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PREVALENCE OF POLIOMYELITIS

For the week ended July 29, a total of 177 cases of poliomyelitis was reported in the United States, as compared with 137 cases for the preceding week and with 257 cases for the 1934–38 median. The incidence during the current week was, therefore, about 68 percent of the 5-year median.

The rise in the number of cases reported during the current week is accounted for by an increase of 12 cases in Michigan, which State reported 29 cases as compared with 17 for the preceding week, and by smaller increases in scattered States. Of the cases reported in Michigan, 21 occurred in Detroit.

South Carolina reported 12 cases, the same number as for the preceding week, and the incidence in California dropped from 51 to 46 cases.

In the following article and accompanying table a summary of poliomyelitis incidence, by geographic regions, is given for the 4 weeks ended July 15, and on pages 1456–1457 will be found the reports from States for the week ended July 22.

PREVALENCE OF COMMUNICABLE DISEASES IN THE UNITED STATES

June 18–July 15, 1939

The accompanying table summarizes the prevalence of eight important communicable diseases, based on weekly telegraphic reports from State health departments. The reports from each State are published in the PUBLIC HEALTH REPORTS under the section "Prevalence of disease." The table gives the number of cases of these diseases for the 4-week period ended July 15, 1939, the number reported for the corresponding period in 1938, and the median number for the years 1934–38.

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DISEASES ABOVE MEDIAN PREVALENCE

Influenza.—For the 4 weeks ended July 15, there were 1,599 cases of influenza reported, as compared with 1,384, 1,269, and 2,691 cases for the corresponding period in 1938, 1937, and 1936, respectively. The incidence was about 25 percent above the average incidence for this period in recent years. The South Atlantic and Mountain regions were largely responsible for the current excess incidence. In the South Atlantic region the number of cases (695) was almost three times the average seasonal incidence, and in the Mountain region the incidence was about two and one-half times the 1934–38 average incidence.

Number of reported cases of 8 communicable diseases in the United States during the 4-week period June 18–July 15, 1939, the number for the corresponding period in 1938, and the median number of cases reported for the corresponding period 1934–38 [1]

Division	Current period	1938	5-year median	Current period	1938	5-year median	Current period	1938	5-year median	Current period	1938	5-year median
	Diphtheria			Influenza [2]			Measles [3]			Meningococcus meningitis		
United States [1]	986	1,145	1,249	1,599	1,384	1,269	20,185	32,457	32,457	124	150	296
New England	23	17	59	7	12	5	3,929	2,013	2,772	5	2	7
Middle Atlantic	137	208	289	18	23	22	4,126	10,786	10,052	34	24	72
East North Central	176	235	299	136	108	124	2,029	10,566	10,566	20	25	42
West North Central	63	85	108	108	47	118	950	1,800	1,460	12	11	16
South Atlantic	164	210	180	695	339	237	1,741	3,128	1,694	27	35	53
East South Central	63	78	84	117	91	91	273	547	547	12	38	38
West South Central	196	141	155	299	598	380	1,078	546	546	6	10	18
Mountain	66	77	45	130	71	52	758	1,129	740	2	4	9
Pacific	98	94	111	89	95	133	5,301	1,942	1,959	6	1	21
	Poliomyelitis			Scarlet fever			Smallpox			Typhoid and paratyphoid fever		
United States [1]	390	157	653	4,732	6,366	8,017	381	648	534	1,369	1,706	1,770
New England	5	9	15	426	758	652	6	0	0	33	20	20
Middle Atlantic	18	18	27	1,247	1,708	2,381	0	0	0	73	95	132
East North Central	32	20	24	1,601	1,962	3,037	104	137	98	100	137	139
West North Central	12	13	11	381	510	751	127	228	220	56	57	114
South Atlantic	148	27	27	230	341	330	6	4	4	415	486	486
East South Central	19	41	41	153	127	127	11	17	5	238	334	345
West South Central	43	13	16	124	262	163	33	52	22	348	438	438
Mountain	20	4	4	181	225	225	34	81	81	54	84	62
Pacific	93	12	44	389	473	584	60	129	70	52	55	55

[1] 48 States. Nevada is excluded and the District of Columbia is counted as a State in these reports.
[2] 44 States and New York City.
[3] 47 States. Mississippi is not included.

DISEASES BELOW MEDIAN PREVALENCE

Diphtheria.—The incidence of diphtheria reached a new low level for the current period. The number of cases reported (986) was less than 90 percent of the number reported for this period in 1938, and less than 80 percent of the 1934–38 average incidence. A few more cases than normally occur at this season of the year were reported

from the West South Central and Mountain regions, but in all other regions the incidence was relatively low.

Measles.—The incidence of measles for the country as a whole was relatively low. The number of cases, approximately 20,000, reported for the current period was about 60 percent of the number reported for the corresponding period in 1938, which figure (32,457) also represents the preceding 5-year average incidence for this period. The Pacific region continued to report an unusually large number of cases, and in the West South Central and New England regions the incidence remained considerably above the normal seasonal expectancy, but in other regions the incidence was either about normal or fell considerably below the average of recent years.

Meningococcus meningitis.—The incidence of this disease (124 cases) for the 4 weeks ended July 15 was the lowest reported for this period in the 11 years for which these data are available. Each section of the country shared in the favorable situation that now exists with respect to this disease. The nearest approach to the current low incidence was in 1934, when 134 cases were reported for this period. The current incidence was only about one-half of the 1934–38 median incidence, but there were 3 years during the preceding 5 years in which the incidence of the disease was high, thus establishing a high median level for that period. Since 1936 the number of reported cases has been decreasing.

Poliomyelitis.—During the 4 weeks ended July 15 there were 99 cases of poliomyelitis reported from South Carolina, the same number as that reported during the preceding 4-week period. North Carolina and Georgia, adjoining States, reported 15 and 22 cases, respectively, as against 3 and 10 cases for the preceding 4-week period. An appreciable increase in the number of cases was also reported from California, Texas, and Michigan, but in other States the number of cases reported did not exceed the normal increase of this disease that is expected at this season of the year. The number of cases (390) for the country as a whole was two and one-half times the number reported for this period last year. During 1938 the incidence of poliomyelitis was the lowest in ten years. The average number of cases reported for this period during the years 1934–38 was 653; the current incidence is only about 60 percent of that figure.

Scarlet fever.—The incidence of scarlet fever remained at a comparatively low level. For the current period the reported cases totaled 4,732, as compared with 6,366, 8,017, and 9,638 for the corresponding period in the years 1938, 1937, and 1936, respectively. The East South Central region reported a few more cases than might be expected, but in all other regions the figures represented decreases from last year's figures, as well as very significant decreases from the 1934–38 average figure for this period.

Smallpox.—The number of cases (381) of smallpox reported for the 4 weeks ended July 15 was about 60 percent of the number reported for the corresponding period in 1938, and about 75 percent of the 1934–38 average incidence for this period. Six cases were reported from Connecticut, in the New England region, and the East North Central and South Central regions reported excesses over the average incidence in those regions, but in the West North Central, Mountain, and Pacific regions the incidence was relatively low.

Typhoid fever.—The number of cases of typhoid fever increased about 50 percent during the current period over the preceding 4-week period, but the total number of cases (1,369) was only about 80 percent of the preceding 5-year average incidence for this period. Each section of the country except the New England shared in the favorable situation. In the State of Washington the number of cases dropped from 113 for the 4 weeks ended June 17 to 26 during the current period. An increase in this disease is normally expected at this season of the year and the peak is not usually reached until the latter part of August.

MORTALITY, ALL CAUSES

The average mortality rate from all causes in large cities for the 4 weeks ended July 15, based on data received from the Bureau of the Census, was 10.1 per 1,000 inhabitants (annual basis). The average rate for this period for the years 1934–38 was 10.7. The current rate is the lowest since 1933, when the rate for this period was 9.9.

DERMATITIS CAUSED BY A NEW INSECTICIDE

By Louis Schwartz, *Medical Director*, and Leon H. Warren, *Acting Assistant Surgeon, United States Public Health Service*

A chemical company began experiments about a year ago on the manufacture of a new synthetic insecticide. Although it was known that when the insecticide was applied to the skin of experimental warm-blooded animals it would cause dermatitis, there were no cases of dermatitis among the chemists and others who were experimenting with the material during the 6 or 7 months before it was placed on the market. When the production for commercial use was begun, dermatitis broke out among 9 of 12 workers employed in the manufacturing process, and the company was forced to suspend operations pending the installation of safer manufacturing apparatus.

Dermatitis also occurred among the workers in factories to which the product was sold for the purpose of blending with insecticidal spraying solutions. In these factories most of the cases of dermatitis occurred among the workers employed in filling cans with the insecti-

cidal solution. The Labor Department of the State in which the chemical company manufacturing the new insecticide is located requested the United States Public Health Service to investigate the cause of the dermatitis and make recommendations for the safe manufacture of the insecticide.

