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## THE IDENTIFICATION AND LOCALIZATION OF LEAD IN BONE TISSUE<sup>1</sup>

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Although storage of lead in bone tissue had previously been referred to by several investigators, it was in effect rediscovered by Minot (1) and added significance given to its presence in that particular tissue. Previously, its presence in bone tissue had been generally regarded somewhat as accidental. As early as 1861, Gusserow (2) demonstrated the presence of lead in bone tissue and ascribed this deposition to the formation of a compound of lead with calcium. However, Gusserow's experiments were buried in oblivion for nearly 60 years. Other investigators occasionally published data concerning the lead content of bone tissue, but these data were considered as incidental to the rather widespread deposition of lead throughout the tissues. At various times the lead content of the brain, liver, and kidneys has been considered of prime importance and it has also been assumed by some that in whichever tissue the degree of deposition was greatest, tissue damage at that site would be correspondingly great.

The lead stored in bone tissue is of great significance in the study of lead poisoning; in a sense the behavior of lead in the organism as a whole is dependent upon the efficiency of this storage. The composition, manner of deposition, and site of deposition are therefore of importance particularly with reference to low grade lead absorption and incipient plumbism. Certain factors may modify the degree of lead storage in bone tissue, but storage in bone is less affected by various conditions than is storage in the softer tissues.

Up to a certain point, the human organism can tolerate absorption of lead by efficiently excreting it. Beyond this point, whether the lead enters the system by ingestion or by inhalation, the excretory mechanism is not the sole means adopted to meet this danger. It is perhaps fortunate that the properties of lead are such that precipitation of lead occurs in the bone tissue, so that the circulating lead can be kept to a minimum.

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<sup>1</sup> From the Division of Industrial Hygiene, National Institute of Health.

The impression is common that since lead is an accumulative poison, all of the lead absorbed is stored. However, the proportion of storage in relation to the amount absorbed is still not known, and little is known about the persistence of the bone-stored lead.

It was recently found that not all compounds of lead produce the same amount of bone lead deposition (3). When equivalent amounts of lead are fed both as carbonate and as arsenate, much more lead is deposited as a result of absorption from the carbonate than from the arsenate. This cannot be ascribed entirely to differences in solubility of the two, since lead arsenate is so completely broken down in the body that the greater part of the arsenic is excreted through the kidneys (4).

While lead is deposited in other tissues, the quantity is neither as great nor is it immobilized to the extent that it is in bone tissue. It appears, in fact, to be quite loosely held in the liver and kidneys as compared with the bones. While it was shown, as stated above (Minot, loc. cit.), that lead tends to be fixed in the bones and in time to decrease in amount in the softer tissues, the mobility of lead outside the osseous system nevertheless has not been fully appreciated. Recent experimental work (5) has shown that in as short a period of time as two weeks the liver and kidney content of stored lead may be diminished by 50 percent after restoration to a normal diet. Compared with the bones, storage in the soft tissues would therefore appear to be incidental.

Far less has been accomplished with reference to the question of the site of deposition in the bone tissue apart from the question of gross storage. Since the bone trabeculae are the first to be affected in bone changes, this would also appear to be the logical site of initial lead deposition. Minot and Aub (6) showed that this bone material was especially rich in lead. Behrens and Baumann (7), using Thorium B as an indicator, found the epiphyseal zone to be richest in lead following intravenous injection. The demonstration of lead in bone by Thorium B or by analytical means, however, localizes it only in a gross sense. The microscopic demonstration and identification of lead *in situ* in bone has been difficult heretofore owing to the colloidal nature of the material in which lead is deposited, the irregularity of deposition, and the insolubility of the deposited lead compound.

Sulfide staining which has been advocated at various times (8, 9) to demonstrate lead deposits in tissues is frequently uncertain and indefinite. Lead salts in the presence of protein usually give only a faint generalized stain which is not always apparent under the microscope—particularly in the early stages of lead absorption. However, sulfide staining in the later stages of lead deposition when granular deposits are present and in connection with other methods is very useful. Sieber (loc. cit., p. 275) decalcified bone in formic

acid containing hydrogen sulfide and used dark-field illumination to demonstrate the particulate material.

In the present investigation, it was shown that this particulate material may be demonstrated directly by dark-field illumination without treatment with hydrogen sulfide and furthermore that only the surface of the deposited lead compound is darkened by the sulfide.

Since particulate lead has been demonstrated in lung tissue by a slow crystallization of the very insoluble lead chromate in acetic acid solution (10), this procedure was tried with bone tissue known to contain lead. It was found that, although slow in action, lead chromate would crystallize under these conditions. It usually requires several days for this to occur owing to the great insolubility of the deposited lead. The lead deposit apparently dissolves very slowly and crystallizes about the particles or diffuses to the surface where it is revealed as minute crystals of lead chromate, usually too small for angular measurement and frequently somewhat irregular in shape owing to the presence of organic material. It was found possible to decalcify bone by treatment with sulfurous acid without affecting the deposited lead. Micro tests of the crystalline compound remaining in bone tissue after complete decalcification were positive for lead and for the phosphate group and indicated the likelihood that the deposited substance is a lead phosphate.

#### METHOD

It is preferable to saw the bone lengthwise previous to decalcification. The bone may be preserved in 10 percent formalin and is decalcified in a saturated solution of sulfur dioxide in distilled water with frequent changes. It is finally rinsed free from sulfite. Sections can be cut satisfactorily from paraffin. Decalcification of rather large pieces of bone is fairly rapid. No lead is removed from the bone in this process. Repeated analyses of the material dissolved from leaded bone, together with the washings, have been negative for lead, while analyses of similar pieces of calcified and decalcified bone check satisfactorily. Additional evidence from the examination of ground bone sections indicates that the lead deposit is not affected by the decalcifying medium.

Bone sections are carried through xylene and the alcohols to water and finally to a 1 percent potassium chromate-5 percent acetic acid solution in which they are left immersed for several days. Frequently a sufficient deposit of lead chromate crystals is evident after 24 hours, but usually immersion for a few days is advisable.

It is preferable to examine the unstained sections, under a polarizing or crystallographic microscope, since the lead chromate deposit is thrown into brilliant relief against the background, while under ordi-

nary transmitted light the crystals are occasionally difficult to detect and may escape observation. The crystals are monoclinic prisms, usually well defined (fig. 1), but occasionally distorted possibly owing to the protein medium in which they form. Crystallization commonly occurs in groups of minute crystals, however, rather than in single large forms (fig. 2). The crystals average 2.5 microns in length. They are soluble in hydrochloric acid, are birefringent, and have a high mean index of refraction.

#### DISCUSSION

The identification of lead in bone tissue by the above procedure is definite since other metal chromates are either soluble in acetic acid or can easily be differentiated from crystalline lead chromate. Thus bismuth, which might conceivably be deposited in bone tissue following medication with its derivatives, forms a chromate insoluble in water. However, precipitated bismuth chromate, being amorphous, (as verified by X-ray diffraction study) lacks any distinguishing crystallographic properties and furthermore is too soluble in 5 percent acetic acid to be a matter of consequence.

An opportunity to verify this method occurred in the examination of bones of a control dog. Microscopically, lead chromate was found to be deposited in tissues which normally should have been free of lead. Subsequent chemical analyses revealed that lead storage had occurred in this animal and thus confirmed the microscopic findings.

It was found that in addition to hydrogen sulfide, basic dyes such as methyl green stain particulate lead deposits in bone (fig. 3), and in fact this particulate lead deposit in the later stages of lead deposition is directly visible under the microscope without staining (fig. 4). However, in the earlier stages of deposition the lead is so diffused in the tissues that no particulate staining occurs with sulfide or dyes and in either case neither of these methods serves to *identify* the lead. The crystalline chromate method, on the other hand, definitely identifies the lead even in the absence of any particulate deposition (fig. 5).

Study of bone sections thus prepared from various lead-poisoned animals revealed deposits of chromate around the Haversian canals and extending in the lamellae for some distance from the center. There is no evidence of deposition along the protoplasmic processes extending to the osteocytes and no evidence that the deposit is greater about the Volkmann canals than the Haversian. The cancellous tissue is rich in lead, as might be expected. It is of interest in this connection that Behrens and Baumann (*loc. cit.*, p. 252) report the compacta as entirely free from lead. However, the compact tissue in animals that had been poisoned over a long period of time in the present study also shows an extensive amount of deposited lead and, furthermore, lead was always demonstrable in the compact

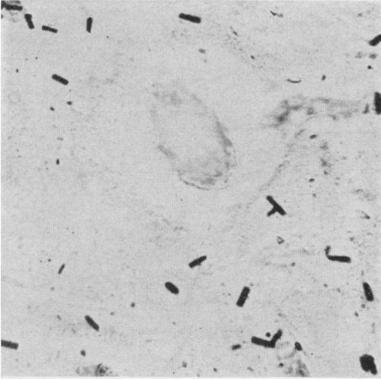


FIGURE 1.—Ground section of tibia of lead-poisoned dog showing lead chromate crystals.

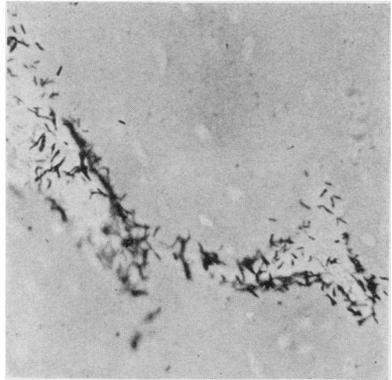


FIGURE 2.—Section of femur of lead-poisoned dog showing grouping of chromate crystals.

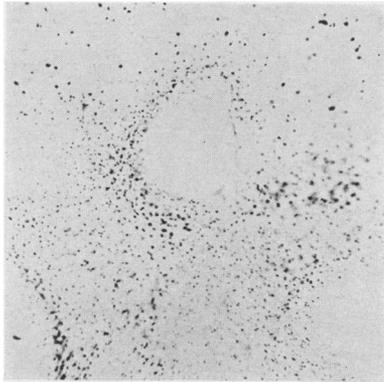


FIGURE 3.—Particulate lead in cancellous tissue stained with methyl green  $\times 320$ .

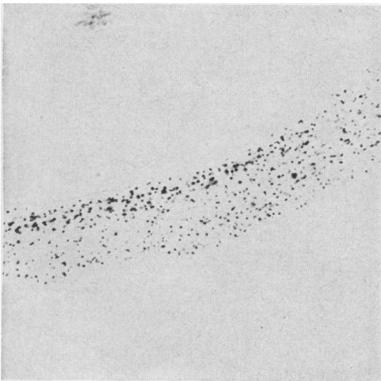


FIGURE 4.—Unstained lead deposit in cancellous tissue of a lead-poisoned dog (humerus)  $\times 320$ .

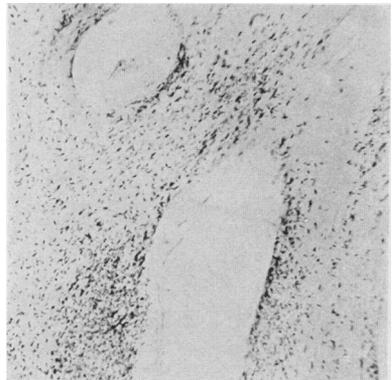


FIGURE 5.—Lead chromate crystals in a section of the compact tissue of the femur of a lead-poisoned dog.

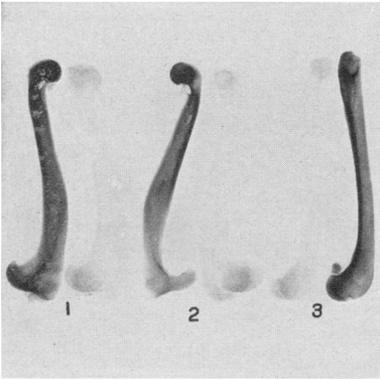


FIGURE 6.—X-ray photographs of calcified and decalcified bones of a lead-poisoned dog.

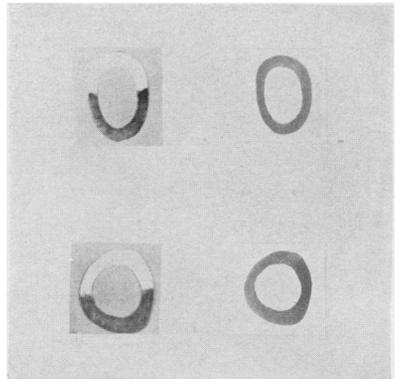


FIGURE 7.—Normal and X-ray photographs of thin sections of bone treated with lead chloride.

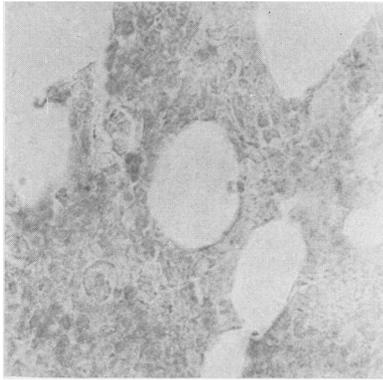


FIGURE 8.—Marrow from normal dog bone (femur) stained with methyl green.

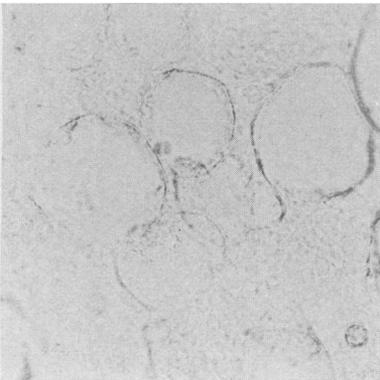


FIGURE 9.—Marrow of guinea pig femur after 2 weeks of lead absorption. Unstained.

