	0	0	7	1	0	0	0	0
District of Columbia	7	6	0	0	0	0	0	0	0	0	0
Virginia	44	87	0	0	0	32	0	0	2	0	0
West Virginia	12	64	0	0	0	0	0	0	0	0	0
North Carolina	63	113	0	0	0	0	0	0	0	0	1
South Carolina	30	34	0	0	2	0	0	0	0	0	5
Georgia	16	19	0	1	2	0	0	0	0	0	36
Florida	4	17	0	0	1	0	0	0	0	0	7
E. SO. CEN.											
Kentucky	30	123	0	0	2	0	0	0	0	1	0
Tennessee	14	15	0	0	0	12	0	0	0	0	3
Alabama	28	8	0	0	0	0	0	0	0	1	11
Mississippi [2]	------	------	0	0	0	0	0	0	0	0	2
W. SO. CEN.											
Arkansas	30	16	0	1	1	0	0	0	0	2	0
Louisiana	3	6	0	1	2	0	0	2	0	0	1
Oklahoma	0	4	0	0	0	0	0	0	0	0	0
Texas	77	115	0	4	150	0	3	0	0	0	32
MOUNTAIN											
Montana	7	43	0	0	0	0	0	0	0	0	0
Idaho	1	4	0	0	0	0	0	0	0	0	0
Wyoming	17	2	0	0	0	0	0	0	0	0	0
Colorado	14	32	0	0	1	0	0	0	0	0	0
New Mexico	8	7	0	1	9	0	0	0	0	0	0
Arizona	2	25	0	0	0	5	1	0	0	0	0
Utah [2]	11	27	0	0	0	1	0	0	0	0	0
Nevada	1	1	0	0	0	0	0	0	0	0	0
PACIFIC											
Washington	13	98	0	0	0	0	0	0	0	0	0
Oregon	3	24	0	0	0	0	0	0	0	0	0
California	245	142	0	1	9	0	9	0	0	0	1
Total	2,804	3,388	1	16	207	58	19	2	4	4	100
44 weeks	152,531	180,873	------	------	------	------	------	------	------	------	------

[1] New York City only.
[2] Period ended earlier than Saturday.