Most insecticides are either primary skin irritants or sensitizers, and some are both. Insecticidal sprays basically contain pyrethrum dissolved in a petroleum distillate. Other cheaper poisons, such as para-dichlorbenzene, the thiocyanates, derris extract, isobutyl undecylenamid, and the like, are commonly substituted for a portion of the pyrethrum to reduce the cost of manufacture, since pyrethrum is rather expensive. Such substances as citronella and pine oil are added to give the preparation an agreeable odor. Considering the fact that insecticidal sprays are widely used, and that the labels on them usually state that they are nontoxic to human beings, it is remarkable that more cases of dermatitis have not been reported from their use.

Schwartz (*1*) reported an outbreak of dermatitis caused by pyrethrum among the workers in a plant manufacturing a well-known insecticide. Sulzberger and Weinberg (*2*) reported dermatitis in a user of Black Flag, an insect powder containing pyrethrum. Kesten and Laszlo (*3*) also reported dermatitis caused by pyrethrum, but no cases of dermatitis had been reported to be caused by other ingredients of insecticidal sprays.

Many poisonous chemicals are used for insecticidal purposes. The silicofluorides, the chlorobenzols, and naphthalene are used to kill moths and to make fabrics mothproof. The fluorides are used in powder form to kill roaches. The arsenicals, the organic mercury compounds, lime, copper salts, derris root, and nicotine are used as agricultural parasiticides. The chlorophenols, chromates, zinc chloride, coal tar, and creosote oil are used as wood preservatives and termite repellents. All of these substances, except perhaps nicotine, are primary skin irritants if used in strong concentrations. Nicotine is rapidly absorbed through the skin and is a powerful systemic poison.

The new synthetic insecticide which caused the outbreak of dermatitis herein reported is a product known as alpha naphthyl isothiocyanate ($C_9H_{17}NCS$). It is a white crystalline substance with a melting point of 55.5° C. and a boiling point of 142° C. It is but slightly soluble in water (0.0002) at 20° C., but soluble in kerosene up to 12 percent by weight. It has a slight odor, is colorless and nonstaining. It is loosely called mustard oil but differs from true oil of mustard (C_3H_5NCS) which is allyl isothiocyanate.[1] Alpha naphthyl isothiocyanate alone is not sufficiently poisonous to flies to be satisfac-

[1] Mustard oil has no relation to mustard gas ($C_4H_8Cl_2S$), dichlorethyl sulfide, used in warfare.

tory as an insecticide. However, when it is mixed with pyrethrum it has a satisfactory toxic power. Since alpha naphthyl isothiocyanate is much less expensive than pyrethrum, its substitution for a portion of the latter reduces the cost of insecticide manufacture. It is said that by adding 1 percent of alpha naphthyl isothiocyanate to an insecticide about 60 percent of the usual amount of pyrethrum can be eliminated, and the insecticide will still have the desired toxic effect.

MANUFACTURING PROCESS

Alpha naphthyl isothiocyanate is made by allowing carbon disulfide and ammonia to react with alpha naphthylamine in a completely enclosed kettle. Ferrous sulfate solution is added in order to precipitate the resulting compound and the fluid is run through an open filter press, the solid material being scraped off the filters and spread out on trays to dry. The dermatitis was contracted by the men while working on the open filter press and in handling the trays containing the wet and dry crude compound while carrying them into and out of the drying room. The remainder of the manufacturing process consists in purifying the crude material by dissolving in various solvents, such as carbon tetrachloride and acetone, bleaching with sulfuric acid, neutralizing, and filtering in order to remove impurities. It is then ground into a powder and placed in containers for shipment. The crude product as it comes out of the drying room is about 60 percent pure, while the final commercial product is 95 percent pure.

The workers employed in the manufacture of this insecticide were furnished with clean clothes, rubber gloves, and gas masks, and all the operations were completely enclosed except the first filtering, the drying described above, the pouring of the alpha naphthylamine into the kettles containing the carbon disulfide and the ammonia mixture, and the grinding of the commercial product.

Nine to ten days after new men began working in the factory they developed a papulo-vesicular eruption, usually beginning at the wrists or collar line and spreading to other parts of the body. In two of the workers affected, the eruption was accompanied by chills and fever lasting from 3 to 5 days and necessitating the hospitalization of one of them. In the milder cases the dermatitis consisted of an erythema which lasted from 4 to 7 days and then faded. In the severe cases the eruption lasted several weeks and was followed by desquamation.

During the inspection of the factory it was noted that the men rotated from one operation to another, so that all of them were exposed to the chemicals at the open filter, in the drying room, and at the grinders. At the time that the inspection was made the manufacturing operation had been discontinued pending the installation of

totally enclosed machinery, and the workers who had been affected with dermatitis had all recovered.

The impurities in the crude product as it came from the first filter press were suspected by the makers of the insecticide to be the cause of the dermatitis. These impurities consisted of ammonium naphthyl dithiocarbamate, ferrous sulfide, and free sulfur. In order to determine the exact chemical causing the dermatitis, a series of patch tests was performed on six of the workers who had had dermatitis, and on three who had been exposed to the chemical but did not develop any skin lesions. The latter were to serve as controls. The patches were allowed to remain on for 24 hours.

The first patch consisted of the dried crude product which contains about 60 percent of alpha naphthyl isothiocyanate and about 40 percent of impurities. Patch No. 2 consisted of the commercial product containing 95 percent of alpha naphthyl isothiocyanate; patch No. 3 consisted of a 5-percent solution of the commercial product in white mineral oil; patch No. 4 consisted of a 1-percent solution of the commercial product in white mineral oil; patch No. 5 consisted of a 0.5-percent solution of the commercial product in white mineral oil; and patch No. 6 consisted of a 0.25-percent solution of the commercial product in white mineral oil.

Table 1 shows the results of the patch tests. It will be noted that two of the controls showed no reactions to any of the patches, while one of the controls reacted to patch No. 2. Four of those who had dermatitis reacted to patch No. 1 and all of them reacted to patch No. 2; three reacted to patch No. 3; and two reacted to patches Nos. 4, 5, and 6.

It will also be noted from this table that the reactions obtained are in direct proportion to the concentration of the chemical and to the degree of severity of the dermatitis from which the patients had suffered. Thus, the ones with the most severe dermatitis were the ones who reacted to the weaker dilutions of the chemical, and those with the least severe cases reacted only to the stronger dilutions.

The patients were again seen 72 hours after the first patches were removed in order to observe any late reactions which may have developed.

Table 2 shows the reading of the patch tests made 72 hours after the removal of the patches. At that time all the cases showed reactions to patches 1 and 2. In other words, late reactions to patch No. 1 developed in two of the cases of dermatitis and in all three of the controls, and late reactions to patch No. 2 developed in those controls who showed no reaction after 24 hours. It will also be seen that the reactions had increased in severity. Patches 1 and 2 showed actual ulcerations in many of the cases. One of the workers, V. Y., was not seen at the time of the second examination, but the attending physician

stated that he had developed reactions under all the patches, whereas at the 24-hour reading he had reactions only under patches 1 and 2.

TABLE 1.—*History and reactions to patch tests after 24 hours*

Patient	Age	History	Patch test					
			1	2	3	4	5	6
			Crude powder (filter press) (60 per-cent)	Kess-cocide powder (com-mer-cial) (95 per-cent)	Dilutions in white mineral oil (per-cent)			
					5	1	0.5	0.25
W. B.	21	Onset of dermatitis on wrists after 10 days' exposure. Became generalized. Chills and fever. Hospitalized 9 days. Duration 1 month. Did not return to former exposure.	++++	++++	+++	++	++	+
P. L.	24	Onset on arms after 10 days' exposure. Became generalized. Had chills and fever. Hypersensitive to poison ivy.	++++	++++	+++	++	+	+
W. W.	22	Onset on wrists after 9 days' exposure. Spread up arms and back. Returned to work after 10 days. No recurrence.	+++	++++	+	−	−	−
V. Y.	26	Onset on wrists after 10 days. Became generalized. Did not return to former exposure.	+++	+++	−	−	−	−
J. H.	29	Onset on arms after 15 days. Duration 2 days. Returned to exposure without recurrence.	−	+++	−	−	−	−
H. B.		Onset on wrists after 9 days' exposure. Healed in 3 days. Did not return to former exposure.	−	++	−	−	−	−
CONTROLS								
W. S.	22	Worked 6 weeks. Immune	−	++	−	−	−	−
L. W.	42	Worked several months. Immune	−	−	−	−	−	−
R. R.	33	Worked 5 months. Immune	−	−	−	−	−	−

The two cases showing reactions at this time to all the patches, and the worker who was not seen, V. Y., had previously had generalized cases of severe dermatitis.