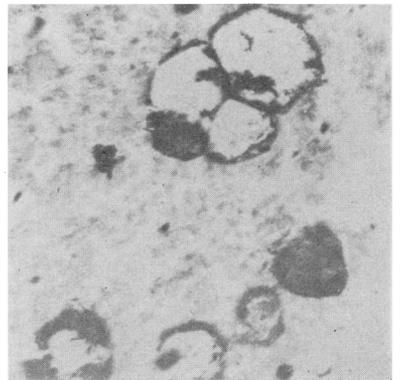


FIGURE 10.—Marrow of dog fed large amounts of lead for 8 months. Unstained.

tissue of animals exposed to lead for shorter periods of time. Conforming to Behrens' observations, the cartilage has always been found to be free from lead. The distinction frequently made between the lead content of the epiphysis and diaphysis is not as great as might be inferred from occasional analyses or from the literature in general. Analysis of the shaft of the long bones as well as the ends of the bone in the case of two lead-poisoned dogs showed the values given in table 1.

TABLE 1.—Analyses of the epiphyses and diaphyses of bones of lead-poisoned dog

Dogs	Amount of lead fed (grams of Pb)	Days of experiment	Portion analyzed	Femur mg/10 g.	Humerus mg/10 g.	Radius mg/10 g.	Ulna mg/10 g.	Tibia mg/10 g.	Fibula mg/10 g.	Scapula mg/10 g.	Average mg/10 g.
1	7.28	260	{ Shaft.....	2.17	2.34	2.37	4.03	3.52	2.01	-----	2.74
			{ Epiphysis....	3.52	3.05	3.38	3.16	2.75	2.01	-----	2.97
6	2.49	129	{ Shaft.....	2.88	2.24	2.39	2.53	3.18	2.49	} 2.49	{ 2.57
			{ Epiphysis....	2.19	2.06	2.39	2.71	1.89	2.18		

It is apparent from this table that the average values for the diaphyses do not differ greatly from those of the epiphyses. Similarly, Barth (11) found that no differences could be established between epiphyses, diaphyses, and flat bones. A distinction, however, should be made with reference to the basis of comparison—whether it refers to moist bone samples or oven-dried samples. The epiphysis contains more water than the shaft and comparison of samples of bone dried at 105–110° C. indicates a higher percentage content of lead in the epiphysis than in the diaphysis.

Moreover, further work in progress indicates that when the calcium-lead ratios are compared, the epiphyseal values are generally somewhat greater than the diaphyseal.

The radioactivity experiments of Behrens and Baumann clearly show the greater part of the lead in the epiphysis. However, in their experiments they injected the salts containing Thorium B intravenously and since the blood supply of the epiphysis is much greater than that of the shaft, it is not surprising that a deeper shadow would be obtained in the one case than in the other. Furthermore, deposition of lead in bone tissue is ordinarily a slow process whereas these experiments were completed within a matter of hours. X-ray evidence both for and against segregation of lead into a distinct line or zone in this manner has been accumulated (12). The point at issue so far as this study is concerned is not that of the structure of the bones as revealed by X-rays, but whether or not the opacity of the bands is due to deposited lead salts.

Microscopical examination of bone by the above method not only shows an abundant deposition in the cancellous tissue but also a

marked lead deposit in the compact tissue, while the only evidence of an extra deposit in the epiphysis is a line just below the articular cartilage. This line has proved to be only a few microns thick in every animal examined and it is doubtful whether an X-ray photograph would be sufficient to reveal this in the presence of so much calcium.

TABLE 2.—*Effect of SO<sub>2</sub>-decalcification upon the lead content of bones*

[Lead content (mg. Pb/10 g. bone)]

Number	Bone	Calcified portion	Decalcified portion
1	Humerus.....	1.05	1.10
2	Humerus.....	1.12	.92
3	Femur.....	1.05	1.16

X-ray photography of bones of dogs which had received large doses of lead carbonate over a period of months failed to reveal opaque areas which have heretofore been indicated as characteristic of lead poisoning. In the case of one dog, each of three bones was cut lengthwise, one-half decalcified, and both calcified and decalcified bones X-rayed together (fig. 6). Subsequent analysis showed that lead was present in both the calcified and decalcified bone to the same extent (table 2). Since the lead was not removed from the bone by decalcification and since the decalcified bone is very transparent to X-rays, the conditions were optimal for revealing the lead. It is apparent that the small amounts of lead deposited in bone are insufficient to register by means of X-rays and that darkened areas which have heretofore been accepted as lead deposits are likely to be due to calcium. From what is now known concerning the distribution of lead in bone, it would be surprising indeed if such small amounts of lead so evenly distributed would be revealed by X-rays in the presence of such large amounts of calcium. The lead content of human bones with known exposure to lead is also insufficient in amount to impart any marked opacity to X-rays. In seven cases of lead poisoning reported in man (13), the lead content of the bones was found to range from 0.22 mg./10 g. of bone to 1.5 mg./10 g. of bone. Tompsett and Anderson (14) found the lead content of bones in two cases of occupational exposure to lead to vary from 0.085 to 1.194 mg.Pb/10 g. of bone. Even in animals subjected to severe lead poisoning the largest amount of lead in the bone tissue of rats in the present study was 15 mg./10 g. of bone.

Further study was made of thin sections of compact bone tissue which had been partly submerged in a saturated solution of lead chloride for 24 hours, then washed and X-rayed. The lead deposit was converted to sulfide for photography (fig. 7) and the sections were then ashed and analyzed. The lead content of the sections

were as shown in table 3. It will be seen from the above that although a substantial amount of lead is present asymmetrically distributed, no indication of its presence is indicated in the X-ray photographs.

TABLE 3.—Lead content of X-rayed bone

Number	Thickness of section (mm.)	Weight (grams)	Lead content (mg. Pb/10 g. bone)
1	1.14	1.55	12.7
2	.92	.97	5.5

Deposition of lead in the red marrow, particularly within the fat cells, was found to occur in all cases of extensive lead absorption. In the earlier stage of lead absorption a noncrystalline deposit occurs about the periphery of the fat cells, while in the later stage a crystalline aggregation is to be noted in the fat cells, occasionally completely filling a cell. Figures 8, 9, and 10 show, respectively, normal marrow, marrow with a beginning deposition of lead about the border of the fat cells following the ingestion of lead for 2 weeks, and finally marrow of a dog fed large amounts of lead for 8 months. Chemical analysis and microscopical observation show yellow marrow to be practically free from lead in lead-poisoned animals but the evidence of an accumulation of lead in the fat cells of the red marrow is of particular interest with respect to the anemia found in lead poisoning. The reason for the presence of lead in the fat cells of the red marrow is somewhat obscure although deposition of the lead in bone in general occurs at precisely those points where the blood supply is rich. Whether the phosphate ion concentration is greater at the fat cell interface, whether phosphatase is more abundant at this point, or whether deposition in the cell is accidental requires experimental verification. Apparently deposition begins in the fat cells even before lead is appreciably deposited in the cancellous tissue.

A sequence of deposition is noted if one studies the cancellous or compact bone of animals in different stages of lead absorption. A study of this sequence of lead deposition by both the chromate crystal method and staining method indicates that, in the early stages, lead is simply diffused through the tissue in colloidal form as indicated by a brownish staining with the sulfide technique. Later on there is a distinct segregation of the lead into crystalline masses localized in clumps around the Haversian and Volkmann canals, in the compact tissue, in the fat cells of the red marrow, and throughout the cancellous tissue. The microscopic evidence would therefore appear to indicate that lead is deposited in bone in two stages: (a) initially, as colloidal lead diffused through the tissue substance, possibly as an adsorption phase, and (b) finally, as lead absorption continues, as segregated, definite crystalline masses.

## SUMMARY

A method of positively identifying lead in bone tissue has been evolved which permits detailed investigation of the lead deposit. It has been shown that while the epiphyseal portion of bone is rich in lead, particularly in the early stage of lead absorption, deposition occurs throughout the compact tissue on all surfaces over which blood passes. X-ray studies of the bones of lead-poisoned animals indicate that the amount of lead, whether segregated or diffused, even in the absence of calcium, is insufficient to be revealed by ordinary X-ray photography. Deposition of lead was shown to occur in the fat cells of the red marrow.

Lead storage in bone tissue occurs first as colloiddally dispersed and finally as segregated crystalline masses.

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## THE MICROCLIMATE OF DIURNAL RESTING PLACES OF *ANOPHELES QUADRIMACULATUS* SAY IN THE VICINITY OF REELFOOT LAKE<sup>1</sup>

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During the summer of 1941 reconnaissance of the land surrounding Reelfoot Lake, Tennessee, revealed many concentrations of *Anopheles quadrimaculatus* Say in barns or similar artificial shelters. As many as 10,000 mosquitoes (estimated number) were found in a single building. Nine shelters were selected for study.

The following report discusses the movements of mosquitoes out of these shelters at dusk and into them at dawn, especially in relation to the climatic conditions inside the shelters and in the open at the time these movements took place. Conditions that prevailed in the resting places during the daylight hours of rest are also considered.

There are three qualifications which a resting place must possess to be attractive to adult mosquitoes of any species. In the first place it must be within flight range of a breeding place of the mosquito; second, it must be close to a blood meal; third, it must have a microclimate which is preferred or at least tolerated by the mosquito.

Of the nine shelters selected for study, all met the first and second requirements (table 4). The third requirement, that the resting place must have a microclimate which is preferred or at least tolerated by the mosquito, has been investigated in connection with *A. maculipennis* (2) but no detailed study of these conditions in relation to *A. quadrimaculatus* has been found in the literature. Only the suggestions that the imagos prefer places with "darkness, or a dim, diffused light; cool temperature; [high] humidity; and little if any movement of air" have been found (1).

As it has been suggested that each type of *Anopheles* may have a preferred type of microclimate, and as a change in type of structure might discourage large concentrations of mosquitoes in a given region, an intensive study of several resting places of *A. quadrimaculatus* was undertaken.

Measurements of temperatures, humidities, and light intensities have been recorded. An index of evaporation for comparing resting places is also presented.

This study was made during the months of August and September 1941. Facilities of the Reelfoot Lake Biological Station of the Tennessee Academy of Sciences were generously lent for the investigation.

<sup>1</sup> From the Division of Infectious Diseases, National Institute of Health.

MOVEMENTS AT DUSK

*Method of study.*—Hygrothermographs were placed in each resting place the night before observations were to be made and were removed at the same hour the following night. A hygrothermograph was also maintained nearby in a standard weather instrument shelter during

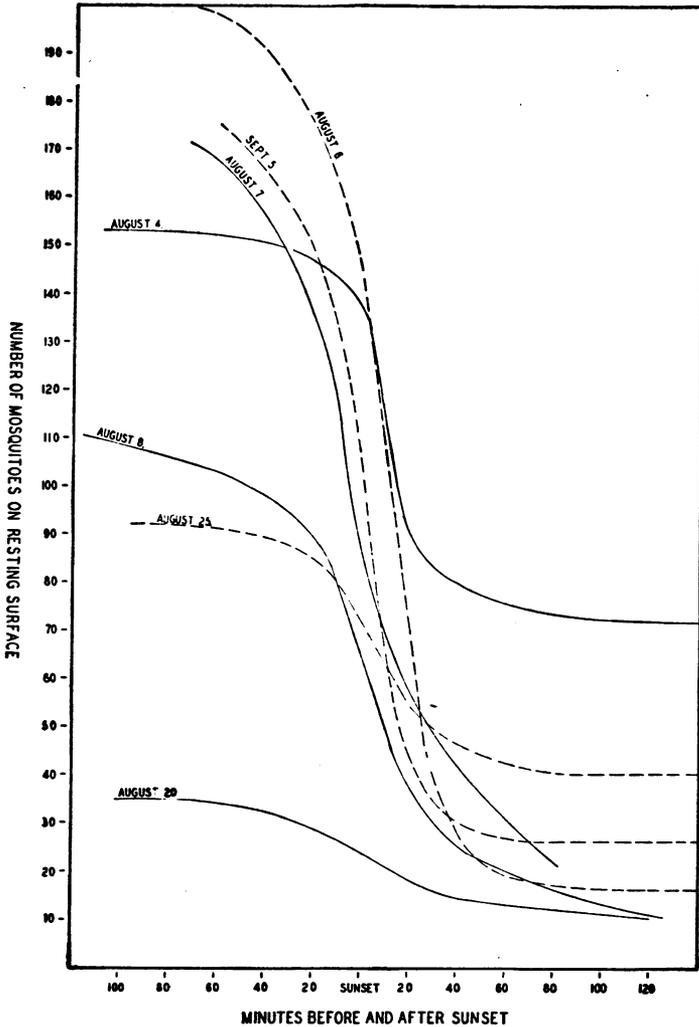


FIGURE 1.—Smoothed curves representing the evening exodus of *Anopheles quadrimaculatus* from the diurnal resting place.

all the experiments. The mosquitoes<sup>2</sup> on selected surfaces in the structures to be studied were counted, with the aid of a flashlight, at 10- to 30-minute intervals for about 2 hours before and 2 hours after sunset. Often an additional count was made later in the evening.

<sup>2</sup> In the following paragraphs "mosquitoes" refers to the females of *Anopheles quadrimaculatus* in all cases.