WEEKLY REPORTS FROM CITIES

City reports for week ended Oct. 24, 1942

This table lists the reports from 86 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								
Baltimore, Md	0	0	2	2	3	7	9	0	6	0	1	53
Billings, Mont	0	0	-----	0	0	0	1	0	0	0	0	8
Birmingham, Ala	2	0	2	1	0	0	1	0	3	0	2	0
Boise, Idaho	0	0	-----	0	0	0	0	0	0	0	0	0
Boston, Mass	0	0	-----	0	8	0	12	2	37	0	1	46
Bridgeport, Conn	0	0	2	3	0	0	1	0	2	0	0	0
Brunswick, Ga	0	0	-----	0	0	0	0	0	0	0	0	0
Buffalo, N. Y	0	0	-----	1	15	1	6	0	4	0	0	8
Camden, N. J	0	0	-----	0	1	0	0	0	3	0	0	9
Charleston, S. C	1	0	3	0	0	0	0	0	0	0	0	0
Charleston, W. Va	0	0	-----	0	0	0	0	0	1	0	0	0
Chicago, Ill	8	0	1	0	5	0	19	4	23	0	1	73
Cincinnati, Ohio	5	0	-----	1	4	0	2	2	18	0	1	13
Cleveland, Ohio	2	0	5	0	0	1	7	0	24	0	0	69
Columbus, Ohio	0	0	-----	0	0	0	3	0	18	0	0	5
Concord, N. H	0	0	-----	0	0	0	0	1	0	0	0	0
Cumberland, Md	0	0	-----	0	0	0	0	0	0	0	0	0
Dallas, Texas	3	0	-----	0	0	0	2	0	6	0	2	1
Denver, Colo	6	0	17	0	2	1	3	0	6	0	0	6
Detroit, Mich	6	1	-----	0	12	0	10	0	18	0	0	99
Duluth, Minn	0	0	-----	0	0	0	1	0	5	0	0	8
Fall River, Mass	0	0	-----	0	1	0	1	0	3	0	0	2
Fargo, N. Dak	0	0	-----	0	1	0	0	0	2	0	0	0
Flint, Mich	0	0	-----	0	0	0	0	0	1	0	0	4
Fort Wayne, Ind	0	0	-----	0	0	0	1	0	0	0	0	0
Frederick, Md	0	0	-----	0	0	0	0	1	0	0	0	0
Galveston, Texas	0	0	-----	0	0	0	0	0	1	0	0	0
Grand Rapids, Mich	0	0	-----	0	0	0	2	0	1	0	0	5
Great Falls, Mont	0	0	-----	0	1	0	2	0	0	0	0	4
Hartford, Conn	0	0	3	0	0	1	0	0	3	0	0	19
Helena, Mont	0	0	-----	0	0	0	0	0	1	0	0	0
Houston, Tex	2	0	-----	0	0	0	10	1	0	2	0	14
Indianapolis, Ind	0	0	-----	0	1	0	7	0	9	0	0	6
Kansas City, Mo	1	0	-----	0	1	0	7	0	18	0	0	1
Kenosha, Wis	0	0	-----	0	0	0	0	0	4	0	0	3
Little Rock, Ark	0	0	-----	0	0	0	1	0	1	0	0	0
Los Angeles, Calif	6	0	14	2	9	3	11	7	20	0	1	37
Lynchburg, Va	2	0	-----	0	0	0	0	0	1	0	1	2
Memphis, Tenn	0	0	-----	0	1	0	3	0	6	0	1	18
Milwaukee, Wis	0	0	-----	0	16	0	0	0	42	0	0	36
Minneapolis, Minn	2	0	-----	0	2	0	7	2	18	0	0	6
Missoula, Mont	0	0	-----	0	0	0	1	0	0	0	0	0
Mobile, Ala	0	0	-----	2	0	0	4	0	1	0	1	2
Nashville, Tenn	0	0	-----	0	0	0	1	0	1	0	0	0
Newark, N. J	0	0	4	0	10	0	5	1	9	0	0	20
New Haven, Conn	0	0	-----	0	0	0	3	0	1	0	0	18
New Orleans, La	0	0	4	4	0	0	3	0	1	0	0	0
New York, N. Y	17	1	12	0	10	8	38	2	66	0	6	127
Omaha, Nebr	0	0	-----	0	1	0	3	0	4	0	0	2
Philadelphia, Pa	2	0	1	0	77	2	25	1	21	0	2	124
Pittsburgh, Pa	5	0	2	2	3	0	12	0	9	0	2	6
Portland, Maine	0		-----	0	1	0	2	0	4		0	18
Providence, R. I	2	0	-----	0	0	0	0	0	2	0	0	17
Pueblo, Colo	1	0	-----	0	1	0	2	0	0	0	0	0
Racine, Wis	1	0	-----	0	0	0	1	0	0	0	0	3
Raleigh, N. C	1	0	-----	0	0	0	0	0	5	0	0	15
Reading, Pa	0	0	-----	0	0	0	0	0	0	0	0	1
Richmond, Va	0	0	-----	0	0	0	5	0	2	0	1	4

City reports for week ended Oct. 24, 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								
Roanoke, Va	0	0	------	0	0	0	0	0	2	0	0	0
Rochester, N. Y	0	0	------	0	0	0	1	0	3	0	0	6
Sacramento, Calif	2	0	------	0	0	0	1	0	6	0	0	1
Saint Joseph, Mo	0	0	------	0	0	0	3	0	1	0	0	0
Saint Louis, Mo	1	0	------	0	0	0	11	0	15	0	0	3
Saint Paul, Minn	0	0	------	0	2	0	3	0	3	0	0	16
Salt Lake City, Utah	0	0	------	0	25	0	0	2	0	0	0	13
San Antonio, Tex	1	0	1	1	0	0	2	3	2	0	0	3
San Francisco, Calif	1	0	------	0	2	0	13	0	4	0	0	7
Savannah, Ga	1	0	1	0	0	1	0	0	2	0	0	1
Seattle, Wash	3	0	------	0	3	0	4	0	1	0	0	7
Shreveport, La	1	0	------	0	0	0	10	0	1	0	0	0
South Bend, Ind	0	0	------	0	0	0	0	0	0	0	0	1
Spokane, Wash	1	0	1	1	7	0	2	0	2	0	0	0
Springfield, Ill	0	0	------	0	0	0	1	0	3	0	0	10
Springfield, Mass	0	0	------	0	1	0	5	0	47	0	0	3
Superior, Wis	0	0	------	0	0	0	0	0	0	0	0	1
Syracuse, N. Y	0	0	------	0	0	0	3	1	1	0	0	4
Tacoma, Wash	0	0	------	0	20	0	0	0	0	0	1	1
Topeka, Kans	0	0	------	0	0	0	0	0	1	0	0	0
Trenton, N. J	0	0	1	0	0	0	2	1	4	0	0	1
Washington, D. C	3	0	------	0	0	3	9	0	14	0	0	4
Wheeling, W. Va	0	0	------	0	1	0	0	0	3	0	0	5
Wichita, Kans	0	0	------	0	0	0	3	0	8	0	2	1
Wilmington, Del	0	0	------	1	0	0	6	0	0	0	0	2
Wilmington, N. C	3	0	------	0	0	0	4	0	0	0	0	13
Winston-Salem, N. C	0	0	------	0	0	0	1	0	8	0	1	2
Worcester, Mass	0	0	------	0	1	0	11	0	15	0	0	23