TABLE 2.—*Reactions to patch tests after 96 hours*

Patient	Patch test					
	1	2	3	4	5	6
W. B.	++++	++++	++++	+++	+++	++
P. L.	++++	++++	+++	+	+	+
W. W.	++++	++++	+++	−	−	−
V. Y.[1]						
J. H.	++	+++	−	−	−	−
H. B.	++	+++	−	−	−	−
CONTROLS						
W. S.	+++	+++	−	−	−	−
L. W.	+++	++++	−	−	−	−
R. R.	+++	++++	−	−	−	−

[1] Was not seen but reported that there were reactions under all patches.

The results of the patch tests show that those individuals who had had the most severe cases of dermatitis gave the strongest reactions to the patch tests. It is also seen that the controls reacted strongly to patches 1 and 2, but did not react to the weaker dilutions which produced reactions in the workers who had previously had severe cases of dermatitis. These facts indicate that the chemical is a primary skin irritant, and that it is also a sensitizer, as shown by the facts that there was a definite period of incubation of about 10 days after the first exposure before the dermatitis developed and that those cases who had become sensitized and who developed dermatitis reacted even to a solution of 0.25 percent of the chemical. Since the controls did not react to a 5 percent dilution in white mineral oil (patch 3), such a patch may be applied safely in order to discover hypersensitivity to the chemical.

An investigation was made of the occurrence of dermatitis in the two insecticide spray manufacturing plants which had purchased alpha naphthyl isothiocyanate to replace some of the pyrethrum used in their product.

In one of these plants, 13 employees out of a total of 58 who had possible contact with the new insecticide spray developed dermatitis.

For a number of years this company had been manufacturing an insecticide containing the insecticidal principles of 14 percent pyrethrum flowers and 1 percent ground derris root (prepared from derris mellicantus, a product imported from the East Indies, the insecticidal principles of which are said to be rotenone and deguelin), in deodorized kerosene. (Deodorized kerosene is made by repeated sulfonation of kerosene until the odor has disappeared.) During this period seven mild cases of dermatitis had occurred among the employees, all of whom had recovered while working and had apparently developed an immunity.

Seven days after this company changed its formula to consist of 7 percent pyrethrum flowers, 1 percent powdered derris, 1½ percent insecticide L.[2], and ¼ percent alpha naphthyl isothiocyanate, dissolved in a base of deodorized kerosene, dermatitis broke out among the workers. Six women of thirty who were engaged in filling cans with the insecticide, and 3 men of 12 who were filling cans were affected. Three men out of ten who were blending the chemicals to make the insecticide spray and one mechanic who came in contact with the insecticide while preparing the vats containing it were also affected. The 13 cases included 7 who had previously contracted mild dermatitis from the old formula containing only the insecticidal principles of pyrethrum flowers and derris root but who had recovered and apparently developed an immunity. None of the 13 persons had severe

[2] Insecticide L. contains about 12½ percent of butyl carbitol thiocyanate, 37½ percent of beta thiocyanoethyl laurate, and 50 percent deodorized kerosene.

enough cases of dermatitis to cause loss of time from work. The mild cases consisted of a simple erythema of the wrists and forearms lasting a few days and the more severe cases had papules and vesicles lasting as long as 6 weeks.

The formula of the insecticide was then changed to contain 7 percent pyrethrum flowers, 1 percent derris, and 2 percent of insecticide L. in deodorized kerosene. All the cases of dermatitis recovered and no new cases have occurred since.

In this factory patch tests were performed on a number of workers who did not develop dermatitis from the new blend of insecticide. The following is an excerpt from the report on these patch tests:

> Although no patch tests were done on any of the employees who developed dermatitis, tests were run on normal individuals with alpha naphthyl isothiocyanate and with insecticide L., as well as with a sample of the finished insecticidal spray containing both substances. Three persons were patch tested with a 10 percent solution of alpha naphthyl isothiocyanate in white mineral oil. In one case a rash was produced after 12 hours, in another case after 30 hours, and in the third case there was no effect.
>
> Two cases were tested with a 10 percent solution of insecticide L. in white mineral oil. In one case there was no reaction in 1 hour and in another case a barely visible area of redness after 30 hours. A sample of the insecticidal spray containing both substances was patch tested on three normal individuals. In one case a slight redness developed after 2 hours, in the second case only after 6 hours, and in the third case there was no reaction after 6 hours.
>
> The commercial alpha naphthyl isothiocyanate powder was placed on three normal individuals and one showed no reaction in 1 hour, but the other two showed marked reactions after 24 hours.
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> Finally, the undiluted insecticide L. was tried on three normal individuals, one of whom showed no reaction after 1 hour, the other showed no reaction after 6 hours, and the third showed a barely visible reaction after 43 hours.
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> On the basis of these findings it was concluded that the dermatitis was due entirely to alpha naphthyl isothiocyanate, and arrangements were made to discontinue the use of the substance. Since then no other cases of dermatitis have developed.

The second company manufacturing an insecticidal spray had no cases of dermatitis while blending a mixture of 7 percent pyrethrum extract and ½ percent of insecticide L. in deodorized kerosene. About 1 week after the formula was changed to a blend consisting of 5 percent pyrethrum extract, 1 percent of insecticide L., and ½ percent of alpha naphthyl isothiocyanate, in deodorized kerosene, dermatitis broke out, affecting 8 employees out of a total of 13. The dermatitis consisted of erythema, papules, and vesicles beginning on the wrists and forearms. Some of the cases were mild and some more severe. In one patient the dermatitis became generalized; he developed a temperature and albuminuria, and was confined to bed. He was away from work for 2 months, but entirely well at the time of the investigation. After this company discontinued the new formula

and returned to the old one, the cases of dermatitis recovered and no new cases have since developed.

A visit was made to the factory manufacturing insecticide L., and it was found that this preparation contained butyl carbitol thiocyanate, beta thiocyanoethyl laurate, and some impurities consisting mainly of dibutoxy diethyl ether in a base of deodorized kerosene.

Butyl carbitol thiocyanate is made by allowing butanol to react with dichlor diethyl ether and sodium hydroxide, forming carbitol chloride, which is added to sodium thiocyanate to form butyl carbitol thiocyanate, sodium chloride, and dibutoxy diethyl ether. The product is then treated with water to remove the salt and other impurities and is passed through activated carbon to remove odors.

Beta thiocyanoethyl laurate is made by treating lauric acid (obtained from coconut oil) with phosphorous trichloride to form lauryl chloride. This is converted into the chlorester of lauric acid, which is then washed and dried and treated with sodium thiocyanate.

The processes are all enclosed except the filtration to remove sludge.

The finished product sold for commercial purposes contains about 50 percent of these insecticides in 50 percent deodorized kerosene. The product also contains some impurities, chiefly dibutoxy diethyl ether. Both the insecticides are liquids and the finished product in deodorized kerosene is a red liquid.

The superintendent of the factory stated that skin tests had been performed with this product and that one out of 30 persons coming in contact with the insecticide showed a skin reaction. He also stated that at one time a chemist who was hypersensitive to the product had worked in the plant, but that there were no cases of dermatitis among the workers employed in the manufacturing process.[3]

DISCUSSION

While this investigation shows that alpha naphthyl isothiocyanate is a primary skin irritant and a sensitizer, it is not recommended that its manufacture be permanently discontinued, because nearly all insecticides are skin irritants or sensitizers, and when workers come in contact with new insecticidal preparations they are likely to develop

[3] Patch tests were performed on 9 volunteer workers who had never had dermatitis, employed in manufacturing insecticide L, with the following constituents of insecticide L:

Patch test No. 1.—50 percent butyl carbitol thiocyanate in deodorized kerosene.

Patch test No. 2.—37½ percent beta thiocyanoethyl laurate plus 12 percent of butyl carbitol thiocyanate in deodorized kerosene.

Patch test No. 3.—5 percent of patch test No. 2 in white mineral oil.

Patch test No. 4.—1 percent of patch test No. 2 in white mineral oil.

Patch test No. 5.—½ percent of patch test No. 2 in white mineral oil.

Patch test No. 6.—¼ percent of patch test No. 2 in white mineral oil.

All 9 patch test subjects reacted to patch test No. 1. One subject reacted to both patch test No. 1 and patch test No. 2. There were no reactions to any of the other patch tests.

It is of interest to note that while undiluted ordinary kerosene, when applied as a patch test for 24 hours will cause a reaction on normal skin, the deodorized kerosene (deodorized by repeated sulfonation) used as a solvent for these insecticides did not cause such a reaction.

dermatitis. It is the general experience that they can be safely manufactured under proper working conditions. However, before this new insecticide is placed on the market for use in insecticidal sprays, actual experiments should be performed as to its effect on the skin of those who may be exposed to its action when it is sprayed, according to directions, in an enclosed room to destroy insects. Such tests can be made by spraying the insecticide containing alpha naphthyl isothiocyanate in a closed room in which a group of persons are exposed to its action for 15 minutes a day for at least 10 days, and then observing them for 10 days more. If one case of dermatitis should develop among 200 persons thus exposed, the product should not be placed on the market.