TABLE 1.—The number of mosquitoes of an original 100 which leave the resting surface during various 10-minute periods before and after sunset, compared with mean microclimatic conditions<sup>1</sup>

	Minutes before and after sunset																
	80-70	70-60	60-50	50-40	40-30	30-20	20-10	10-S.	S.-10	10-20	20-30	30-40	40-50	50-60	60-70	70-80	80-90
Shelter 1, Aug. 4.....	0.4	0.6	1.2	1.5	1.6	2.2	4.9	7.4	16.1	42.0	8.5	4.9	3.1	2.2	1.2	0.8	0.6
Shelter 2, Aug. 6.....	.5	.7	1.6	2.4	3.8	5.4	7.3	8.2	19.0	20.6	18.5	5.4	3.5	1.4	1.7	2.4	.3
Shelter 2, Aug. 7.....		2.6	3.3	4.0	5.1	8.0	11.9	20.8	10.6	7.6	6.3	4.6	4.0	3.3	3.0	2.3	
Shelter 2, Aug. 8.....	1.5	1.8	2.3	2.8	4.0	5.5	9.0	12.0	18.0	12.5	7.0	5.0	3.5	2.5	2.5	2.0	1.5
Shelter 2, Aug. 20.....	1.0	2.0	2.0	4.0	6.0	8.0	10.0	12.0	12.0	12.0	8.0	6.0	4.0	3.0	2.0	2.0	2.0
Shelter 1, Aug. 25.....	1.0	1.0	1.4	1.4	2.4	5.8	7.9	14.4	17.3	18.3	8.8	5.8	4.8	3.8	1.9	1.3	.5
Shelter 2, Sept. 5.....	2.2	2.2	2.9	3.8	4.2	5.4	7.7	20.0	30.1	9.6	5.1	3.2	1.6	3.8	4.3	1.2	
Mean ratio.....	1.6	2.1	2.1	2.8	3.9	5.8	8.4	13.5	17.6	17.5	8.9	5.0	3.5	2.4	1.7	1.3	
Mean temperature in resting place.....	83.0		82.6		82.2		81.4		80.6		79.3		78.1		77.3		76.4
Mean temperature in instrument shelter.....	81.5		79.9		77.9		76.1		74.3		72.9		72.4		71.9		71.3
Mean humidity in resting place.....	57.6		60.4		62.3		65.9		66.9		68.0		68.7		70.7		73.1
Mean humidity in instrument shelter.....	91.9		96.1		98.4		99.4		100.0		100.0		100.0		100.0		100.0
Mean evaporation in resting place.....	12.6		11.7		10.6		9.8		9.4		8.7		7.9		7.3		6.5
Mean evaporation in instrument shelter.....	2.5		1.0		.4		.2		0		0		0		0		0
Mean outside light intensity.....	1,430		1,040		740		260		48		9		2		0		0

<sup>1</sup> Temperatures are in degrees Fahrenheit, humidities in percentage saturation, evaporation as vapor pressure deficit, and light intensity in foot candles.

Also, at the same interval of time, readings of light intensity in the open were made with an illumination meter. Owing to the fact that the meter could not be used below intensities of 1 to 2 foot candles, it was impossible to make the light intensity readings inside the structures during the dusk hours, and subsequently during the hours of dawn.

Temperature and humidity readings from the instrument shelter are referred to as temperatures and humidities in the open. The function of the shelter is to prevent the transformation of radiant energy from the sun from being reflected in the readings of the weather instruments. During the period of the experiment air movements were at a minimum.

*Discussion.*—The procedure outlined was carried out on seven occasions at two shelters. The first shelter was located in a treeless pasture and had a loft almost completely closed off from that part of the barn in which the greatest number of mosquitoes rested. The second shelter was almost surrounded by trees and had an open loft.

Figure 1 shows on smoothed curves the changes in density of mosquitoes on the selected surfaces; table 1 gives the percentage of the original number which left during any 10-minute period, as well as the mean microclimatic conditions.

It can be seen from figure 1 and table 1 that egress of mosquitoes was greatest during the 20 minutes following sunset. This was true in six of the seven experiments; only on one dark, rainy evening was the response different. On this occasion the greatest number left between 20 minutes before and 10 minutes after sunset. Behavior was similar in both shelters studied.

Table 2 gives data at 10- or 20-minute intervals concerning temperature, relative humidity, evaporation, and light intensity (light intensity in the open only), in the resting place and in the open. Means are to be found at the foot of table 1. These data, except for light intensity, are derived from the respective hygrothermographic records, evaporation indices being calculated in the manner outlined in the last section of this paper.

During the period of greatest activity at dusk, temperatures in the open were on the decline after reaching their maximum 3 to 4 hours before sunset. Outside humidities had been rising over a similar period. The decline and rise, respectively, of temperature and humidity within the resting place lagged about an hour or two behind that in the open. Evaporation rates began their decline at the same time the other climatic conditions changed their trends, but by the time the mosquitoes began their evening movement evaporation in the open had ceased, while in the resting place the rate had not greatly fallen. Indeed, during the few hours preceding sundown evaporation

indices (assuming still conditions) in the open were less than the rates in the shelter. Light intensities in the open declined from several thousand foot candles at noon to a mean of 48 foot candles at sunset, and at about 30 to 40 minutes after sundown no light could be measured with the meter used. It is very likely that intensities inside the shelter were proportional to those in the open.

A study of the data in table 2 indicates that of all the factors measured only light intensity in the open can be consistently correlated with the evening mosquito activity. Only this factor was undergoing marked change at the time of the initiation of the evening exodus, and it is the only factor immune to very considerable variation during precipitation. It was observed by the investigators that adults of *A. quadrimaculatus* do not leave their diurnal resting place during the temperature and evaporation decline and humidity increase which accompanies rain; the effect of rain is often greater on these factors than the effect of the coming of night. Significantly, light intensity at sunset on the night of early egress was 2 foot candles.

In order to determine whether decline in intensity of light is the stimulus initiating the evening movement out of the resting place, an experiment was designed in which a light was placed in one portion of shelter 2, several hours before sunset, so as to throw about 6 foot candles of light on the counting surface. The shelter was divided into two parts by an improvised partition in such a manner that half could be kept dark and used as a control. Counts were made in both halves as in the previous experiments, but were continued until later in the evening. Figure 2 shows the smoothed curves of the changes in density of mosquitoes on the lighted and unlighted surfaces for two such experiments. In the first experiment the screen between the lighted surface and the unlighted was much less efficient than in the second. At no time were mosquitoes attracted to the 60-watt Mazda lamp, which was powered by a portable generator.

The results of these experiments bear out the conclusion that light intensity decline was the chief stimulus initiating the evening movement. In the first experiment the mosquitoes on the lighted surface at no time showed an abrupt change in density (although a gradual decline was noted); the mosquitoes on the control surface left at the usual time. In the second experiment only about one-fifth of the mosquitoes on the lighted surface had left before 2 hours after sunset. At 9:05 p. m. the light was extinguished and in the ensuing 25 minutes density dropped by over three-fifths (fig. 2). It may be that the gradual decline noted in both experiments was due to unknown factors, but the writers believe that it was principally due to the unpreventable disturbance attending the counting operation.

TABLE 2.—Microclimatic conditions within and outside of evening exodus of *Anopheles quadrimaculatus* 1

		Minutes before and after sunset (S.)																					
		100	90	80	70	60	50	40	30	20	10	S.	10	20	30	40	50	60	70	80	90	100	
<b>Shelter 1, Aug. 4:</b>																							
Shelter temperature.....	80	79.5		79		78.5		78		76.5		75.5		74		73		72		71.5		71	
Outside temperature.....	52	80		78		74		76		74		72		71		70		69		68.5		68	
Shelter humidity.....	81	47		52		55		55		96		57		60		64		64		66		68	
Outside humidity.....	14.7	13.1		12.2		11.0		11.0		10.5		10.0		8.7		8.1		7.5		6.8		6.2	
Shelter evaporation.....	5.4	4.0		3.0		1.8		1.8		1.5		0		0		0		0		0		0	
Outside evaporation.....				800		500		400		200		120		4		0		0		0		0	
Outside light intensity.....																							
<b>Shelter 2, Aug. 6:</b>																							
Shelter temperature.....	88	88.5		87.5		86		87		86		86.5		84.5		83.5		83		82		81.5	
Outside temperature.....	85	80		82		78.5		80		78.5		77		76		75.5		75		74.5		74	
Shelter humidity.....	54	52		57		63		63		65		67		69		70		70		72		72	
Outside humidity.....	94	97		100		100		100		100		100		100		100		100		100		100	
Shelter evaporation.....	15.9	16.6		14.4		12.3		12.3		11.4		10.8		9.9		9.1		8.7		8.3		7.9	
Outside evaporation.....	1.5	1.4		0		0		0		0		0		0		0		0		0		0	
Outside light intensity.....		1,925		1,500		550		550		230		150		3		0		0		0		0	
<b>Shelter 2, Aug. 7:</b>																							
Shelter temperature.....	88.5	88		87.5		86.5		87		86.5		86		83.5		81		80		78.5		77	
Outside temperature.....	87	86		85		84		81		81		74		71		72		72		72		72	
Shelter humidity.....	84	50		52		59		59		64		60		62		67		68		72		77	
Outside humidity.....	84	92		100		100		100		100		100		100		100		100		100		100	
Shelter evaporation.....	16.0	17.2		16.1		13.4		13.4		11.7		12.9		11.7		9.3		8.6		6.9		5.8	
Outside evaporation.....	5.5	2.9		0		0		0		0		0		0		0		0		0		0	
Outside light intensity.....		2,250		1,575		950		500		230		124		4		0		0		0		0	
<b>Shelter 2, Aug. 8:</b>																							
Shelter temperature.....	86.5	86.5		86.5		85.5		86		85.5		85		84		82.5		81.5		80.5		80	
Outside temperature.....	88.5	84		82		78.5		80.5		78.5		77		76		75.5		75		74.5		74	
Shelter humidity.....	86	69		60		62		61		62		64		65		66		67		68		68	
Outside humidity.....	96	94		100		100		100		100		100		100		100		100		100		100	
Shelter evaporation.....	14.2	13.2		12.9		12.6		12.6		12.3		11.2		10.8		10.1		9.3		8.8		8.6	
Outside evaporation.....	3.0	2.1		0		0		0		0		0		0		0		0		0		0	
Outside light intensity.....		2,650		1,900		1,000		400		275		180		11		2		0		0		0	
<b>Shelter 2, Aug. 20:</b>																							
Shelter temperature.....	74	73.5		73		72.5		72.5		71.5		70.5		69		67.5		67		66		65.5	
Outside temperature.....	75	73		71		69		69		67.5		66		64.5		64		63		62		61	
Shelter humidity.....	64	66		73		76		76		78		78		77		77		78		80		82	
Outside humidity.....	94	94		96		100		100		100		100		100		100		100		100		100	
Shelter evaporation.....	7.4	7.3		5.9		4.9		4.5		4.5		4.3		4.4		4.2		3.9		3.5		3.0	
Outside evaporation.....	1.6	1.5		0		0		0		0		0		0		0		0		0		0	
Outside light intensity.....		2,250		1,650		1,300		900		550		44		10		2		0		0		0	
<b>Shelter 1, Aug. 25:</b>																							
Shelter temperature.....	82.5	82.5		82		81.5		82		81.5		81		80		79.5		79		78.5		77.5	
Outside temperature.....	85	84		83		82		81		81		79.5		78.5		77.5		77		76.5		76	
Shelter humidity.....	74	79		81		83		83		84		87		86		90		90		91		93	



## MOVEMENTS AT DAWN

*Method of study.*—Hygrothermographs were manipulated as in the dusk experiments, and selected surfaces were counted similarly once or twice before dawn and subsequently at 10- to 30-minute intervals. As before, light intensity readings in the open were taken at the same interval. The same two structures used in the dusk studies were employed, and counts were made on five occasions.

*Discussion.*—On August 5 and August 22 the greatest inward movement of mosquitoes into shelter 1 was during the 30 minutes just

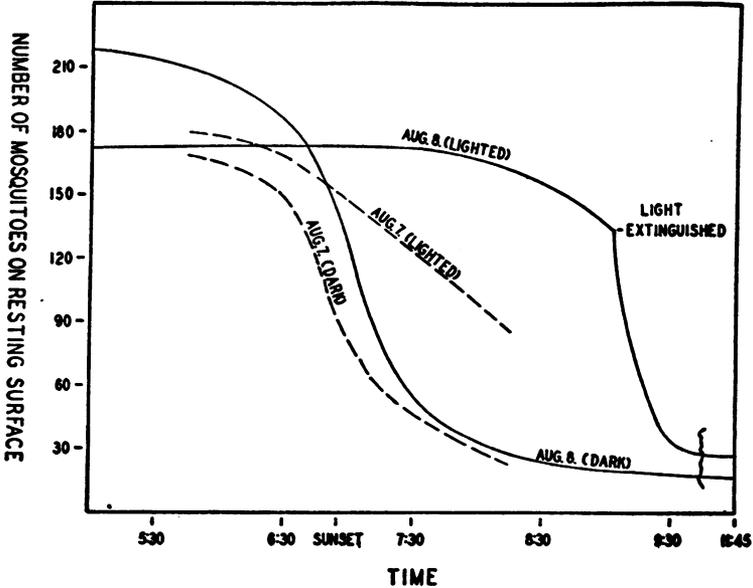


FIGURE 2.—Smoothed curves illustrating the effect of light on the dusk movement of *Anopheles quadrimaculatus*.

before and just after sunrise. (See fig. 3 which shows on smoothed curves the changes in density of mosquitoes on the selected surfaces.) In both cases there was very little temperature variation and relative humidities were still rising in the resting shelter, this rise being a continuation of the previous evening's rise. In the open, humidity was stable at saturation and temperatures were just beginning their morning rise (table 3). Evaporation was, therefore, greater in the resting place than in the open at the time of entry. On September 2 little movement of mosquitoes into shelter 1 was noted.

On August 6 in shelter 2 the number of mosquitoes on the selected surfaces (on this occasion two surfaces, *a* and *b* of fig. 3, were used) was observed to increase until late in the morning. This slow increase suggested to the investigators that mosquitoes might first enter the eaves and then be driven down by light entering the loft of the barn.

Subsequently, on August 18, counts were made in this same shelter both in the eaves and on one of the wall surfaces studied on August 6. Results of this experiment were inconclusive; mosquitoes continued to appear both in the eaves and below until late in the morning.