Dysentery, amebic—Cases: Baltimore, 2; Birmingham, 1; Boston, 1; Chicago, 1; Mobile, 1; New York, 2; San Francisco, 1.
Dysentery, bacillary—Cases: Baltimore, 7; Charleston, S. C., 10; Chicago, 3; Hartford, 3; Los Angeles, 4; New Haven, 1; New York, 13; Richmond, 3; St. Louis, 1; San Francisco, 1; Shreveport, 1.
Dysentery, unspecified—Cases: San Antonio, 3.
Leprosy—Cases: New Orleans, 2.
Typhus fever—Cases: Charleston, S. C., 1; Houston, 2; Mobile, 1; Nashville, 3; New Orleans, 1; Savannah, 5.

Rates (annual basis) per 100,000 population, for the group of 86 cities in the preceding table (estimated population, 1942, 33,639,009)

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 Oct. 24, 1942	14.26	11.78	3.26	38.44	51.00	89.28	0.31	4.19	161.21
Average for week 1937–41	16.60	9.40	[1] 5.14	[2] 54.66	[1] 38.20	85.67	0.47	5.79	155.05

[1] 3-year average, 1939–41.
[2] Median.

PLAGUE INFECTION IN TACOMA, WASHINGTON

Under date of October 27, 1942, plague infection was reported proved in 2 pools of fleas and lice from rats, *Rattus norvegicus*, taken in Tacoma, Wash., on October 9 and 10, respectively; one consisted

of 115 fleas and 81 lice from 53 rats, and the other of 100 fleas from 44 rats.

TULAREMIA INFECTION IN FIELD MOUSE IN SOUTH DAKOTA

The plague laboratory in San Francisco reports the finding of tularemia infection in a field mouse (*Microtus pennsylvanicus*) found dead on September 22, 1942, 7½ miles southwest of Newell, near U. S. Highway No. 212.

TERRITORIES AND POSSESSIONS

Hawaii Territory

Plague (rodent).—Information dated October 12, 1942, states that 2 rats proved positive for plague were found in Paauhau area, Hamakua District, Island of Hawaii, and 1 rat proved positive for plague was found in Makawao, 9 miles from Kahului, Island of Maui. Information dated October 15, 1942, states that 2 other rats proved positive for plague were found in Paauhau area, Hamakua District, Island of Hawaii, T. H.

FOREIGN REPORTS

CANADA

Provinces—Communicable diseases—Week ended October 10, 1942.—During the week ended October 10, 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	British Columbia	Total
Cerebrospinal meningitis		2	1	2	4			1	3	13
Chickenpox	1	11		46	61	62	18	5	37	241
Diphtheria		13	2	33	2	11	1	2		64
Dysentery					1		3			4
Encephalomyelitis							1			1
German measles				3	6		3		4	16
Influenza					2		1			3
Lethargic encephalitis						2				2
Measles				77	28		8	2	8	123
Mumps		14	1	37	128	9	30	47	99	365
Pneumonia		2			10				13	25
Poliomyelitis		3	2	6	5	2			3	21
Scarlet fever		3	5	78	64	12	25	19	22	228
Trachoma									1	1
Tuberculosis	3		4	84	50	10	11	9	21	192
Typhoid and paratyphoid fever		1		21	1		1		1	25
Undulant fever				5	1					6
Whooping cough		1		287	90	20	14	78	11	501
Other communicable diseases		3		5	254	32		3	6	303

CUBA

Habana—Communicable diseases—4 weeks ended October 17, 1942.—During the 4 weeks ended October 17, 1942, certain communicable diseases were reported in Habana, Cuba, as follows:

Disease	Cases	Deaths	Disease	Cases	Deaths
Diphtheria	14		Poliomyelitis	20	4
Malaria	7		Tuberculosis	7	1
Measles	3		Typhoid fever	17	1

GREAT BRITAIN

England and Wales—Infectious diseases—Years 1939, 1940, and 1941.—During the years 1939, 1940, and 1941, cases of certain infectious diseases were reported in England and Wales as follows:

Disease	Cases (including non-civilians)		
	1939	1940	1941
Cerebrospinal fever	1,500	12,771	11,077
Diphtheria (including croup)	47,341	46,281	50,797
Dysentery	1,941	2,860	6,670
Erysipelas	14,141	13,123	12,237
Lethargic encephalitis	159	211	187
Malaria	2	2	2
Measles [1]	15,879	409,521	409,715
Ophthalmia neonatorum	4,594	4,390	4,195
Pneumonia [2]	42,312	47,875	50,942
Polioencephalitis	88	128	83
Poliomyelitis	744	951	876
Puerperal pyrexia and puerperal sepsis	9,250	7,627	7,356
Scarlet fever	78,101	65,302	59,432
Smallpox	1	1	
Typhoid and paratyphoid fever	1,479	2,833	4,763
Typhus fever	1		
Whooping cough	8,690	53,607	173,331

[1] Notifiable from Oct. 23, 1939.
[2] Includes influenza with pneumonic complications.

England and Wales—Vital statistics—Years 1939, 1940, and 1941.—The following table shows the numbers of births and deaths with rates per 1,000 population in England and Wales for the years 1939, 1940, and 1941. The figures for 1940 and 1941 are provisional and crude death rates are substituted for standard death rates:

	1939		1940		1941	
	Number	Rate per 1,000 population	Number	Rate per 1,000 population	Number	Rate per 1,000 population
Live births	619,352	14.9	607,029	14.6	587,228	14.2
Deaths, all causes	499,804	12.1	581,537	14.0	535,180	12.9
Maternal deaths	1,997	[1] 3.1	1,640	[1] 2.6	1,678	[1] 2.76
Infant mortality	31,190	[1] 50	33,892	[1] 56	34,550	[1] 59
Deaths from:						
Cancer	67,154	1.62	68,922	1.66	69,227	1.67
Cerebrospinal fever	517	.012	2,584	.062	2,163	.052
Diarrhea and enteritis	4,345	.105	4,433	.107	4,654	.112
Diphtheria	2,133	.051	2,480	.060	2,641	.064
Dysentery	96	.002	185	.004	329	.008
Erysipelas	248	.006	214	.005	190	.005
Influenza	8,020	.193	11,482	.277	6,901	.166
Lethargic encephalitis	572	.014	729	.018	704	.017
Malaria	20	.000	46	.001	19	.000
Measles	303	.007	857	.021	1,145	.028
Ophthalmia neonatorum	12	.000	7	.000	4	.000
Pneumonia	23,403	.564	29,195	.704	26,418	.637
Polioencephalitis	48	.001	54	.001	47	.001
Poliomyelitis	95	.002	107	.003	113	.003
Puerperal pyrexia and puerperal sepsis	402	.010	339	.008	288	.007
Scarlet fever	181	.004	154	.004	133	.003
Tuberculosis	25,623	.618	28,144	.679	28,670	.692
Typhoid and paratyphoid fever	112	.003	135	.003	148	.004
Typhus fever					1	.000
Whooping cough	1,229	.030	678	.016	2,383	.057

[1] Per 1,000 live births.

JAMAICA

Communicable diseases—4 weeks ended September 26, 1942.—During the 4 weeks ended September 26, 1942, cases of certain communicable diseases were reported in Kingston, Jamaica, and in the island outside of Kingston, as follows:

Disease	Kingston	Other localities	Disease	Kingston	Other localities
Chickenpox	4	3	Scarlet fever	1	
Diphtheria	4	3	Tuberculosis	36	64
Dysentery	2	3	Typhoid fever	6	56
Erysipelas		1	Typhus fever	3	1
Leprosy		5			

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-named 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 of each month.

(Few reports are available from the invaded countries of Europe and other nations in war zones.)

Plague

Indochina.—For the period October 11–20, 1942, 4 fatal cases of plague were reported in Indochina.

Morocco.—For the week ended October 17, 1942, 13 cases of plague were reported in Morocco.

Typhus Fever

France (occupied zone).—During the month of September 1942, 1 case of typhus fever was reported in France (occupied zone).

Morocco.—During the week ended October 17, 1942, 39 cases of typhus fever were reported in Morocco.

Rumania.—During the week ended October 24, 1942, 8 cases of typhus fever were reported in Rumania.

Tunisia.—During the period September 21–30, 1942, 101 cases of typhus fever were reported in Tunisia.

Turkey.—During the week ended October 24, 1942, 6 cases of typhus fever were reported in Turkey. For the week ended October 3, 1942, 4 cases of typhus fever were reported instead of 44 as previously published.