If, as a result of such an experiment, it is decided to continue the manufacture of alpha naphthyl isothiocyanate, it is recommended that the manufacturing process be totally enclosed. That is, the open filter should be replaced by a closed one and the wet material should be transferred by an enclosed belt to a totally enclosed drying chamber and removed from it in the same manner to a totally enclosed grinder, or some other enclosed method of handling should be devised. The workers should be furnished and compelled to wear protective clothing such as rubber gloves, rubber boots, and aprons, and clean work clothes should be furnished them daily by the management. Adequate shower baths should also be installed and the workers should be compelled to use them before leaving the factory. In addition to this the following directions should be placed on containers of the product before it is sold to the insecticide spray manufacturers:

This chemical is irritating to the skin and toxic; therefore the solid material or its solutions should not be allowed to come in contact with the skin, to be inhaled, or to be otherwise taken into the system. It should be handled in the following manner in its manufacture or when blending it into an insecticide mixture:

Do not inhale the dust, wear a respirator.

Do not expose the eyes, wear goggles.

Do not expose any part of the body to the solid or to the solutions, wear protective garments.

Do not employ individuals who have skin eruptions to handle this chemical.

Do not employ individuals who have any abrasions or burns or chapping of the skin until such injuries have healed.

Keep clean the skin of those who handle the chemical.

Use mechanical means wherever possible for handling the solid chemical or its solutions.

CONCLUSIONS

Experiments on animals with most of the insecticides show that they are toxic and irritating to the skin, and in sufficient concentration are capable of producing death. The petroleum distillate which is usually used as a solvent for the insecticide is also toxic and, if not specially treated, is irritating to the skin. In spite of this, insecticide sprays containing these toxic substances are usually sold to the public

without any warning that their contents are injurious. They are usually sold in cans, the contents of which are to be poured into a spray gun furnished with the can. The directions on the can state that the insecticide is to be sprayed from the spray gun into the air of a closed room or on garments. The only hint as to the toxicity of the product is given by the statement usually found on the can that the sprays are harmless if used according to directions. The emphasis in such a statement is in direct contrast to the emphasis contained on poison labels of other poisons such as iodine, phenol, and the like, which are also harmless if used according to directions. The statement that the insecticides are harmless tends to make customers careless in their use. The directions on the cans should be worded in such a manner as to impress on the consumer the fact that the contents are toxic and irritating to the skin unless they are used according to directions. This would place the emphasis on their toxicity and tend to make people more careful in using them. Indeed, placing poison labels on all insecticides would better safeguard not only the health of the consumer but also the financial interests of the manufacturer by helping to protect him against law suits. The directions for use should state that in spraying these substances protective clothing should be worn, the face and other portions of the skin should be exposed as little as possible to their action, and that the containers should be kept away from articles of food and out of the reach of children.

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STUDIES ON THE STANDARDIZATION OF GAS GANGRENE ANTITOXIN (*SORDELLII*)

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Although *Clostridium sordellii* is not regarded as widely distributed in nature and infections in humans and animals have been infrequently reported, it no doubt has a sufficiently important role in gas gangrene to justify the standardization of its antitoxin.

Clostridium sordellii was first isolated by Sordelli in 1922 (*1*). He recovered it from 2 out of 11 cases of acute edematous wound infections in Buenos Aires (*2, 3, 4*). As this organism was found to possess the putrefactive properties of *Cl. sporogenes* and pathogenic properties similar to *Cl. oedematiens* (*Cl. novyi*), Sordelli named it *Bacillus*

oedematis sporogenes. In 1927 Hall and Scott (*5*) suggested the name *B. sordellii* to replace the trinomial. That same year Meleney, Humphreys, and Carp (*6*, *7*), in New York, isolated this organism from 2 cases of post-operative infections due to contaminated catgut. Believing it to be a new species they named it *Cl. oedematoides*. Later it was shown to be the same as *Cl. sordellii* (*8*, *9*). In 1931, Hall and Gray (*10*) recovered *Cl. sordellii* from a case of septic peritonitis in Denver. It has also been recovered in this country from cases of icterohemoglobinuria in cattle. Only 2 cases have been reported from Europe (*11*).

The similarity and possible identity (*12*, *13*) of *Cl. sordellii* to *Cl. bifermentans*,[1] first isolated by Tissier and Martelly in 1902 (*14*), increases the importance of these organisms in gaseous gangrene. *Cl. bifermentans* is quite commonly found in wound infections but has not been considered of great significance. Weinberg et al. (*11*) have found it to be of greater frequency in wound infections than *Cl. histolyticum*. The *bifermentans* strains isolated from wounds have been described as nonpathogenic, but since a nonpathogenic variant of *Cl. sordellii* has been described (*5*) it would be difficult, if not impossible, to differentiate the two. The importance of the apparently nontoxin-producing strains in mixed wound infections is a question that calls for investigation.

The standardization of *sordellii* antitoxin was conducted in a manner similar to that employed in standardizing the other gas gangrene antitoxins. Dried toxin and antitoxin to be used as standards were prepared. With the purpose of establishing a unit of measurement which may be considered for international adoption, comparative tests were made with the provisional unit of the Argentine Republic (*16*).

PREPARATION OF THE *SORDELLII* TOXIN

A 1-percent Witte peptone meat infusion broth with a pH of 7.6 distributed in 2-liter Erlenmyer flasks was used for cultivating the organism. The culture used was labeled *Cl. sordellii XR* and was received from Hilda Hempl Heller. This strain was chosen after a comparative study of the toxin production of 8 different cultures

[1] In 1936, while making a study of the proteolytic anaerobic bacteria, Clark and Hall (*18*) noted the close similarity of *Clostridium bifermentans* to *Clostridium sordellii*. They found that these two organisms could not be differentiated in morphology, colony formation, biochemical reactions, or serologically, but only by toxin production. They also found that *bifermentans* antisera would neutralize to a certain extent highly toxic *Cl. sordellii* strains. They did not recommend that the two species be grouped together because of difference in pathogenicity. Stewart (*15*), in 1938, confirmed their work and proposed that the two be consolidated under the name of *Cl. bifermentans* which has priority over *Cl. sordellii*. Since then the question has arisen (*15*) as to the possible identity of *Cl. sordellii* with Koch's "*oedematis maligni*," thus increasing the complexity of the relationship of the proteolytic anaerobes. For this reason the name *Cl. sordellii* is retained awaiting further study of the problem.

labeled *Cl. sordellii*. These varied in toxicity from a nontoxin-producing strain to this highly pathogenic strain. Each flask of broth was inoculated with a 24-hour meat culture, then incubated at 37.5° C. for 20 hours. The growth was filtered through sterile filter paper pulp, then through Mandler filters. The filtered toxin had a minimal lethal dose of 0.0001 cc. for a 17- to 20-gram mouse when inoculated intravenously. The toxin was precipitated with ammonium sulfate, transferred to a Buchner funnel containing filter paper to remove as much of the fluid as possible, and then dried over phosphorus pentoxide. The yield of toxin from 60 liters of filtrate was 446 grams.

The minimum lethal dose of the dried toxin was found to be from 0.001 mg. to 0.002 mg. when inoculated intravenously into a 17- to 20-gram mouse.

DETERMINATION OF THE "TEST DOSE" OF TOXIN

The test dose of this toxin was determined by testing against a glycerinated antitoxin received from Dr. Alfredo Sordelli of the Argentine Republic. This antitoxin contained 5 units per cc. as determined by subcutaneous inoculations in guinea pigs (*16*).

In determining the "test dose" of the toxin the glycerinated antitoxin received from the Argentine Republic was diluted with 0.85 percent saline so that 1 cc. contained 1 unit. The toxin was diluted so that 1 cc. contained 2 mg. of toxin. The toxin was used in amounts varying from 0.11 cc. (0.22 mg.) to 0.22 cc. (0.44 mg.) with 0.2 cc. of the antitoxin dilution or 0.2 unit plus sufficient saline to equal 0.5 cc. The mixtures were kept at room temperature for one hour, following which they were injected intravenously in 17- to 20-gram mice. The animals were kept under observation for 4 days.

Results of the "test dose" determination of the United States toxin with the Argentine Republic antitoxin are given in table 1.

TABLE 1.—*Preliminary test for determination of the "test dose" of toxin; antitoxin constant, toxin varied*

Toxin (mg.)	Antitoxin	Number of mice used	Mice surviving	
			Number	Proportion
0.22	0.2	3	3	3/3
0.26	0.2	3	3	3/3
0.30	0.2	3	3	3/3
0.34	0.2	3	3	3/3
0.38	0.2	3	1	1/3
0.42	0.2	3	0	0/3
0.44	0.2	3	0	0/3

The results indicated that the "test dose" was approximately 0.38 mg. when tested against 0.2 unit of antitoxin. The test was repeated using six mice to the dose. The results are given in table 2.