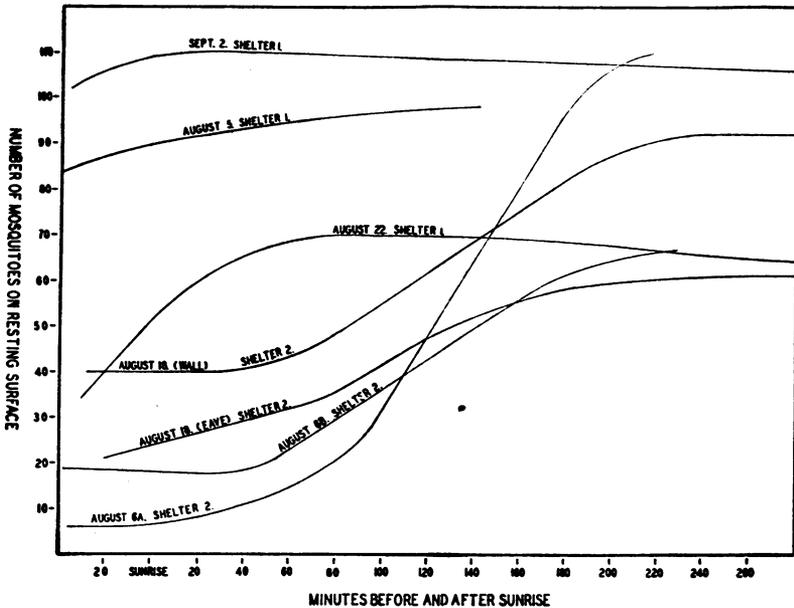


FIGURE 3.—Smoothed curves representing the early morning movement of *Anopheles quadrimaculatus* into the diurnal resting place.

TABLE 3.—Mean microclimatic conditions within and outside of diurnal resting places at time of dawn entry of *Anopheles quadrimaculatus*<sup>1</sup>

	Minutes before and after sunrise (S.)							
	40	20	8.	20	40	60	80	100
<b>Shelter 1:</b>								
Temperature.....	68	68	67.5	67.5	67.5	68	68	68.5
Humidity.....	91	92	92	95	96	97	96	96
Evaporation.....	1.7	1.4	1.4	1.0	.9	.7	.8	.8
<b>Shelter 2:</b>								
Temperature.....	69	69.5	69	68.5	69	69.5	70.5	71.5
Humidity.....	92	92	93	93	94	96	99	96
Evaporation.....	1.8	1.7	1.5	1.3	1.2	.6	.3	1.3
<b>Outside:</b>								
Temperature.....	65.5	65.5	65.5	66	66.5	67.5	69	71
Humidity.....	100	100	100	100	100	100	95	89
Evaporation.....	0	0	0	0	0	0	1.0	2.3
Light intensity.....	0	25	110	400	720	1,100	1,800	2,200

<sup>1</sup>Temperatures are in degrees Fahrenheit, humidities in percentage saturation, evaporation in vapor pressure deficit (in millimeters of mercury), and light intensity in foot candles in the open.

In the case of the second resting place, temperatures in the shelter were rising and humidities falling during the period of mosquito entry. However, this rise and fall was not nearly so decided as the coincident rise and fall in the instrument shelter (table 3). On the other hand,

the entrance of mosquitoes started before humidity fell below saturation outside and before outside temperatures had risen to any great degree. Evaporation, at the time of the initiation of the inward movement, was greater in the shelter than on the outside, but the inward movement was still going on when this situation was reversed.

More careful observations must be made before generalizations concerning the morning movements of mosquitoes into daytime resting places can be made, but the observations above give some leads which might be followed up. It appears that the inward movement is a gradual one dependent upon when sunlight (this, of course, affects the other climatic conditions) strikes the individual mosquito. This was indicated by the presence of mosquitoes on the outer surfaces of the barns many minutes after sunup, since these mosquitoes appeared to move only when the sunlight swept the surfaces. In the evening the mosquitoes which left the resting places were subject to uniform conditions within the shelter, and, as has been shown, left at the same time. At dawn mosquitoes in the open were subject to a variety of conditions depending upon the position they occupied with respect to shadow, and the stimulus which initiated the inward movement was not likely to act on the entire population at the same time. It seems significant that in the case of the shelter which was located in the open the inward movement was earlier and less gradual than in the case of the shelter located in deep shadow, as is evidenced by the differences of the curves in figure 3.

#### DAYTIME MICROCLIMATIC CONDITIONS

*Method of study.*—The microclimates of nine structures used as resting places by *Anopheles quadrimaculatus* were measured. The visible characteristics of these shelters are summarized in table 4. Hygrothermographs were again employed simultaneously in the resting place and in the instrument shelter. In table 5 maximum, minimum, and mean temperatures are tabulated at 2-hour intervals from 8 a. m. to 4 p. m., as are the corresponding values of relative humidity. These readings correspond with the tabulated light intensities taken at the same hours both within and without the shelter. The light measurements within the shelter were made by placing the quartz disk of the illumination meter on three different places on the surface where mosquitoes were resting. Means of these groups of three readings in actual foot candles are given in table 5. The intensities in the open were taken by placing the quartz disk parallel to the ground in an unshaded area, and are recorded in hundreds of foot candles. The same method of taking readings in the open was followed in the first two sections of the study.

Not only relative humidities, which bear little relation to actual evaporation, but also indices of relative evaporation are given in

order that conditions in the shelters can be compared with conditions prevailing elsewhere. For this index the authors use vapor pressure deficit in millimeters of mercury, basing this on the assumption that the body temperature of a mosquito at rest is the same as that of the surrounding atmosphere. In the diurnal shelter this is probably true, as metabolic activity, which might affect body temperature, is at a minimum, and an insect as small as a mosquito could probably not appreciably lower its body temperature by evaporation without upsetting the physiological water balance. Relations shown between evaporation in the shelter and outside are valid only for still conditions for, other things being equal, evaporation is greater in the wind.

TABLE 4.—*External characteristics of artificial diurnal resting places of Anopheles quadrimaculatus studied around Reelfoot Lake*

Location	Maximum number mosquitoes noted (estimated)	Distance (feet) from good breeding place	Distance (feet) from good blood source	Visible characteristics
Shelter 1. Wollaston barn.	10,000	500	(1)	Barn, tin roof, closed loft, dirt floor, not shaded. Cows and swine at night.
Shelter 2. McQueen barn.	8,000	200	50	Barn, shingle roof, open loft, wooden floor, well shaded. No stock.
Shelter 3. Powell barn.	4,000	2,000	-----	Barn, shingle roof, closed loft, dirt floor (mostly), not shaded. Cows.
Shelter 4. Morton barn.	4,000	1,200	-----	Barn, shingle roof, half-open loft, wooden floor (mostly), half shaded. Mules.
Shelter 5. Biological station.	400	1,000	200	Under part of frame building, concrete floor, whitewashed rafters, lattice-work sides.
Shelter 6. Hammer Scott barn.	3,000	500	-----	Barn, shingle roof, loft open, dirt floor (mostly), unshaded. Cows.
Shelter 7. Miller barn.	4,000	1,200	-----	Barn, shingle roof, open loft, dirt floor, shaded. Cows.
Shelter 8. Spout Springs barn.	4,000	3,000	-----	Barn, shingle roof, closed loft, dirt and wooden floor, unshaded. Cows.
Shelter 9. Bridge at Walnut Log.	200	500	400	Small wooden bridge with wide cracks, over intermittent brook.

<sup>1</sup>Stock in shelter at least at night.

Maximum, minimum, and mean values for evaporation are given in table 5.

The measurements of temperature, humidity, evaporation, and light intensity outlined above were made on 24 rainless days during a 38-day period spanning August and September.

*Discussion.*—Table 6 summarizes the difference between climatic conditions in the shelter and in the open; mean differences and the probable error of these means are given. Their differences are graphically shown in figure 4 where means of temperature, humidity, and evaporation from the resting place and the instrument shelter are plotted.

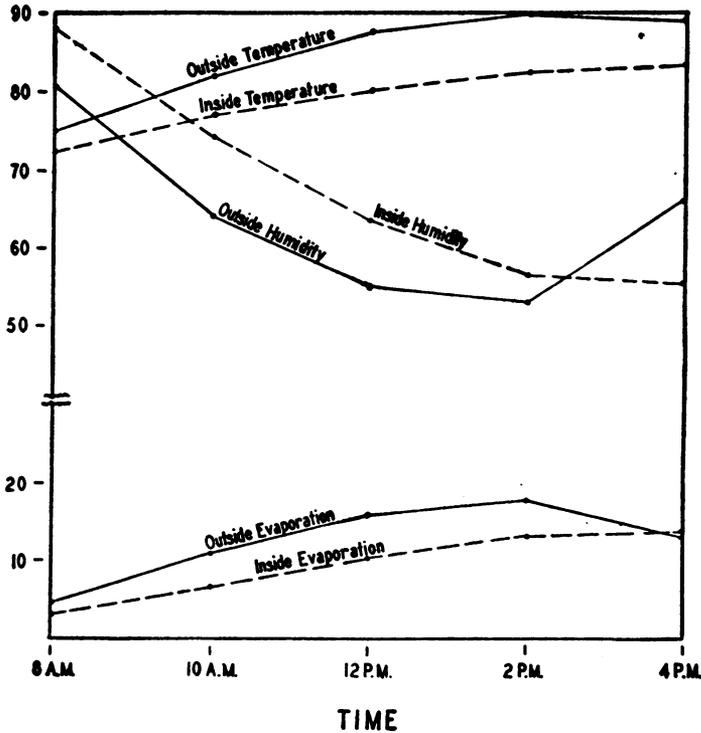


FIGURE 4.—Means of temperature, humidity, and evaporation inside the diurnal resting place and in the instrument shelter. Temperature is in degrees Fahrenheit, humidity is percentage of saturation, and evaporation is expressed as the vapor pressure deficit.

TABLE 5.—Microclimatic conditions in instrument shelter and in resting places at 2-hour intervals

	Instrument shelter						Resting place					
	8 a. m.	10 a. m.	12 noon	2 p. m.	4 p. m.	Mean	8 a. m.	10 a. m.	12 noon	2 p. m.	4 p. m.	Mean
Temperature, ° F.:												
Mean	75	82	87.5	90	89	84.5	72.5	77	80	82.5	83.5	79
Maximum	82	90	96	98	99	93	81	85	92	93	93	88
Minimum	66	70	71	79	78	73	64	64	66	69	71	67
Relative humidity:												
Mean	80.5	64	55	53	66	64	88	74.5	63.5	56.5	55.5	67.5
Maximum	100	87	72	72	100	83	100	93	87	76	78	84
Minimum	62	40	40	38	47	47	70	59	45	40	36	51
Evaporation:												
Mean	4.5	10.7	15.8	17.7	12.8	12.3	2.7	6.4	10.2	13.1	13.6	9.2
Maximum	8.3	19.2	23.6	26.6	24.1	18.4	6.6	11.1	20.3	20.7	20.0	14.7
Minimum	0	3.6	5.9	8.6	0	5.4	0	1.7	3.1	4.6	5.8	3.7
Light intensity: <sup>1</sup>												
Mean	47.1	100	125.5	121	49	88.5	1.8	2.8	3.1	3.4	2.6	2.8
Maximum	71	160	170	168	70	116	7	8	8	8	9	7
Minimum	6	12	44	70	20	51	0	1	0	0	0	0

<sup>1</sup> Outside intensities in hundreds of foot candles; inside intensities in actual foot candles.

TABLE 6.—Means of differences between inside microclimatic conditions and outside conditions measured simultaneously

	8 a. m.	10 a. m.	Noon	2 p. m.	4 p. m.	Mean
Temperature, ° F.:						
Mean.....	-2.21	-5.08	-7.25	-7.29	-5.67	-5.71
PE.....	±0.304	±0.325	±0.287	±0.318	±0.384	±0.290
Relative humidity:						
Mean.....	+7.17	+10.58	+8.92	+4.00	-10.50	+3.67
PE.....	±1.586	±0.952	±0.943	±0.957	±1.504	±0.950
Evaporation:						
Mean.....	-1.75	-4.31	-5.59	-4.63	+0.84	-3.10
PE.....	±0.287	±0.370	±0.299	±0.357	±0.583	±0.296

PE=Probable error.

- or + signs signify that measurements in the shelter were less or greater, respectively, than the same measurements outside.

It can be seen that during the early and late daylight hours the conditions outside and inside the diurnal resting place are most nearly similar. During the hot portion of the day temperatures and evaporation rates are more moderate inside the shelter—a condition which evidently makes the shelters suitable for *Anopheles quadrimaculatus*. Relative humidity is inversely higher.

Evaporation rates are definitely lower in the resting places than in the instrument shelter although the difference amounts to only about 20 or 30 percent. A consideration of the evaporation rate which would hold if the mosquito were struck by direct sunlight, however, shows what a great protective effect the diurnal shelter affords. The temperature of *A. quadrimaculatus* adults subjected to the radiant energy of the sun was not measured, but basing an estimate on work on other insects, it seems likely that a temperature of from 40 to 42 degrees centigrade might be reached (Wigglesworth (3), pages 359-360). Under such conditions the mean noon evaporation rate in the shelter would be only a small fraction of the rate in the open. It should be emphasized, too, that the above comparisons are based on still conditions. The daytime shelter affords protection from wind, which would have the effect of further increasing the outside evaporation rates.

No attempt was made to measure the variation of conditions within the shelters, but instruments were in each case placed in that part of the shelter favored by mosquitoes.

#### SUMMARY AND CONCLUSIONS

1. The evening egress of mosquitoes from the diurnal resting places occurs most rapidly in the 20 minutes after sunset. The only microclimatic condition which could be correlated with this egress was the covariant light intensity; at the time of most rapid exodus, light intensities in the open varied from a mean of about 48 foot candles at sunset to 2 foot candles 20 minutes later.

2. An experiment on the effect of artificial light on mosquitoes in the daytime resting places supported the conclusion that light intensity is the principal factor initiating the evening movement from the shelter to the open. It was found that a large portion of the mosquitoes could be made to remain through the evening in a lighted diurnal shelter.

3. During the hours just after sunrise *Anopheles quadrimaculatus* females tend to enter the diurnal resting place, but this inward movement is gradual, not concerted, and seems to depend upon when direct sunlight strikes the mosquitoes in the open.

4. During the daylight hours temperatures and humidities within the diurnal resting places of *A. quadrimaculatus* were found to be lower and higher, respectively, than outside conditions measured simultaneously. During the most severe part of the day temperatures were on the average 7° F. lower and humidities 8 percent higher than the outside measurements. Evaporation within the resting place was found to be only about two-thirds the outside rate.

5. Evaporation rates inside the diurnal resting places would only be a small percentage of the rate which would hold if a mosquito were exposed to the direct light of the sun and the direct action of the wind.

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## ROCKY MOUNTAIN SPOTTED FEVER: DURATION OF POTENCY OF TICK-TISSUE VACCINE <sup>1</sup>

By R. R. PARKER, *Director, Rocky Mountain Laboratory*, and EDWARD A. STEINHAUS, *Associate Bacteriologist, United States Public Health Service*

Three lots of Rocky Mountain spotted fever vaccine of the Spencer-Parker type,<sup>2</sup> one manufactured in 1928 and two in 1929, were retested for potency on March 9, 1942. Their respective protective values appeared to be undiminished. In the interim they had been stored at a temperature range of from 34° to 40° F.

The only previous tests of duration of potency were reported by Spencer and Parker<sup>3</sup> in 1930. The results of these earlier tests suggested "that some batches of vaccine will retain full potency for more than a year when kept in the ice box."

<sup>1</sup> From the Rocky Mountain Laboratory (Hamilton, Mont.), Division of Infectious Diseases, National Institute of Health.

<sup>2</sup> Prepared from the infected tissues of the Rocky Mountain wood tick, *Dermacentor andersoni*.

<sup>3</sup> Spencer, R. R., and Parker, R. R.: Studies on Rocky Mountain spotted fever: Improved method of manufacture of the vaccine and a study of its properties. *Hygienic Laboratory Bulletin* 154, pp. 63-72, 1930.

The method of testing of potency in 1942 was identical with that used for the original tests. Each lot of vaccine was tested by injecting each of 6 male guinea pigs subcutaneously with 1 cc. Twelve days later each test animal and each of 4 control animals received intraperitoneally 1 cc. of citrated heart blood taken on the third day of fever from a guinea pig ill with a highly virulent western Montana strain of Rocky Mountain spotted fever (the average cc. contains 500 infectious doses). The vaccine is considered usable if a minimum of 4 of the 6 test animals are completely protected and the control guinea pigs have typical temperature curves and characteristic scrotal lesions. This criterion of potency is purely arbitrary, but has proved satisfactory in testing nearly 10,000 lots of vaccine prepared during the past 16 years. Records of the control guinea pigs used in 1928 and 1930 are not available, but they were obviously satisfactory since the 3 lots were released for administration.

*Vaccine 347.*—Prepared May 1, 1928. First potency test May 2, 1928: five guinea pigs completely protected; one had fever lasting 3 days. Potency retest March 9, 1942, approximately 14 years after manufacture: five guinea pigs completely protected; one had fever lasting one day.

*Vaccine 467.*—Prepared December 12, 1929. First potency test February 14, 1930: four guinea pigs completely protected; one valueless because of pneumonia; one lost during test. Potency retest March 9, 1942, approximately 12 years after manufacture: four guinea pigs completely protected; two guinea pigs had fever which lasted 2 days.

*Vaccine 470.*—Prepared December 31, 1929. First potency test January 10, 1930: two guinea pigs were completely protected; two had fever lasting 1 day (for one this fever was obviously not due to Rocky Mountain spotted fever), one had fever lasting 2 days, and one had fever lasting 5 days. Potency retest March 9, 1942, approximately 12 years later: six guinea pigs completely protected.

*Virus controls.*—Of the four guinea pigs used as controls on the virus employed for immunity tests of March 9, 1942, all had characteristic temperature curves and scrotal lesions; two died of spotted fever.

It is perhaps of interest that for a number of years the return of unused vaccine has been requested each fall. (Vaccine is dispensed for the most part during the first 6 months of the calendar year.) This returned vaccine consists of remnants of numerous lots (from a few to several hundred cc.), some prepared the same year as issued, others 1 and 2 years old. Much of it has been kept for months at room temperature. These remnants have been pooled in lots of 1,000 to 3,000 cc. and retested for potency and sterility, and reissued if satisfactory for use. Only rarely has a pooled lot failed to meet the potency requirements.

CONCLUSION

Individual lots of Rocky Mountain spotted fever vaccine prepared from the tissues of infected *Dermacentor andersoni* may retain their protective value for at least as long as 12 to 14 years.

**DISABLING MORBIDITY AMONG INDUSTRIAL WORKERS, THIRD QUARTER OF 1942, WITH A NOTE ON THE OCCURRENCE OF THE RESPIRATORY DISEASES, 1933-42<sup>1</sup>**

By W. M. GAFAFER, *Senior Statistician, United States Public Health Service*

The data on the frequency of sickness and nonindustrial injuries causing disability for 8 consecutive calendar days or longer during the third quarter and the first 9 months of 1941 and 1942, presented

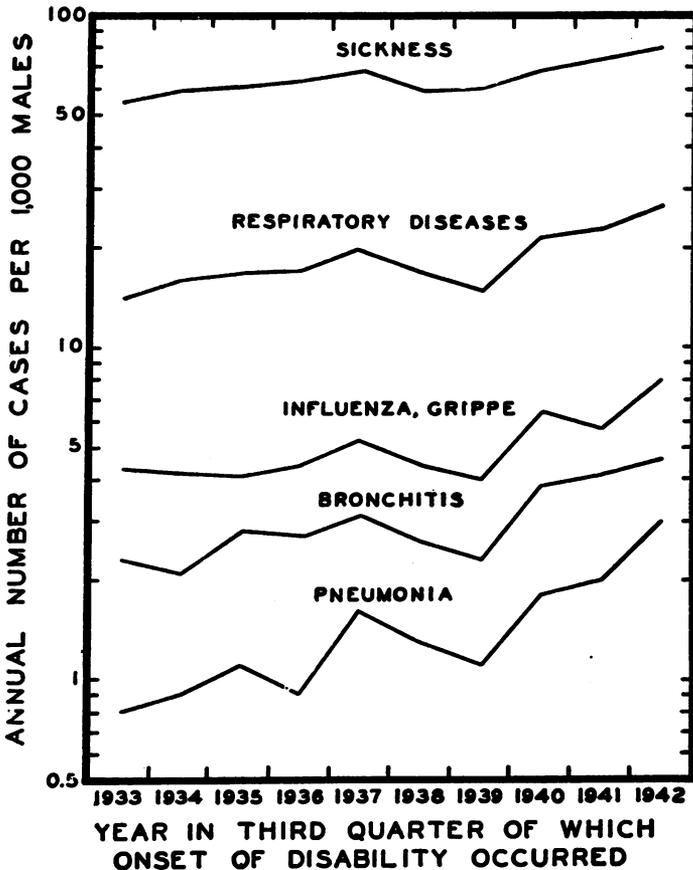


FIGURE 1.—Frequency of disabling cases of respiratory diseases lasting 8 consecutive calendar days or longer among MALE employees in various industries, the third quarters of 1933-42, inclusive. (Vertical logarithmic scale.)

<sup>1</sup> From the Division of Industrial Hygiene, National Institute of Health. For the second quarter of 1942 see PUBLIC HEALTH REPORTS, 57: 1620-1622 (1942).

**TABLE 1.—Frequency of disabling cases of sickness and nonindustrial injuries lasting 8 consecutive calendar days or longer among MALE employees in various industries, by cause, the third quarter of 1942 compared with the third quarter of 1941, and the first 9 months of 1942 compared with the first 9 months of the years 1937-41, inclusive**

Cause (numbers in parentheses are disease title numbers from the International List of Causes of Death, 1939)	Annual number of cases per 1,000 males				
	Third quarter		First 9 months		
	1942	1941	1942	1941	1937-41
Sickness and nonindustrial injuries <sup>1</sup> .....	90.9	86.1	104.1	105.5	97.9
Nonindustrial injuries (169-195).....	12.2	13.4	11.7	11.9	11.4
Sickness <sup>1</sup> .....	78.7	72.7	92.4	93.6	86.5
Respiratory diseases.....	26.7	22.6	39.0	44.7	38.8
Influenza and grippe (33).....	8.0	5.7	14.6	21.7	18.9
Bronchitis, acute and chronic (106).....	4.6	4.1	6.3	5.6	4.8
Diseases of the pharynx and tonsils (115b, 115c).....	4.6	4.7	5.4	6.0	5.3
Pneumonia, all forms (107-109).....	3.0	2.0	5.0	4.1	3.3
Tuberculosis of the respiratory system (13).....	.8	.9	.7	.8	.8
Other respiratory diseases (104, 105, 110-114).....	5.7	5.2	7.0	6.5	5.7
Digestive diseases.....	17.3	16.5	16.6	15.2	14.4
Diseases of the stomach, except cancer (117, 118).....	5.1	4.4	4.7	4.0	3.9
Diarrhea and enteritis (120).....	2.6	2.3	1.9	1.6	1.3
Appendicitis (121).....	4.7	5.1	5.1	5.1	4.8
Hernia (122a).....	1.8	1.5	1.8	1.6	1.6
Other digestive diseases (115a, 115d, 116, 122b-129).....	3.1	3.2	3.1	2.9	2.8
Nonrespiratory-nondigestive diseases.....	32.8	30.1	34.8	30.6	30.6
Diseases of the heart and arteries, and nephritis (90-99, 102, 130-132).....	3.4	3.5	4.3	4.1	4.2
Other genitourinary diseases (133-138).....	2.5	2.8	2.5	2.4	2.4
Neuralgia, neuritis, and sciatica (87b).....	1.9	1.8	2.2	2.0	2.2
Neurasthenia and the like (part of 84d).....	1.2	1.1	1.1	1.0	1.0
Other diseases of the nervous system (80-85, 87, except part of 84d, and 87b).....	1.1	1.2	1.1	1.2	1.1
Rheumatism, acute and chronic (58, 59).....	3.6	3.5	4.0	4.0	4.0
Diseases of the organs of locomotion, except diseases of the joints (156b).....	2.7	2.7	3.0	2.9	2.8
Diseases of the skin (151-153).....	3.9	3.8	3.0	2.9	3.0
Infectious and parasitic diseases <sup>2</sup> (1-12, 14-24, 26-29, 31, 32, 34-44).....	1.9	2.1	2.8	2.6	2.5
All other diseases (45-57, 60-79, 88, 89, 100, 101, 103, 154, 155, 156a, 157, 162).....	10.6	7.6	10.8	7.5	7.4
Ill-defined and unknown causes (200).....	1.9	3.5	2.0	3.1	2.7
Average number of males covered in the record.....	264,945	238,407	260,677	227,704	953,078
Number of organizations.....	22	22	.....	.....	.....

<sup>1</sup> Industrial injuries, venereal diseases, and a few numerically unimportant causes of disability are not reported.

<sup>2</sup> Except influenza, respiratory tuberculosis, and the venereal diseases.

**TABLE 2.—Frequency of disabling cases of respiratory diseases lasting 8 consecutive calendar days or longer among MALE employees in various industries, the third quarters of 1933-42, inclusive**

Year in third quarter of which onset of disability occurred	Rate or average annual number of cases per 1,000 males					Ratio of rate to mean for 1933-42				
	All sickness	Respiratory diseases	Influenza, grippe	Bronchitis, acute and chronic	Pneumonia, all forms	All sickness	Respiratory diseases	Influenza, grippe	Bronchitis, acute and chronic	Pneumonia, all forms
1933-42 (mean).....	64.2	18.5	5.1	3.0	1.5	1.00	1.00	1.00	1.00	1.00
1933.....	54.8	14.0	4.3	2.3	.8	.85	.76	.85	.76	.55
1934.....	59.1	15.9	4.2	2.1	.9	.92	.86	.83	.69	.62
1935.....	60.7	16.6	4.1	2.8	1.1	.95	.90	.81	.92	.76
1936.....	63.2	17.0	4.4	2.7	.9	.98	.92	.87	.89	.62
1937.....	66.9	19.5	5.2	3.1	1.6	1.04	1.05	1.03	1.02	1.10
1938.....	59.0	16.8	4.4	2.6	1.3	.92	.91	.87	.86	.90
1939.....	59.6	14.7	4.0	2.3	1.1	.93	.79	.79	.76	.76
1940.....	67.2	21.2	6.4	3.8	1.8	1.05	1.15	1.26	1.25	1.24
1941.....	72.7	22.6	5.7	4.1	2.0	1.13	1.22	1.12	1.35	1.38
1942.....	78.7	26.7	8.0	4.6	3.0	1.23	1.44	1.58	1.51	2.07

in table 1, are derived from analyses of periodic reports from industrial sick benefit associations, group insurance plans, and company relief departments.

While the rate for all sickness for the third quarter of 1942 represents only an 8-percent increase when compared with the corresponding rate for 1941, comparisons of the 1942 rate with the corresponding rates for previous years show the following percentage increases:

1933.....	44	1936.....	25	1939.....	32	1933-42.....	23
1934.....	33	1937.....	18	1940.....	17		
1935.....	30	1938.....	33	1941.....	8		

Thus the third quarter frequency for all sickness when compared with the corresponding rate for 1933 (54.8) shows an excess of 44 percent and when compared with the mean for 1933-42 (64.2) the excess is 23 percent.

*Respiratory diseases, third quarters, 1933-42.*—Interest in table 1 centers chiefly around the third quarter increase of 18 percent in the frequency of the respiratory diseases which reflects principally the following increases: influenza and grippe, 40 percent; bronchitis, 12 percent; and pneumonia, 50 percent.

Of interest are the rates yielded by these causes during the past 10 years. Table 2 shows the pertinent data and includes the third quarter rates for the three causes referred to, for the respiratory group of diseases, and for all sickness; the table also shows the ratio of each rate to the corresponding mean for the 10-year period. The actual rates are shown graphically in figure 1. It will be observed that each cause and cause group presents an increasing trend and that in each instance the third quarter rate for 1942 is the maximum for the 10-year period.