TABLE 2.—*Determination of the "test dose" of toxin; antitoxin constant, toxin varied*

Toxin (mg.)	Antitoxin	Number of mice used	Mice surviving	
			Number	Proportion
0.36	0.2	6	6	6/6
0.38	0.2	6	2	2/6
0.40	0.2	6	0	0/6

The "test dose" of the toxin (0.38 mg.) was then tested against varying amounts of the Argentine antitoxin, 10 percent above and 10 percent below the 0.2 unit. The following results were obtained, confirming 0.38 mg. as the "test dose" of toxin.

TABLE 3.—*Determination of the "test dose" of toxin; toxin constant, antitoxin varied*

Antitoxin (units)	Toxin	Number of mice used	Mice surviving	
			Number	Proportion
	Mg.			
0.18	0.38	6	0	0/6
0.2	0.38	6	4	4/6
0.22	0.38	6	6	6/6

TESTS ON THE UNITED STATES STANDARD ANTITOXIN

The *sordellii* serum was obtained from a commercial manufacturing firm. It was measured accurately in 10 cc. amounts into chemically free resistance glass ampules. These were thoroughly dried over phosphorus pentoxide.

The weights of the dried residue contained in 10 ampules were determined and the mean weight found to be 0.9669 g.

The dried residue of one of the ampules was dissolved in 10 cc. of 0.85 percent salt solution and then made up to 100 cc. with 66 percent glycerine. From this dilutions were made up to 1/2000 for preliminary tests.

TABLE 4.—*Assay of the United States standard antitoxin against 0.38 mg. of toxin. Preliminary test*

Dilution of antitoxin	Amount of dilution	Number of mice used	Mice surviving	
			Number	Proportion
	(cc.)			
1/100	0.2	3	3	3/3
1/500	0.2	3	3	3/3
1/1000	0.2	3	3	3/3
1/1500	0.2	3	0	0/3
1/2000	0.2	3	0	0/3

Dilutions were then made between 1/1000 and 1/1500.

TABLE 5.—*Assay of the United States standard antitoxin against 0.38 mg. of toxin. Second test*

Dilution of antitoxin	Amount of dilution	Number of mice used	Mice surviving	
			Number	Proportion
	(cc.)			
1/1100	0.2	3	3	3/3
1/1200	0.2	3	3	3/3
1/1300	0.2	3	3	3/3
1/1400	0.2	3	3	3/3
1/1500	0.2	3	0	0/3

From the results obtained it was assumed that 0.2 cc. of a 1/1450 dilution of the United States standard antitoxin was equivalent to 0.2 unit. Varying amounts of a 1/1450 dilution were then tested against the "test dose" of toxin, or 0.38 mg.

TABLE 6.—*Assay of the United States standard antitoxin against 0.38 mg. of toxin. Third test*

Antitoxin dilution	Amount of antitoxin	Number of mice used	Mice surviving	
			Number	Proportion
	(cc.)			
1/1450	0.18	6	0	0/6
1/1450	0.2	6	3	3/6
1/1450	0.22	6	6	6/6

The 1/1450 dilution of the United States standard antitoxin was then tested against varying amounts of the toxin using approximately 10 percent above and 10 percent below the determined "test dose" of 0.38 mg.

TABLE 7.—*Assay of the United States standard antitoxin against varying amounts of toxin; antitoxin constant*

Amount of 1/1450 antitoxin dilution	Units of antitoxin	Toxin	Number of mice used	Mice surviving	
				Number	Proportion
Cc.		*Mg.*			
0.2	0.2	0.34	6	5	6/6
0.2	0.2	0.38	6	3	3/6
0.2	0.2	0.42	6	0	0/6

The standard antitoxin was diluted so that 1 cc. of a 1/72.5 dilution of the glycerinated antitoxin contained 20 units; this, when further diluted 1/20 with saline, will contain 1 unit, since 1 cc. of the undiluted antitoxin contains 1,450 units (1/72.5×1/20 equals 1/1,450).

On the basis of the mean weight of the dried residue of 10 cc. of the standard antitoxin (0.9669 g.) this amount contains 14,500 units and one unit is contained in 0.06668 mg. of standard antitoxin.

POTENCY OF COMMERCIAL ANTITOXINS TESTED

Several commercial antitoxins were available for testing. These were tested against the "test dose" with the following results:

1. 800 units per cubic centimeter.
2. 800 units per cubic centimeter.
3. 1,700 units per cubic centimeter.
4. 1,450 units per cubic centimeter.

THE PROVISIONAL UNIT

On the basis of the tests performed, the unit proposed by Sordelli may be accepted as the provisional unit for the present pending international acceptance of this unit or a multiple of it.

DETERIORATION OF *SORDELLII* TOXIN

Tests were made to determine the effect of light and temperature on the toxin. Specimens of the dried toxin were placed in dry, stoppered bottles and exposed to the following conditions:

Ten degrees C. in vacuum jar for 12 months; sunlight outside window for 102 days; room temperature in the dark for 102 days; warm room (37.5° C.) in the dark for 102 days. Toxin was also exposed to the air in a desiccator at 10° C. for 12 months. The specimens were then tested for deterioration by determining the "test dose" and the minimum lethal dose of each. The following results were obtained:

	Minimum lethal dose (mg.)	*Test dose (mg.)*
10° C. in vacuum jar	0. 001–0. 002	0. 38
Sunlight outside window	. 004	. 38
Room temperature in the dark	. 002	. 38
Warm room (37.5° C.)	. 008	. 76
Exposed to air in desiccator at 10° C	. 004– . 006	. 60

The results indicate that dry *sordellii* toxin is relatively stable at a low temperature in the absence of oxygen, but unstable at high temperatures and when exposed to oxygen.

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THE INFLUENCE OF DIET ON THE CHRONIC TOXICITY OF SELENIUM

By M. I. Smith, *Chief Pharmacologist, Division of Pharmacology, National Institute of Health, United States Public Health Service*

Throughout the whole course of investigations in this laboratory on the chronic toxicity of selenium there has been one characteristic that has stood out prominently, namely, the great variation in individual as well as in species susceptibility. Thus, when rats received in their diet 10 to 15 parts per million of selenium as sodium selenite or selenate, some of the animals would die relatively early with more or less extensive pathological changes in the tissues and organs, while a considerable percentage would often survive indefinitely and show scarcely any effects (*1*). More recently, while investigations were being made with naturally occurring food selenium in rats, rabbits, and cats, similar variations in individual susceptibility were noted, although in this series of experiments differences in species susceptibility were most pronounced (*2*). Under the conditions of the above experiments, the toxicity and pathologic effects of this type of selenium appeared to be most uniform and most pronounced in rats and least pronounced in cats, though the earlier work (*1*) had indicated a much higher susceptibility to selenium in the rabbit and cat as compared with the rat. Analysis of the various factors involved in the discrepancies made it appear probable that dietary factors might in some way determine the toxicity of selenium.

In an attempt to answer this question, the present experiments were all made upon the Wistar strain of albino rats of inbred stock

maintained on a diet of Purina dog chow. The animals were placed on the experimental rations at the age of about 30 to 35 days when they usually weighed from 50 to 60 grams. Animals of each sex were kept in groups of 5 or 6 in metal cages provided with raised wire-mesh bottoms and clean running tap water. Records were kept of the weights of the animals and their average food consumption, from which the average daily selenium intake per kilogram of body weight was computed.

TABLE 1.—*Composition of experimental diets*

Diet number	25	27	28	29	30
Seleniferous whole wheat flour, 20 p. p. m. selenium, 2.31 percent N	50	50	50	0	0
Control whole wheat flour, no selenium, 2.10 percent N	0	0	0	50	50
Commercial casein	0	0	20	0	0
Dried brewer's yeast, 9.22 percent N	5	5	5	5	5
McCollum's salt mixture No. 185	4	4	4	4	4
Cod-liver oil	2	2	2	2	2
Olive oil	8	0	8	0	8
Beef fat	0	39	0	39	0
Corn starch	31	0	11	0	31

The composition of the experimental diets is shown in table 1. Diets 25, 27, and 28 contained 10 parts per million of naturally occurring food selenium. The seleniferous whole wheat flour which was used to the extent of 50 percent in these diets was from a supply of wheat containing 20 parts per million of selenium, grown in Gregory County, South Dakota. Its nitrogen content was 2.31 percent. The control diets 29 and 30 contained an equivalent amount of a commercial selenium-free whole wheat flour having a nitrogen content of 2.10 percent. The seleniferous diet No. 25 thus consisted, like the control diet No. 30, of about 10 percent protein, 10 percent fat, and about 75 percent carbohydrate. The seleniferous diet No. 27, like the control diet No. 29, consisted of about 10 percent protein, 41 percent fat, and about 43 percent carbohydrate, the last derived chiefly from the wheat flour. The high-protein seleniferous diet No. 28 consisted of about 30 percent protein, 10 percent fat, and about 55 percent carbohydrate. The caloric distribution in the seleniferous diets was about as follows: In diet 25 about 9 percent of the total calories was derived from protein, 20 percent from fat, and over 70 percent from carbohydrate. In the high-protein diet 28 about 28 percent of the food energy was derived from protein, 20 percent from fat, and about 52 percent from carbohydrate. In the high-fat diet 27 only 30 percent of the food energy was derived from carbohydrate, 7 percent from protein, and 63 percent from fat. The caloric values of diets 25, 28, and 30 were about the same, while the caloric value of the high-fat diets 27 and 29 was about 36 percent higher. It might be supposed from this that the food consumption of the animals on

diet 27, and consequently the selenium intake, would be lower than in the animals on diets 25 and 28. Actual check-up of the food and selenium intake per kilogram of body weight showed that it was about the same in the three groups, viz, from about 0.8 to 1.0 mg. of selenium per kilogram per day.