## LIST OF STATE AND INSULAR HEALTH OFFICERS

(As of January 15, 1943)

State	Name and designation	Location
Alabama.....	Dr. B. F. Austin, State Health Officer.	Montgomery.
Alaska.....	Dr. Walter W. Council, Commissioner of Health.	Juneau.
Arizona.....	Dr. G. F. Manning, State Superintendent of Health.	Phoenix.
Arkansas.....	Dr. William B. Grayson, State Health Officer.	Little Rock.
California.....	Dr. Wilton L. Halverson, State Director of Public Health.	San Francisco.
Colorado.....	Dr. R. L. Cleere, Secretary, State Board of Health.	Denver.
Connecticut.....	Dr. Stanley H. Osborn, State Commissioner of Health.	Hartford.

<i>State</i>	<i>Name and designation</i>	<i>Location</i>
Delaware.....	Dr. Edwin Cameron, Executive Secretary, State Board of Health.	Dover.
District of Columbia.	Dr. George C. Ruhland, District Health Officer.	Washington.
Florida.....	Dr. Henry Hanson, State Health Officer	Jacksonville.
Georgia.....	Dr. T. F. Abercrombie, State Director of Public Health.	Atlanta.
Hawaii.....	Dr. M. F. Haralson, Territorial Commissioner of Public Health, Hawaii Board of Health.	Honolulu.
Idaho.....	Dr. E. L. Berry, State Director of Public Health.	Boise.
Illinois.....	Dr. Roland R. Cross, State Director of Public Health.	Springfield.
Indiana.....	Dr. Thurman B. Rice, Acting State Director of Public Health.	Indianapolis.
Iowa.....	Dr. Walter L. Bierring, State Commissioner of Health.	Des Moines.
Kansas.....	Dr. F. C. Beelman, Secretary and Executive Officer, State Board of Health.	Topeka.
Kentucky.....	Dr. A. T. McCormack, State Health Commissioner.	Louisville.
Louisiana.....	Dr. David E. Brown, President, State Board of Health.	New Orleans.
Maine.....	Dr. Roscoe L. Mitchell, Director, State Department of Health and Welfare.	Augusta.
Maryland.....	Dr. Robert H. Riley, State Director of Health.	Baltimore.
Massachusetts.....	Dr. Paul J. Jakmauh, State Commissioner of Public Health.	Boston.
Michigan.....	Dr. H. Allen Moyer, State Health Commissioner.	Lansing.
Minnesota.....	Dr. A. J. Chesley, Secretary, State Board of Health.	St. Paul.
Mississippi.....	Dr. Felix J. Underwood, Secretary, State Board of Health.	Jackson.
Missouri.....	Dr. James Stewart, State Health Commissioner.	Jefferson City.
Montana.....	Dr. W. E. Cogswell, Secretary, State Department of Public Health.	Helena.
Nebraska.....	Dr. C. A. Selby, State Director of Health.	Lincoln.
Nevada.....	Dr. Edward E. Hamer, State Health Officer.	Carson City.
New Hampshire...	Dr. A. L. Frechette, Secretary, State Board of Health.	Concord.
New Jersey.....	Dr. J. Lynn Mahaffey, State Director of Health.	Trenton.
New Mexico.....	Dr. James R. Scott, Director, Department of Public Health.	Santa Fe.
New York.....	Dr. Edward S. Godfrey, Jr., State Commissioner of Health.	Albany.
North Carolina....	Dr. Carl V. Reynolds, State Health Officer.	Raleigh.

<i>State</i>	<i>Name and designation</i>	<i>Location</i>
North Dakota.....	Dr. Frank J. Hill, Acting State Officer.	Bismarck.
Ohio.....	Dr. R. H. Markwith, State Director of Health.	Columbus.
Oklahoma.....	Dr. Grady F. Mathews, State Health Commissioner.	Oklahoma City.
Oregon.....	Dr. Frederick D. Stricker, State Health Officer.	Portland.
Pennsylvania.....	Dr. A. H. Stewart, Secretary of Health.	Harrisburg.
Puerto Rico.....	Dr. A. Fernos Isern, Health Commissioner.	San Juan.
Rhode Island.....	Dr. Edward A. McLaughlin, State Director of Public Health.	Providence.
South Carolina.....	Dr. James A. Hayne, State Health Officer.	Columbia.
South Dakota.....	Dr. J. F. D. Cook, Superintendent, State Board of Health.	Pierre.
Tennessee.....	Dr. W. C. Williams, State Commissioner of Public Health.	Nashville.
Texas.....	Dr. George W. Cox, State Health Officer.	Austin.
Utah.....	Dr. William M. McKay, State Health Commissioner.	Salt Lake City.
Vermont.....	Dr. Charles F. Dalton, Secretary, State Board of Health.	Burlington.
Virgin Islands.....	Dr. Knud Knud-Hansen, Commissioner of Public Health.	Charlotte Amalie.
Virginia.....	Dr. I. C. Riggins, State Health Commissioner.	Richmond.
Washington.....	Dr. Donald G. Evans, Director, State Department of Health.	Seattle.
West Virginia.....	Dr. C. F. McClintic, State Commissioner of Health.	Charleston.
Wisconsin.....	Dr. Carl N. Neupert, State Health Officer.	Madison.
Wyoming.....	Dr. M. C. Keith, State Health Officer.	Cheyenne.

### INCIDENCE OF HOSPITALIZATION, DECEMBER 1942

Through the cooperation of the Hospital Service Plan Commission of the American Hospital Association, data on hospital admissions among about 8,000,000 members of Blue Cross Hospital Service Plans are presented monthly. These plans provide prepaid hospital service. The data cover about 60 hospital service plans scattered throughout the country, mostly in large cities.

Item	December	
	1942	1941
1. Number of plans supplying data.....	65	58
2. Number of persons eligible for hospital care.....	9,483,924	7,283,736
3. Number of persons admitted for hospital care.....	75,195	59,436
4. Incidence per 1,000 persons, annual rate, during current month (daily rate X 365).....	93.3	96.0
5. Incidence per 1,000 persons, annual rate for the 12 months ending December 31.....	107.9	107.0

**DEATHS DURING WEEK ENDED JANUARY 23, 1943**

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

	Week ended Jan. 23, 1943	Correspond- ing week, 1942
<b>Data for 87 large cities of the United States:</b>		
Total deaths.....	9,782	9,114
Average for 3 prior years.....	9,599	-----
Total deaths, first 3 weeks of year.....	30,270	28,184
Deaths under 1 year of age.....	686	516
Average for 3 prior years.....	513	-----
Deaths under 1 year of age, first 3 weeks of year.....	2,190	1,699
<b>Data from industrial insurance companies:</b>		
Policies in force.....	65,281,877	64,888,248
Number of death claims.....	14,910	13,533
Death claims per 1,000 policies in force, annual rate.....	11.9	10.9
Death claims per 1,000 policies, first 3 weeks of year, annual rate.....	11.2	10.3

# PREVALENCE OF DISEASE

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

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## UNITED STATES

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### REPORTS FROM STATES FOR WEEK ENDED JANUARY 30, 1943

#### Summary

Reports for the current week show an incidence above the corresponding 5-year (1938-42) median for only three (measles, meningococcus meningitis, and poliomyelitis) of the nine common communicable diseases included in the following tables.

The total number of meningococcus meningitis cases reported decreased from 354 to 337 for the current week, but increases occurred in a number of States. The largest numbers reported, with last week's figures in parentheses, are as follows: New York, 29 (48)<sup>1</sup>; California, 28 (30); Rhode Island, 23 (25); Maryland, 20 (13); Virginia, 18 (19); Pennsylvania, 15 (12); Missouri, 14 (5); New Jersey, 13 (8); Wisconsin, 13 (1); Washington, 13 (2).

There were 4,852 cases of influenza reported for the week, as compared with 4,387 for the preceding week and a 5-year median of 4,899. The largest numbers continued to be reported in Texas (1,900), South Carolina (678), and Virginia (567). Alabama reported the next largest number, 379 cases.

A total of 10,887 cases of measles was reported for the week, as compared with 8,807 for the preceding week and a 5-year median of 10,844. With reports, respectively, of 2,458 and 1,395 cases, Pennsylvania and New York contributed 35 percent of the current total.

The number of poliomyelitis cases reported increased from 25 for the preceding week to 31. The corresponding 5-year median is 26. Of the current total, 9 cases were reported in California, 5 in Texas, and 3 in Massachusetts.

Other reports for the week include 3 cases of anthrax, 222 of dysentery, 10 of infectious encephalitis, 3 of leprosy, 19 of tularemia, and 44 of endemic typhus fever.

Deaths during the current week in 90 large cities of the United States aggregated 10,181, as compared with 10,066 for the preceding week. The 3-year average for the corresponding weeks, 1940-42, is 9,812. The accumulated total for the first 4 weeks of 1943 is 41,264 as compared with 38,052 for the corresponding period in 1942.

*Telegraphic morbidity reports from State health officers for the week ended January 30, 1943, and comparison with corresponding week of 1942 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 1938- 42	Week ended—		Median 1938- 42	Week ended—		Median 1938- 42	Week ended—		Median 1938- 42
	Jan. 30, 1943	Jan. 31, 1942		Jan. 30, 1943	Jan. 31, 1942		Jan. 30, 1943	Jan. 31, 1942		Jan. 30, 1943	Jan. 31, 1942	
<b>NEW ENG.</b>												
Maine.....	0	0	0	-----	1	10	6	282	143	12	1	0
New Hampshire.....	0	1	0	-----	-----	1	34	3	6	2	0	0
Vermont.....	0	3	0	-----	-----	-----	324	7	22	0	0	0
Massachusetts.....	2	4	4	-----	-----	-----	496	227	227	10	4	2
Rhode Island.....	0	2	0	-----	-----	-----	22	117	7	23	0	0
Connecticut.....	0	0	2	14	5	5	372	107	107	3	1	1
<b>MID. ATL.</b>												
New York.....	12	24	28	114	113	116	1,395	514	564	29	10	4
New Jersey.....	1	8	12	24	13	19	431	0	28	13	2	1
Pennsylvania.....	8	13	26	2	-----	-----	2,458	1,137	1,137	15	7	7
<b>E. NO. CEN.</b>												
Ohio.....	12	11	23	9	15	15	111	152	152	5	3	2
Indiana.....	8	14	18	6	50	25	192	61	61	8	0	1
Illinois.....	14	25	33	4	13	35	273	120	120	7	0	0
Michigan <sup>1</sup> .....	6	2	8	1	2	2	135	141	427	6	1	1
Wisconsin.....	6	3	3	93	51	51	449	241	286	13	0	0
<b>W. NO. CEN.</b>												
Minnesota.....	5	2	3	2	3	4	19	613	235	2	0	0
Iowa.....	3	3	5	5	6	6	86	139	109	0	1	0
Missouri.....	6	5	5	6	5	33	96	55	26	14	1	2
North Dakota.....	1	1	3	23	5	6	42	117	18	0	0	0
South Dakota.....	6	0	3	-----	1	2	154	2	5	1	0	0
Nebraska.....	0	2	2	18	-----	1	201	58	28	1	0	0
Kansas.....	5	7	7	10	7	25	101	246	223	6	0	0
<b>SO. ATL.</b>												
Delaware.....	0	0	1	-----	-----	-----	12	1	1	1	0	0
Maryland <sup>1</sup> .....	9	11	6	13	5	47	32	259	26	20	4	1
Dist. of Col.....	1	0	3	4	1	3	51	11	11	4	4	0
Virginia.....	5	10	12	567	392	617	122	168	168	18	6	5
West Virginia.....	8	3	10	15	34	41	4	369	54	0	1	2
North Carolina.....	11	20	20	12	66	66	14	633	565	8	0	2
South Carolina.....	4	7	8	678	647	711	7	88	25	11	1	1
Georgia.....	7	12	8	154	183	183	27	330	63	5	0	0
Florida.....	11	6	6	7	10	13	11	75	72	1	3	1
<b>E. SO. CEN.</b>												
Kentucky.....	4	7	8	19	6	46	226	35	48	5	1	2
Tennessee.....	1	2	5	105	85	185	133	48	48	2	3	2
Alabama.....	13	15	12	379	644	644	11	62	81	7	3	3
Mississippi <sup>1</sup> .....	11	7	5	-----	-----	-----	-----	-----	-----	8	0	1
<b>W. SO. CEN.</b>												
Arkansas.....	5	8	10	150	267	267	120	204	63	2	0	1
Louisiana.....	8	15	10	7	26	26	69	39	3	7	1	1
Oklahoma.....	12	11	11	141	173	217	11	403	13	0	1	1
Texas.....	70	53	53	1,900	1,685	1,685	147	1,119	102	12	1	3
<b>MOUNTAIN</b>												
Montana.....	2	2	2	25	14	14	84	77	32	0	0	0
Idaho.....	0	1	1	-----	1	1	97	25	25	1	0	0
Wyoming.....	0	0	1	43	37	2	21	20	10	0	1	0
Colorado.....	9	13	10	113	50	45	230	166	57	3	0	0
New Mexico.....	3	4	2	4	-----	10	21	100	84	1	0	0
Arizona.....	3	4	4	155	131	131	15	150	10	1	0	0
Utah <sup>1</sup> .....	1	0	0	9	15	15	516	40	40	4	0	0
Nevada.....	0	0	-----	1	-----	-----	8	3	-----	0	0	-----
<b>PACIFIC</b>												
Washington.....	9	0	2	1	58	13	810	20	82	13	0	0
Oregon.....	4	2	2	35	24	53	448	87	87	7	0	1
California.....	35	11	24	89	155	155	243	1,618	389	28	4	2
Total.....	341	354	383	4,852	4,899	4,899	10,887	10,489	10,844	339	65	55
4 weeks.....	1,355	1,481	1,829	17,421	16,925	16,925	36,101	36,328	36,655	1,280	230	210

See footnotes at end of table.