The experiment ran over a period of 4 months. The results are described in the following paragraphs.

Diet 25.—Twenty-two rats, 7 males and 15 females, weighing 50 to 65 grams, were used in this experiment. The first death occurred in 20 days. At the completion of the experiment 17 animals, 77 percent, had died. With the exception of 2 animals dying at an early date, all the animals, including the 5 survivors, had advanced atrophic nodular cirrhosis of the liver. Of 8 animals surviving 90 days, 5 had anemia with hemoglobin levels of 7 to 9 grams and reticulocyte counts of from 5 to 25 percent. Most of the animals dying at an earlier date had severe anemia as shown by blood smears, the precise extent of which was, however, not determined. Ascites, usually straw colored and at times also bloody, varying in amounts from about 2 to 20 cc., was seen in more than 50 percent of the animals dying within the experimental period of 120 days. In a few there was also some effusion in the pleura and pericardium. Loss of hair was also seen in a few of the animals, though this was relatively inconspicuous in this group. Analysis of the livers for selenium in a group of 6 rats at 100 to 120 days gave a range of from 1,430 to 2,860 micrograms per 100 grams wet weight.

The average weight curves of the animals of this group and the mortality rate are shown in chart 1. Figure 1 is a photograph of a typical rat in this group taken 95 days after the beginning of the experiment. The ascites in this animal was very pronounced. The characteristic atrophic and nodular appearance of the livers of the animals of this group is shown in figure 2. The nodular liver, No. 25, was taken from a rat weighing 125 grams, 120 days after the experiment was begun.

Diet 27.—In this group there were 21 rats, 9 males and 12 females, weighing 50 to 68 grams. Growth was decidedly subnormal in the whole group as shown by the average weight curve in chart 1. However, all but 3 survived the experimental period of 120 days, and the general appearance of these animals was healthy. They were active and, except for the extensive loss of hair and stunted growth, they looked quite normal. The most uniform and most pronounced manifestation in this series of animals was a general scantiness of hair with areas of alopecia varying in size and location. The loss of hair was preceded by a peculiar greased appearance of the animals which set in within 2 to 4 weeks on the diet. In several instances the loss of hair was so extensive that the animals were literally hairless. It is

believed that the several deaths in this group were partly due to this condition which resulted in excessive loss of body heat. Hematological studies failed to reveal any marked abnormalities. Blood films were uniformly negative, reticulocyte counts were within the normal range of from 0.5 to 2.0 percent, and the hemoglobin levels varied usually from about 13 to 15 grams. In 3 of the animals the hemoglobin levels were relatively low, 10 to 12 grams, but this is believed to have been due to their general poor nutritive condition rather than to the toxic effects of selenium.

At necropsy there was neither cirrhosis of the liver nor effusions. The spleens were not enlarged and, indeed, none of the organs or vis-

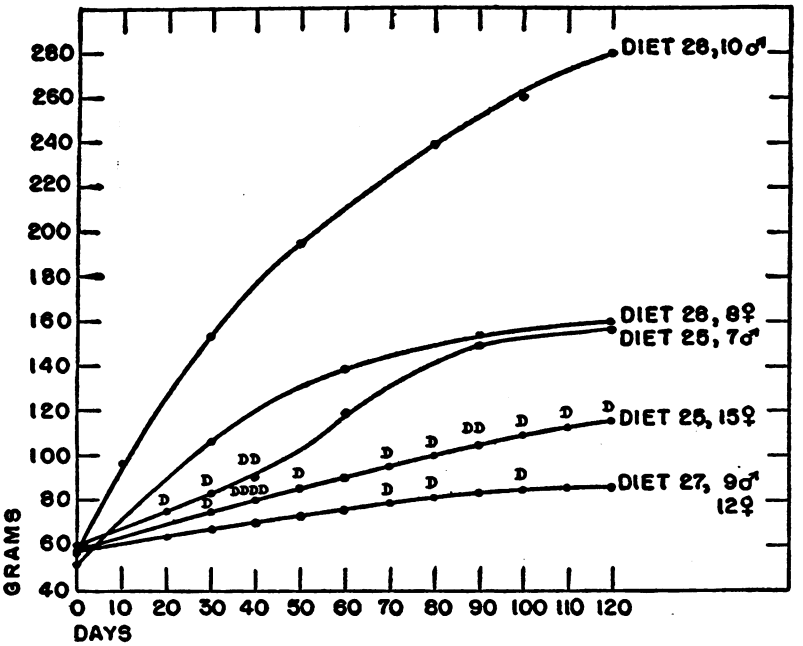

CHART 1.—Average weight curves of rats on low protein-high carbohydrate diet 25, low protein-high fat diet 27, and high protein-low carbohydrate diet 28. *D* indicates death of an animal. All diets contained 10 parts per million of selenium in the form in which it occurs naturally in wheat.

cera showed any gross abnormalities except for their small size in relation to the age of the animal. Microscopic examination of the tissues by Dr. R. D. Lillie of the Division of Pathology disclosed only midzonal fatty degeneration in the liver with many mitotic figures in the polygonal liver cells. This and other features of the pathology of chronic selenium poisoning will be described in detail elsewhere.

Analysis of the livers for selenium in a series of 7 rats of this group at 110 to 120 days revealed a range of from 1,000 to 2,400 micrograms per 100 grams of wet weight.

A typical illustration of the appearance of the animals in this group is shown in figure 3. This photograph was taken 80 days after the

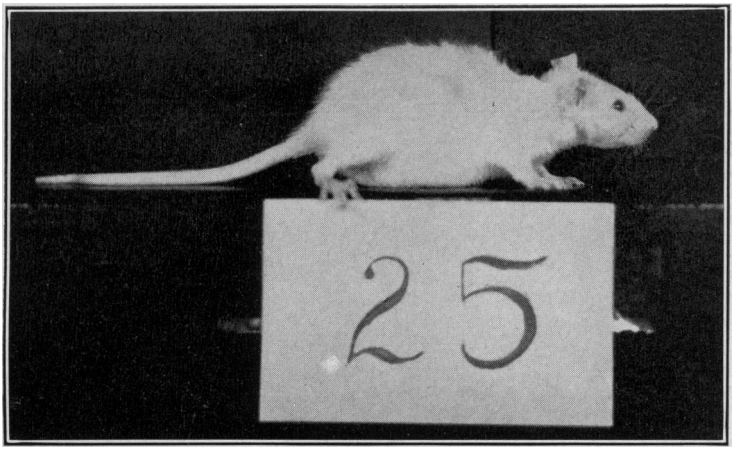

FIGURE 1.—A typical rat receiving 10 parts per million of selenium in a diet of low protein and high carbohydrate content. The animal was photographed 95 days after the beginning of the experiment. It was markedly anemic, had advanced nodular cirrhosis, and the ascites is evident. Weight was 100 grams.

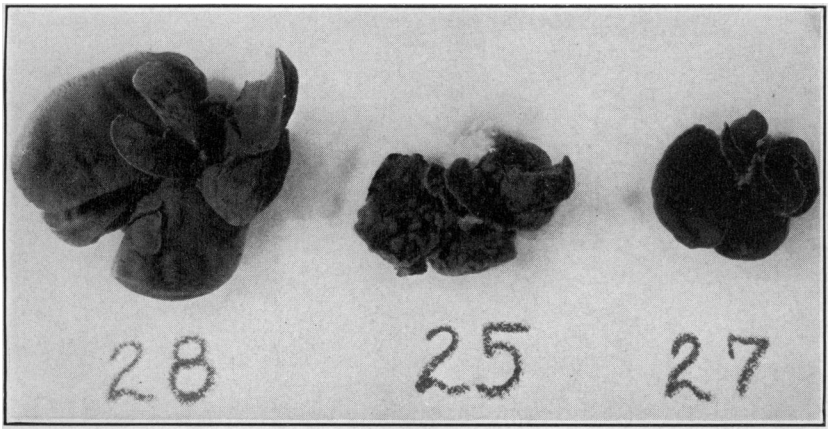

FIGURE 2.—Livers of rats on the 3 experimental diets containing 10 parts per million of selenium as it occurs naturally in wheat. Number 25 is the nodular cirrhotic liver of the rat shown in figure 1, on death of the animal 120 days after the experiment was begun. The rat weighed 125 grams, and had about 25 cc. of clear straw-colored ascitic fluid. Number 27 is the liver of the rat shown in figure 3, which was kept on a low protein-high fat diet. The animal was killed after 120 days on the diet. It weighed 60 grams, and was literally hairless. Number 28 is the liver of a male rat, shown in figure 4, kept on a high protein-low carbohydrate diet. After 60 days on the diet the animal weighed 240 grams, and the liver 9.2 grams. Microscopically, liver 25 showed nodular cirrhosis, liver 27 some midzonal fatty degeneration of the polygonal cells, while liver 28 showed no structural changes.