Telegraphic morbidity reports from State health officers for the week ended January 30, 1943, and comparison with corresponding week of 1942 and 5-year median—  
Continued

Division and State	Poliomyelitis			Scarlet fever			Smallpox			Typhoid and paratyphoid fever		
	Week ended—		Median 1938-42	Week ended—		Median 1938-42	Week ended—		Median 1938-42	Week ended—		Median 1938-42
	Jan. 30, 1943	Jan. 31, 1942		Jan. 30, 1943	Jan. 31, 1942		Jan. 30, 1943	Jan. 31, 1942		Jan. 30, 1943	Jan. 31, 1942	
<b>NEW ENG.</b>												
Maine.....	1	0	0	8	21	17	0	0	0	0	0	1
New Hampshire.....	0	1	0	11	25	8	0	0	0	1	0	0
Vermont.....	0	0	0	4	4	11	0	0	0	0	1	0
Massachusetts.....	3	1	0	416	324	194	0	0	0	2	2	2
Rhode Island.....	0	0	0	29	10	10	0	0	0	0	0	0
Connecticut.....	0	0	0	65	31	74	0	0	0	2	0	0
<b>MID. ATL.</b>												
New York.....	1	3	1	416	388	556	0	0	0	3	3	6
New Jersey.....	0	1	1	88	104	177	0	0	0	1	0	0
Pennsylvania.....	0	1	1	0	348	351	0	0	0	5	6	6
<b>E. NO. CEN.</b>												
Ohio.....	1	0	0	318	339	376	2	0	1	0	3	2
Indiana.....	0	5	0	126	125	188	12	2	7	2	3	1
Illinois.....	0	1	1	201	252	489	0	0	1	1	1	2
Michigan <sup>1</sup> .....	0	1	0	100	207	317	1	1	2	2	1	1
Wisconsin.....	0	0	0	264	214	214	1	0	2	1	2	0
<b>W. NO. CEN.</b>												
Minnesota.....	1	0	0	67	93	125	0	1	13	0	1	1
Iowa.....	0	0	0	63	47	71	1	1	11	0	1	2
Missouri.....	0	1	0	110	56	91	0	1	6	0	0	2
North Dakota.....	0	0	0	16	19	21	0	0	0	0	0	0
South Dakota.....	0	0	0	38	32	21	0	0	0	0	0	0
Nebraska.....	0	0	0	23	34	36	0	2	2	0	0	0
Kansas.....	0	0	0	58	90	114	0	1	2	0	0	0
<b>SO. ATL.</b>												
Delaware.....	0	0	0	6	52	14	0	0	0	0	0	0
Maryland <sup>2</sup> .....	0	0	0	81	75	67	0	0	0	1	2	2
Dist. of Columbia.....	0	0	0	29	13	13	0	0	0	1	1	0
Virginia.....	0	0	0	45	50	50	0	0	0	5	5	2
West Virginia.....	0	0	0	27	56	56	0	0	0	0	0	1
North Carolina.....	1	0	1	63	72	68	0	0	0	1	0	0
South Carolina.....	0	2	1	12	6	6	0	0	0	0	1	1
Georgia.....	0	1	0	33	48	18	1	2	0	2	10	3
Florida.....	1	0	1	11	7	7	0	0	0	0	1	1
<b>E. SO. CEN.</b>												
Kentucky.....	2	0	1	50	100	71	0	0	0	0	2	2
Tennessee.....	0	0	0	43	81	54	0	3	1	2	4	3
Alabama.....	1	1	1	16	18	18	2	0	0	0	2	2
Mississippi <sup>2</sup> .....	0	0	0	11	8	7	0	1	1	1	1	1
<b>W. SO. CEN.</b>												
Arkansas.....	1	0	0	7	6	9	2	1	1	2	3	3
Louisiana.....	1	0	1	10	9	16	0	1	0	4	6	4
Oklahoma.....	0	0	0	29	24	43	0	1	1	1	1	3
Texas.....	5	1	1	56	64	66	0	6	6	2	5	5
<b>MOUNTAIN</b>												
Montana.....	0	0	0	9	32	30	0	0	0	0	0	0
Idaho.....	0	0	0	3	3	9	0	0	0	0	1	1
Wyoming.....	0	0	0	53	12	12	0	0	0	0	0	0
Colorado.....	1	0	0	79	43	36	0	0	4	1	0	1
New Mexico.....	0	1	0	7	9	12	1	0	0	0	0	2
Arizona.....	0	0	0	5	7	7	0	0	0	0	0	0
Utah <sup>1</sup> .....	0	0	0	59	38	25	0	0	0	0	0	0
Nevada.....	0	0	0	0	0	0	0	0	0	0	0	0
<b>PACIFIC</b>												
Washington.....	1	1	1	25	29	61	0	0	1	1	1	1
Oregon.....	1	0	0	20	11	46	0	0	0	0	1	0
California.....	9	2	2	191	110	192	1	2	3	2	1	3
<b>Total.....</b>	<b>31</b>	<b>24</b>	<b>26</b>	<b>3,401</b>	<b>3,746</b>	<b>4,528</b>	<b>24</b>	<b>26</b>	<b>55</b>	<b>46</b>	<b>72</b>	<b>79</b>
<b>4 weeks.....</b>	<b>136</b>	<b>109</b>	<b>109</b>	<b>14,150</b>	<b>14,120</b>	<b>16,488</b>	<b>127</b>	<b>67</b>	<b>319</b>	<b>201</b>	<b>315</b>	<b>329</b>

See footnotes at end of table.

Telegraphic morbidity reports from State health officers for the week ended January 30, 1943, and comparison with corresponding week of 1942 and 5-year median—  
Continued

Division and State	Whooping cough			Week ended Jan. 30, 1943								
	Week ended—		Median 1932-42	Anthrax	Dysentery			Encephalitis, infectious	Leptosy	Rocky Mt. spotted fever	Tularemia	Typhus fever
	Jan. 30, 1943	Jan. 31, 1942			Amebic	Bacillary	Un-specified					
<b>NEW ENG.</b>												
Maine.....	81	47	47	0	0	0	0	0	0	0	0	0
New Hampshire.....	18	26	3	0	0	0	0	0	0	0	0	0
Vermont.....	34	57	57	0	0	0	0	0	0	0	0	0
Massachusetts.....	173	304	189	0	0	0	0	0	0	0	0	0
Rhode Island.....	24	51	51	0	0	0	0	0	0	0	0	0
Connecticut.....	71	132	78	0	0	1	0	2	0	0	0	0
<b>MID. ATL.</b>												
New York.....	388	683	462	1	4	19	0	0	2	0	0	0
New Jersey.....	150	235	187	0	1	0	0	0	0	0	0	0
Pennsylvania.....	379	288	349	0	0	0	0	0	0	1	0	0
<b>E. NO. CEN.</b>												
Ohio.....	277	331	265	0	0	0	0	0	0	0	0	0
Indiana.....	22	58	23	0	0	0	0	0	0	0	1	0
Illinois.....	188	213	112	0	0	2	0	2	0	0	0	0
Michigan <sup>1</sup> .....	326	262	229	0	0	1	0	0	0	0	0	0
Wisconsin.....	210	371	198	0	0	0	0	1	0	0	0	0
<b>W. NO. CEN.</b>												
Minnesota.....	74	136	52	0	1	5	0	0	0	0	0	0
Iowa.....	18	35	21	0	0	0	0	0	0	0	0	0
Missouri.....	28	14	23	0	0	0	1	0	0	0	1	0
North Dakota.....	11	15	15	0	0	0	0	0	0	0	0	0
South Dakota.....	0	11	3	0	0	0	0	0	0	0	0	0
Nebraska.....	3	8	3	0	0	0	0	0	0	0	0	0
Kansas.....	43	66	66	0	0	0	0	0	0	0	0	0
<b>SO. ATL.</b>												
Delaware.....	7	2	7	0	0	0	0	0	0	0	0	0
Maryland <sup>1</sup> .....	73	41	44	0	0	0	1	0	0	0	1	0
Dist. of Col.....	10	22	7	0	0	0	0	0	0	0	0	0
Virginia.....	56	77	77	0	0	0	34	0	0	0	0	0
West Virginia.....	61	49	49	0	0	0	0	0	0	0	0	0
North Carolina.....	99	232	232	0	0	0	0	0	0	0	2	1
South Carolina.....	31	100	66	0	0	0	0	0	0	0	1	2
Georgia.....	27	34	27	0	1	1	3	1	0	0	5	18
Florida.....	26	28	11	0	1	0	0	0	0	0	0	2
<b>E. SO. CEN.</b>												
Kentucky.....	50	106	49	0	0	0	0	0	0	0	0	0
Tennessee.....	53	14	22	0	2	0	1	0	0	0	4	0
Alabama.....	41	26	26	0	1	0	0	1	0	0	1	2
Mississippi <sup>2</sup> .....				0	0	0	0	0	0	0	1	0
<b>W. SO. CEN.</b>												
Arkansas.....	35	15	17	0	1	1	0	0	0	0	1	1
Louisiana.....	10	5	5	1	1	0	0	0	1	0	0	0
Oklahoma.....	7	8	8	0	0	0	0	1	0	0	0	1
Texas.....	295	139	136	1	0	116	0	1	0	0	0	15
<b>MOUNTAIN</b>												
Montana.....	45	11	14	0	0	0	0	0	0	0	0	0
Idaho.....	2	6	6	0	0	0	0	0	0	9	0	0
Wyoming.....	4	10	10	0	0	0	0	0	0	0	0	0
Colorado.....	22	27	32	0	0	0	0	0	0	0	0	0
New Mexico.....	24	39	39	0	0	0	1	1	0	0	0	0
Arizona.....	14	83	12	0	0	0	12	0	0	0	0	0
Utah <sup>1</sup> .....	32	37	46	0	0	0	0	0	0	0	0	0
Nevada.....	0	0		0	0	0	0	0	0	0	0	0
<b>PACIFIC</b>												
Washington.....	28	136	96	0	0	0	0	0	0	0	0	0
Oregon.....	10	36	28	0	0	10	0	0	0	0	0	0
California.....	266	202	202	0	0	0	0	0	0	0	0	2
Total.....	3,846	4,828	4,294	3	13	166	53	10	3	0	19	44
4 weeks.....	15,883	17,374	17,010									

<sup>1</sup> New York City only.<sup>2</sup> Period ended earlier than Saturday.

WEEKLY REPORTS FROM CITIES

City reports for week ended January 16, 1943

This table lists the reports from 88 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	Erysipellitis, infectious, cases	Influenza		Measles cases	Meningitis, meningococcus, cases	Pneumonia deaths	Pollomyelitis cases	Scarlet fever cases	Smallpox cases	Typhoid and paratyphoid fever cases	Whooping cough cases
			Cases	Deaths								
Atlanta, Ga.....	1	0	24	0	2	0	6	0	12	0	0	4
Baltimore, Md.....	1	0	7	3	5	5	31	0	28	0	0	81
Barre, Vt.....	0	0	0	0	0	0	0	0	0	0	0	0
Billings, Mont.....	0	0	0	0	0	0	0	0	0	0	0	0
Birmingham, Ala.....	1	0	19	0	0	0	8	1	1	0	0	1
Boise, Idaho.....	0	0	0	0	0	0	2	0	2	0	0	0
Boston, Mass.....	0	0	1	1	92	1	21	0	111	0	0	44
Bridgeport, Conn.....	0	1	0	0	0	0	0	0	6	0	0	1
Brunswick, Ga.....	0	0	0	0	1	0	2	0	0	0	0	0
Buffalo, N. Y.....	0	0	0	0	68	0	11	0	9	0	1	26
Camden, N. J.....	4	0	0	0	12	0	2	0	1	0	0	7
Charleston, S. C.....	0	0	118	0	0	0	3	0	3	0	0	1
Charleston, W. Va.....	0	0	0	0	0	0	0	0	1	0	0	0
Chicago, Ill.....	5	0	7	4	100	1	40	0	77	0	0	70
Cincinnati, Ohio.....	0	0	1	2	21	3	5	0	25	0	0	5
Cleveland, Ohio.....	1	0	11	4	2	0	8	0	31	1	0	64
Columbus, Ohio.....	0	0	0	2	0	0	4	0	20	0	0	11
Concord, N. H.....	0	0	0	1	0	0	0	0	1	0	0	0
Cumberland, Md.....	1	0	0	0	0	0	0	0	0	0	0	0
Dallas, Tex.....	0	0	1	0	0	0	8	1	1	0	0	4
Denver, Colo.....	8	0	26	1	37	0	4	0	8	0	0	10
Detroit, Mich.....	0	0	1	1	13	0	30	0	36	0	0	147
Duluth, Minn.....	0	0	1	0	0	0	2	0	6	0	0	6
Fall River, Mass.....	0	0	1	1	1	0	2	0	7	0	0	23
Fargo, N. Dak.....	0	0	0	0	0	0	0	0	0	0	0	0
Flint, Mich.....	1	0	0	0	0	0	2	0	6	0	0	19
Fort Wayne, Ind.....	1	0	0	0	0	0	2	0	0	0	0	0
Frederick, Md.....	0	0	0	0	0	0	0	0	0	0	0	0
Galveston, Tex.....	0	0	1	0	0	0	1	0	0	0	0	1
Grand Rapids, Mich.....	0	0	0	0	0	0	4	0	2	0	0	2
Great Falls, Mont.....	0	0	0	0	6	0	0	0	1	0	0	15
Hartford, Conn.....	0	0	0	0	6	0	8	0	3	0	0	6
Houston, Tex.....	1	0	0	0	0	0	13	0	2	0	0	1
Indianapolis, Ind.....	9	0	3	3	70	1	10	0	20	0	0	15
Kansas City, Mo.....	0	0	0	0	10	1	14	1	42	0	0	7
Kenosha, Wis.....	0	0	1	0	1	0	0	0	5	0	0	0
Little Rock, Ark.....	0	0	1	0	0	0	4	0	0	0	0	0
Los Angeles, Calif.....	2	0	20	1	34	1	8	1	29	0	0	32
Lynchburg, Va.....	0	0	0	0	1	0	0	0	1	0	0	0
Memphis, Tenn.....	0	0	10	1	7	3	9	0	8	0	0	9
Kenosha, Wis.....	0	0	0	0	128	0	7	0	130	0	0	40
Minneapolis, Minn.....	1	0	0	0	5	0	7	0	17	0	0	12
Missoula, Mont.....	0	0	0	0	0	0	0	0	1	0	0	1
Mobile, Ala.....	1	0	3	2	0	0	1	0	2	0	0	1
Nashville, Tenn.....	0	0	0	0	10	0	1	0	0	0	0	5
Newark, N. J.....	0	0	7	0	42	3	4	0	18	0	0	10
New Haven, Conn.....	0	0	0	0	2	0	1	0	1	0	0	4
New Orleans, La.....	1	0	1	2	4	1	18	0	8	0	0	1
New York, N. Y.....	14	0	22	4	49	15	78	1	225	0	3	93
Omaha, Nebr.....	1	0	0	0	3	0	5	0	2	0	0	1
Philadelphia, Pa.....	3	0	2	2	977	8	36	0	88	0	0	69
Pittsburgh, Pa.....	2	0	2	1	2	1	19	10	14	0	0	25
Portland, Maine.....	0	0	1	1	1	13	5	0	8	0	0	34
Providence, R. I.....	2	0	0	0	4	4	4	0	7	0	0	13
Pueblo, Colo.....	1	0	0	0	1	0	4	0	3	0	0	1
Racine, Wis.....	0	0	0	0	13	0	0	0	20	0	0	0
Reading, Pa.....	0	0	1	1	40	0	4	0	0	0	0	6
Richmond, Va.....	0	0	1	1	1	4	5	0	1	0	0	4