FIGURE 3.—A typical rat on the seleniferous low protein-high fat diet 27. Note the stunted growth and alopecia. The photograph was taken after 80 days on the diet, when the animal weighed 60 grams.

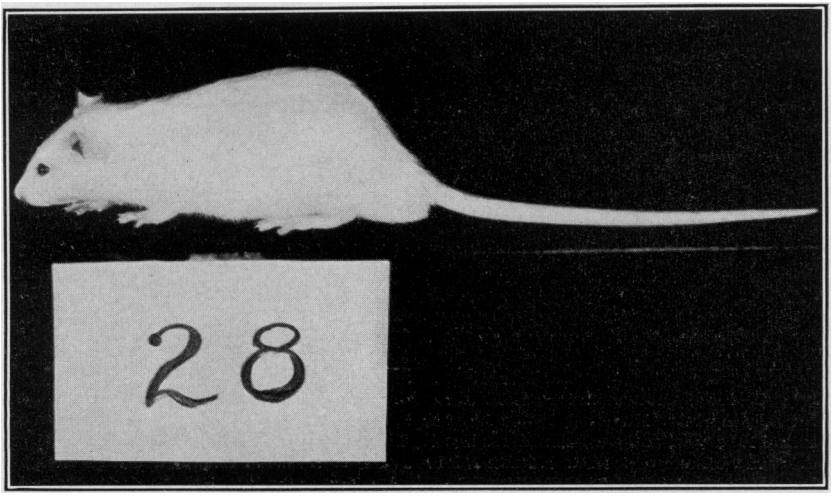

FIGURE 4.—A typical rat on the seleniferous high protein-low carbohydrate diet 28. The photograph was taken 30 days after the beginning of the experiment.

beginning of the experiment. The alopecia, though not as pronounced as in some of the other animals in this group, is sufficiently obvious. The stunted growth of the animal is marked; it weighed only 60 grams. The normal appearance of the liver of this animal at 120 days is shown in figure 2, while the selenium content of this particular liver was 2,140 micrograms percent.

The stunted growth of the animals on diet 27 must be ascribed, in part at least, to the unbalanced nature of the diet rather than to the toxic effects of the selenium. This appears probable for two reasons: First, animals on the control low protein-high fat diet No. 29

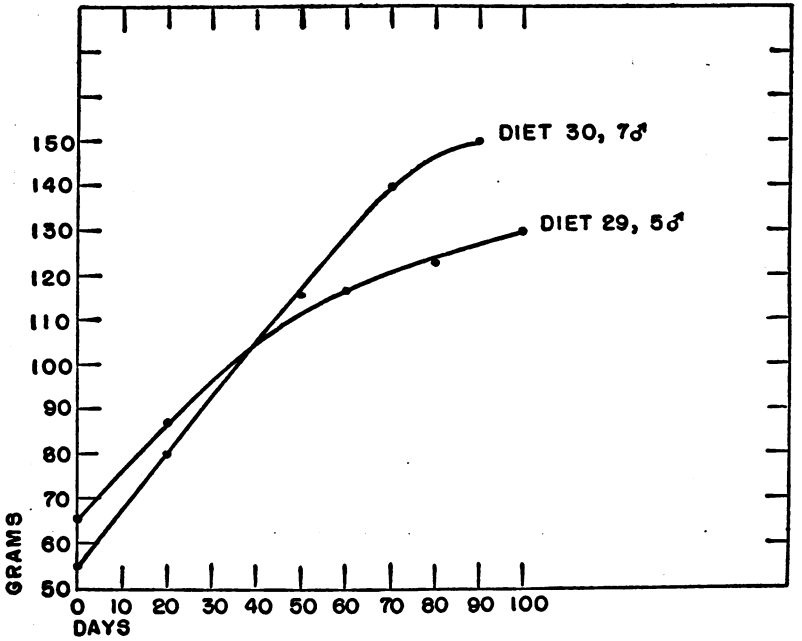

CHART 2.—Average weight curves of rats on control non-seleniferous low protein-high carbohydrate diet 30 and low protein-high fat diet 29.

also showed decidedly subnormal growth, as shown by the average weight curve for 5 males in chart 2. The hemoglobin levels in this control group of animals were also somewhat below normal, the range being from 12.0 to 15.2 grams. It should be added that in appearance these control animals were entirely normal, there being not the slightest suggestion of any disturbance in the skin or appendages, or in any of the internal organs at necropsy.[1] Second, when hairless and stunted animals were taken off diet 27 and placed on a selenium-free semisynthetic adequate diet (No. 242) consisting of 18 percent casein, 5 percent dried brewers' yeast, 4 percent salt mixture No. 185,

[1] Microscopic examination of the livers of these animals showed some midzonal fatty degeneration not unlike that seen with diet 27.

2 percent cod-liver oil, 8 percent olive oil, and 63 percent corn starch, normal growth was promptly resumed, as shown in chart 3. Resumption of hair growth also occurred within 10 days, and by the end of a month the animals appeared entirely normal. Moreover, one of the animals in this group, No. 21 (chart 3), was changed at 120 days to the seleniferous but otherwise adequate high protein diet 28, the composition of which is shown in table 1, and in this animal also normal growth was resumed, and by the end of the experimental period it had a normal covering of hair. The hemoglobin levels of these animals

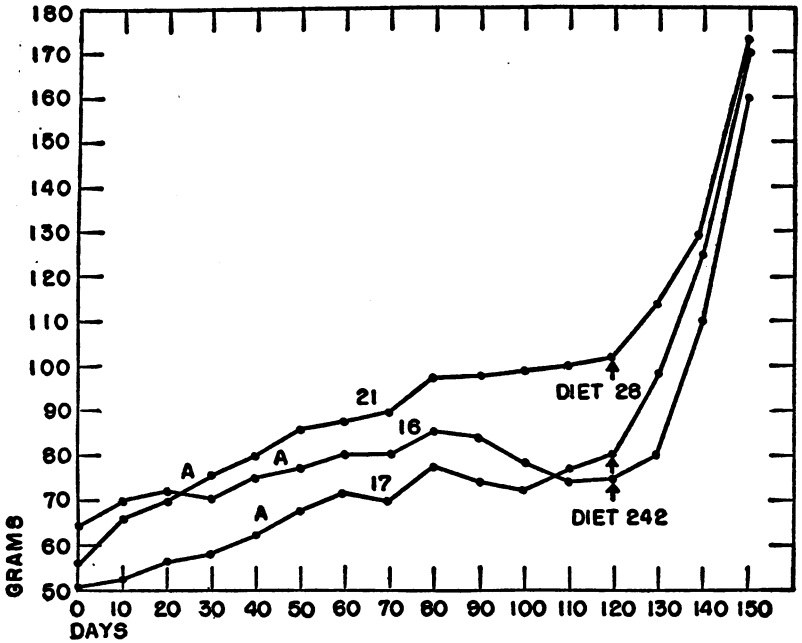

CHART 3.—Temporary and reversible nature of the effects of the seleniferous low protein-high fat diet 27 on stunted growth and alopecia. After 120 days on diet 27 rats 16 and 17 were changed to the balanced non-seleniferous semisynthetic diet 242, and rat 21 was changed to the seleniferous high protein-low carbohydrate diet 28. All rats resumed normal growth, acquired a new coat of hair, and at necropsy showed relatively little or no evidence of structural damage. *A* indicates the onset of alopecia.

rose during this 30-day period from 10.3, 13.2, and 13.1, to 12.2, 14.0, and 15.0 grams, respectively. At necropsy there was no evidence of effusions or any of the tissue damage which is so characteristic of selenium poisoning. The livers of rats 16 and 17 were normal, both in the gross and microscopically, while the liver of rat 21 showed only slight pitting of the surface and microscopically some lymphocyte infiltration of the portal areas, some periportal fibrosis, retraction of the capsule, and some nodular hyperplasia of liver cells.