## City reports for week ended January 16, 1943—Continued

	Diphtheria cases	Enecephalitis, in-fectious, 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	1	0	1	0	0	0
Rochester, N. Y. ....	0	0	0	0	20	0	4	1	6	0	0	27
Sacramento, Calif. ....	4	0	1	1	0	0	7	0	2	0	0	7
Saint Joseph, Mo. ....	0	0	0	0	0	1	2	0	1	0	0	0
St. Louis, Mo. ....	0	1	7	1	8	8	14	0	20	0	0	21
St. Paul, Minn. ....	0	0	0	2	2	0	13	0	12	0	0	37
Salt Lake City, Utah. ....	0	0	0	0	148	1	2	0	20	0	0	9
San Antonio, Tex. ....	1	0	2	2	0	0	0	3	2	0	0	3
San Francisco, Calif. ....	0	0	2	1	14	5	15	0	13	0	0	18
Savannah, Ga. ....	0	0	8	1	0	2	7	0	1	0	0	7
Seattle, Wash. ....	2	0	0	0	41	0	10	0	4	0	0	7
Shreveport, La. ....	0	0	0	0	0	0	8	0	3	0	0	0
South Bend, Ind. ....	0	0	0	1	0	0	0	0	4	0	0	2
Spokane, Wash. ....	0	0	1	1	53	0	4	0	0	0	0	1
Springfield, Ill. ....	0	0	0	0	0	0	4	0	1	0	0	22
Springfield, Mass. ....	0	0	0	0	5	0	1	0	76	0	0	1
Superior, Wis. ....	0	0	0	0	2	0	3	0	2	0	0	6
Syracuse, N. Y. ....	0	0	0	0	6	2	2	0	9	0	0	32
Tacoma, Wash. ....	0	0	0	0	62	0	1	1	4	0	0	1
Tampa, Fla. ....	0	0	0	1	1	0	1	0	0	0	0	1
Terre Haute, Ind. ....	0	0	0	1	0	0	1	0	0	0	0	0
Topeka, Kans. ....	0	0	0	0	16	0	2	0	3	0	0	3
Trenton, N. J. ....	2	0	0	0	1	0	4	0	7	0	0	4
Washington, D. C. ....	2	0	4	3	13	4	8	0	25	0	2	13
Wheeling, W. Va. ....	0	0	0	0	1	0	3	0	1	0	0	3
Wichita, Kans. ....	1	0	0	0	7	0	2	0	5	0	0	6
Wilmington, Del. ....	0	0	0	0	2	0	7	0	1	0	0	1
Wilmington, N. C. ....	1	0	0	0	3	0	3	0	0	0	0	12
Winston-Salem, N. C. ....	0	0	0	0	2	0	3	0	0	0	0	22
Worcester, Mass. ....	0	0	0	0	21	0	13	0	6	0	0	16
Total.....	75	2	309	51	2,203	89	615	7	1,279	1	6	1,214
Corresponding week 1942.	100	4	290	44	1,132	26	507	6	1,125	1	12	1,237
Average, 1933-42.....	118	.....	1,804	185	2,659	.....	1,375	.....	1,212	21	15	1,116

<sup>1</sup> 3-year average, 1940-42.

<sup>2</sup> 5-year median.

*Dysentery, amebic.*—Cases: New York, 1; San Francisco, 1.

*Dysentery, bacillary.*—Cases: Buffalo, 1; Charleston, S. C., 1; Hartford, 1; Los Angeles, 5; New York, 10.

*Typhus fever.*—Cases: Atlanta, 4; Birmingham, 1; Houston, 1; Mobile, 1; New Orleans, 1; Savannah, 1.

### PLAGUE INFECTION IN TACOMA, WASH.

Plague infection has been reported proved in two pools of tissue from rats, *R. norvegicus*, (four rats in each pool), taken in Tacoma, Wash., during the periods January 6 to 8 and January 8 and 9, and in tissue from one rat, proved separately, taken on January 9.

### TERRITORIES AND POSSESSIONS

#### Hawaii Territory

*Plague (rodent).*—During the week ended January 9, 1943, one rat proved positive for plague was reported in Paauhau area, Hamakua District, Island of Hawaii, T. H.

## FOREIGN REPORTS

### CUBA

*Habana—Communicable diseases—4 weeks ended December 12, 1942.*—During the 4 weeks ended December 12, 1942, certain communicable diseases were reported in Habana, Cuba, as follows:

Disease	Cases	Deaths	Disease	Cases	Deaths
Diphtheria.....	18	1	Tuberculosis.....	6	2
Malaria.....	33	1	Typhoid fever.....	24	1
Poliomyelitis.....	4	1			

### SWEDEN

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

Disease	Cases	Disease	Cases
Cerebrospinal meningitis.....	9	Poliomyelitis.....	88
Diphtheria.....	297	Scarlet fever.....	2,538
Dysentery.....	95	Syphilis.....	43
Epidemic encephalitis.....	4	Typhoid fever.....	23
Gonorrhoea.....	1,328	Undulant fever.....	3
Paratyphoid fever.....	5	Weil's disease.....	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.

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

#### Plague

*Belgian Congo—Blukwa.*—During the week ended December 26, 1942, 1 fatal case of plague was reported in Blukwa, Belgian Congo.

#### Typhus Fever

*Mexico—Mexico, D. F.*—Typhus fever has been reported in Mexico, D. F., as follows: For the 5 weeks ended October 31, 1942, 72 cases with 18 deaths. Weeks ended November 7, 30 cases, 3 deaths; November 14, 24 cases, 4 deaths; November 21, 24 cases, 3 deaths; November 28, 18 cases, 4 deaths.

## COURT DECISIONS ON PUBLIC HEALTH

*Process or renovated butter—Federal regulation of—effect on State action.*—(United States Supreme Court; *Cloverleaf Butter Co. v. Patterson, Commissioner of Agriculture and Industries of Alabama, et al.*, 315 U. S. 148; decided February 2, 1942.) The plaintiff company was engaged in the manufacture at Birmingham, Ala., of process or renovated butter from packing stock butter. One-fourth of the company's packing stock butter was obtained in Alabama and three-fourths in other States. The company shipped interstate 90 percent of its finished product. The production of renovated butter was taxed and regulated by the United States and was also regulated by Alabama. The defendant Alabama officials, who had the duty of enforcing the Alabama laws regarding renovated butter, entered petitioner's factory and, in a little more than a year, seized on 16 separate occasions a total of over 20,000 pounds of packing stock butter, the material from which the finished product was made. The defendants also seized some butter moving to the factory in interstate commerce. The company sought to enjoin the defendants from acting under the State statute, either to determine the wholesomeness of renovated butter made from the raw material in the company's hands, to inspect its raw material and plant, or to seize and to detain the company's packing stock butter. The theory of the bill of complaint was that the Federal legislation and regulations concerning the manufacture of process or renovated butter excluded such State action. The Federal district court and circuit court of appeals ruled against the company and the case was carried to the United States Supreme Court.

The latter court in its opinion said: "The controversy comes to this: The Federal law requires \* \* \* 'a rigid sanitary inspection \* \* \* of all factories and storehouses where process or renovated butter is manufactured, packed, or prepared for market, and of the products thereof and materials going into the manufacture of the same,' i. e., packing stock butter. But, as we have seen, the Secretary of Agriculture of the United States cannot condemn the packing stock butter. The Commissioner of Agriculture and Industries of Alabama claims authority under the State statute to condemn packing stock butter held for renovation. Does the State's claim interfere or conflict with the Federal power?" The court determined that the State's claim did interfere or conflict with the purpose or provisions of the Federal legislation. It was pointed out that the manufacture and distribution in interstate and foreign commerce of process and renovated butter constituted a substantial industry

which, because of its multi-State activity, could not be effectively regulated by isolated competing States and that Congress undertook to regulate production in order that the resulting commodity might be free of ingredients deleterious to health. The States were left free to act on the packing stock supplies prior to their delivery into the hands of the manufacturer and to regulate sales of the finished product within their borders. However, once the material was definitely marked for commerce by acquisition of the manufacturer, it passed into the domain of Federal control. Inspection of the factory and of the material was provided for explicitly and confiscation of the finished product was authorized upon a finding of its unsuitability for food through the use of unhealthful or unwholesome materials. By the statutes and regulations, continued the court, the Federal Department of Agriculture had authority to watch the consumer's interest throughout the process of manufacture and distribution. "It sees to the sanitation of the factories in such minutiae as the clean hands of the employees and the elimination of objectionable odors, inspects the materials used, including air for aerating the oils, and confiscates the finished product when materials which would be unwholesome if utilized are present after manufacture. Confiscation by the State of material in production nullifies Federal discretion over ingredients." The court held that, since there was Federal regulation of the materials and composition of the manufactured article, there could not be similar State regulation of the same subject.

The judgment dismissing the bill of complaint was reversed.

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*Amebic dysentery—additional compensation under workmen's compensation act for employer's serious and willful misconduct—violation of health statute by employer.*—(California Supreme Court; *Parkhurst v. Industrial Accident Commission et al.*, 129 P.2d 113; decided September 24, 1942.) An iron worker in the course of his employment on a building project contracted amebic dysentery through the drinking water furnished. The State industrial accident commission awarded him compensation for an injury received in the course of his employment but denied an additional award claimed by reason of the alleged serious and willful misconduct of the employer. In a proceeding by the employee, the issue before the Supreme Court of California was whether the commission's determination that the employee's injury was not caused by the serious and willful misconduct of the employer found support in the record.

It appeared that the employer, who commenced work as a subcontractor after construction was under way, accepted the facilities

furnished by the general contractor. The water was supplied to the men by an open bucket and common dipper, in violation of statutes requiring closed containers and individual drinking cups. According to the supreme court serious and willful misconduct was conduct that the employer knew, or should have known, was likely to cause serious injury, or conduct that evinced a reckless disregard for the safety of others. In the present case, said the court, the employer, by knowingly violating its statutory duty to supply its employees with pure drinking water in closed containers and individual cups, set the conditions for the transmission of various communicable diseases and exposed its employees to the hazard of serious injury therefrom. "It has long been recognized that communicable diseases are readily transmitted by common drinking cups and the statutes in the present case were designed to safeguard employees against that hazard. Violation of these statutes is particularly serious when hundreds of men are employed on the same project at the same time and do not have access to other drinking water. The employer is charged with knowledge of the statute \* \* \* and was found by the commission to know that the water was distributed in violation of the statutory requirements." Violation of the statutes in question was said by the court to be not mere negligence but criminal conduct punishable as a misdemeanor, and a prior California case was quoted wherein it was stated that, where there was a deliberate breach of a law which was framed in the interests of the workingman, it would be held that such a breach amounted to serious misconduct. The court then reviewed the evidence and determined that there was no substantial evidence in the record to support the commission's conclusion that the injury was not caused by the serious and willful misconduct of the employer despite the violation of the statutes.

The order denying additional compensation was annulled.

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*Narcotic drugs—Harrison Act construed.*—(United States Supreme Court; *Young v. United States*, 315 U. S. 257; decided February 2, 1942.) Section 6 of the Harrison Anti-Narcotic Act provided that the act's provisions should not be construed to apply to the manufacture, sale, distribution, giving away, dispensing, or possession of preparations and remedies containing only a limited amount of narcotics, but such section contained a proviso which read as follows: "Provided further, That any manufacturer, producer, compounder, or vendor (including dispensing physicians) of the preparations and remedies mentioned in this section lawfully entitled to manufacture, produce, compound, or vend such preparations and remedies, shall keep a record of all sales, exchanges, or gifts of such preparations and

remedies \* \* \*.” In a case before it involving this proviso, the Supreme Court of the United States expressed itself as being of the view “that Congress, by the use of the words ‘dispensing physicians,’ meant to exclude physicians administering to patients whom they personally attend.”

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*Food containing trichinae—illness caused by—liability of packing company and retailer.*—(New York Supreme Court, Appellate Division, Second Department; *Catalanello et al. v. Cudahy Packing Company et al.*, 34 N.Y.S.(2d)37; decided April 6, 1942.) In an action brought against a packing company and a retailer to recover damages for injuries sustained from eating a processed salami, it appeared that the salami (a) was processed by the defendant packer for consumption without cooking or further processing and so sold by the defendant retailer to one of the plaintiffs and (b) was found by the trial court to contain trichinae sufficient to render the plaintiffs ill. The appellate division of the New York Supreme Court said that the salami would be deemed adulterated so as to be unfit for food within the meaning of a section of the agriculture and markets law and that the violation by the defendants of the duty imposed upon them by certain sections of the said law not to process or sell an article of food which was adulterated constituted an actionable wrong. Since the salami was unfit for food there was also, according to the court, a breach of the implied warranty of the fitness of food for human consumption and, the retailer having been found liable, recovery over by him against the packing company was properly allowed.

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