Diet 28.—Twenty-five rats, 10 males and 15 females, weighing from 50 to 55 grams, were placed on diet 28, which contained 10 parts per million of selenium as it occurs naturally in wheat, and approximately

30 percent protein. Growth was unimpaired in these animals, as shown by the average weight curves given in chart 1 for the 10 males and 8 of the 15 females. Seven of the 15 females were not included in the chart because reproduction was studied in this group, as will be described below. Needless to say, there were no deaths among these animals. The hemoglobin levels for the males ranged from 14.7 to 18.5 grams, and for the females from 14.0 to 18.5 grams. At necropsy at the expiration of the experimental period there was no evidence of any tissue damage in any of the organs of the males, while in 6 of the 15 females there was slight to moderate pitting of the surface of the livers. Microscopically in these livers there was slight to moderate lymphocyte infiltration, slight to moderate proliferation of fibrous tissue in the portal areas, and some retraction of the liver capsule. It is perhaps significant that the livers of the females alone showed some degree of involvement, and relatively more in those that were permitted to reproduce. Ascites or any other abnormalities were not seen in any of the animals.

Figure 4 shows a typical rat in this group, 30 days after the experiment was begun. The animal continued to grow and at 60 days weighed 240 grams. The normal appearance of the liver of this animal, which weighed 9.2 grams, is shown in figure 2.

Analysis of a series of 11 livers from these animals for selenium at the expiration of the experimental period of 120 days showed a range of from 370 to 760 micrograms per 100 grams wet weight. This is decidedly lower than that found in the livers of animals on diets 25 and 27. The meaning of this is not clear at present. On the basis of food consumption, the daily intake per kilogram of body weight was about as high in this as in the other two groups.

Reproduction was studied in a series of 7 females and 3 males in this group. After a period of 2 months on the experimental diet the animals were permitted to mate. Six of the females bore litters of from 3 to 8 within 23 to 27 days. The seventh female bore a litter of 4 in 47 days. Of one litter of 8, 4 died within 24 hours; of another litter of 4, 1 died within 24 hours; and the entire litter of 6 of another animal died within 24 to 48 hours. The latter animal apparently failed to take any interest in her young and was found eating some of them. All the animals, 36 in all, were born alive and appeared entirely normal. The 25 surviving animals were reared normally, while the mothers were continued on the same seleniferous diet No. 28. At the age of 21 to 25 days they weighed 22 to 40 grams each. They were weaned at this time and continued on diet 28. Twelve died in from 2 to 25 days. At 27 days the surviving 13 animals, which had made fairly good growth in the meantime (60 to 90 grams), were killed. All the animals of this group, those that died as well as those that were sacrificed, showed the

typical effects of selenium poisoning, either anemia, ascites, or liver cirrhosis, or all three combined. This only confirms the earlier conclusion that susceptibility to selenium is much greater in very young animals (*2*), although the possibility of an inherited increased susceptibility cannot be excluded. This result is not inconsistent with the other evidence showing that high protein intake affords protection against chronic selenium poisoning. The significant fact is that reproduction was at all possible on a diet of 10 parts per million of selenium, and that the young were born alive and most of them reared normally.

Analysis for selenium of some of the fetuses of the two litters that died within 24 hours revealed a selenium content of 270 and 320 micrograms, respectively, per 100 grams wet weight. This confirms an earlier report from this laboratory on the transmissibility of selenium through the mammalian placenta (*3*), and further demonstrates that the extraordinary susceptibility of the developing chick embryo to selenium, as demonstrated by Franke and his associates (*4*), is not shared by the mammalian fetus.

DISCUSSION

These experiments demonstrate that the chronic ingestion of naturally occurring food selenium may have disastrous effects or may be relatively harmless depending on dietary factors. Ten parts per million of wheat selenium fed in a low protein-high carbohydrate diet caused atrophic nodular cirrhosis, ascites, pleural and pericardial effusions, blood destruction and anemia, and death. Loss of hair on such a diet is in evidence though not especially marked. The same amount of selenium fed in a low protein-high fat diet caused stunted growth and extensive loss of hair, but no effusions, no anemia, and no other structural changes except for some midzonal fatty degeneration of the liver cells. Those effects are not permanent, for when such animals are returned to a normal well-balanced ration body growth and hair growth are promptly resumed and the slight liver damage appears to be repaired. Lastly, the same amount of selenium fed in a high protein-low carbohydrate diet had no demonstrable effects on growth or reproduction, and no structural effects except for a relatively mild degree of chronic interstitial hepatitis in some of the more susceptible individuals. The mechanism for the protective action of these dietary factors against the toxic effects of selenium is not known. It may be assumed for the present that the protective action of the high-fat diet may be due to its protein sparing action. It may be that ultimately the explanation may be found in the differential action of selenium on enzymes concerned with metabolic processes. From the work of Labes and Krebs (*5*), Potter and Elvehjem (*6*), Stotz and Hastings (*7*), C. I. Wright (*8*), and others it is known that selenium

inhibits certain of the respiratory enzymes, more especially those concerned with dehydrogenation of intermediary carbohydrate metabolites. There is less certainty about the effects of selenium on fat and protein metabolism. Cathcart and Orr (*9*), studying the effects of a toxic dose of sodium selenite injected subcutaneously into dogs, found an increased urea and nitrogen output in the urine. In some unpublished experiments carried on by Dr. C. I. Wright in this laboratory it was found that liver arginase in rats was often increased by feeding either inorganic or naturally occurring food selenium. This affords some evidence that at least one enzyme concerned in protein metabolism is not only not inhibited but may be actually enhanced by selenium. It is perhaps significant that the seleniferous diet which was least damaging to the tissues furnished only 30 percent of its total food energy as carbohydrate.

On the assumption that sulfur might be related in some way to the toxicity of selenium, and in view of the fact that methionine is rather low in the wheat protein gliadin and relatively high in casein (*10*) an experiment was carried on to ascertain the effect of added methionine on the toxicity of selenium. A group of rats weighing about 50 grams each were placed on diet 25 into which dl-methionine was incorporated to the extent of 0.8 percent of the diet; that is, the methionine equivalent of 20 percent of casein in the diet was added. This experiment is still in progress, but the results already indicate quite definitely that methionine by itself is not the answer to this problem.

While more work will have to be done to elucidate the present findings, their practical significance is clear. They not only help to explain many of the discrepancies of our own earlier work as well as those of others published in the literature, but they shed much light on the selenium problem as it is related to livestock and human beings in the endemic areas. Disregarding for the present the fat factor, comparison of the protein to selenium ratio in diets 25 and 28 shows a ratio of 1:100 in the former and approximately 1:33 in the latter, if we express this ratio in terms of percent of protein in the diet to micrograms of selenium per 100 grams of diet. Thus, diet 25 with a protein-selenium ratio of only 1:100 is highly toxic, while a protein-selenium ratio of 1:33, as in diet 28, is scarcely, if at all, toxic. In our earlier experiments on cats, which have recently been published (*2*), in which naturally occurring food selenium was fed as a supplement of wheat protein-selenium up to 1.0 mg. per kilo per day in a diet of meat and milk with relatively little untoward effects, analysis in the light of the present results shows that the animals were receiving protein and selenium in their diet in the ratio of about 1:50 on the highest selenium intake of 1 mg. per kilo per day, about 1:30 on the selenium intake of 0.5 mg. per kilo per day, and about 1:10 on the lowest intake of 0.1 mg. selenium per kilo per day. In the experiments on rabbits

(*2*), in which the toxic effects of selenium as it occurs naturally in oats were as a rule more pronounced, simple calculation indicates that in those animals the protein-selenium ratio was only 1:87 in animals receiving oats exclusively and about 1:25 in animals receiving unlimited nonseleniferous cabbage in addition to the seleniferous oats. The seleniferous oats contained 14 parts per million of selenium and 2.6 percent of nitrogen. In the light of the present experiments it is entirely clear, therefore, why the toxic effects of selenium should have been more pronounced in the rabbits than in the cats, even though their daily intake of selenium per kilogram of body weight was less.

More recently seleniferous wheat has been fed in this laboratory to rabbits with daily supplements of 100 grams of non-seleniferous cabbage to provide vitamins and minerals. The effects have been uniformly more severe than those previously noted with seleniferous oats, although the selenium intake in the wheat-fed animals has not exceeded 0.2 to 0.3 mg. per kilo per day. The protein-selenium ratio in these experiments has been close to 1:100.

Similar analysis of results published by other laboratories would, it is believed, explain many of the discrepancies. Moreover, it should be evident that data on the relative toxicity of food selenium from different sources, without adequate regard for the protein and fat content of the diet, as reported heretofore from some laboratories, are quite meaningless.

Application of the results of the present studies to conditions prevailing in the field leads to interesting speculation. It is the experience of farmers in selenium-endemic areas that pigs are usually most readily and most uniformly affected, and that the most conspicuous symptoms are stunted growth and extensive loss of hair. Such animals, we are told, usually make good recoveries if changed to a nutritious selenium-free diet. The analogy with the present experiments on the low protein-high fat diet is perhaps more than superficial, Furthermore, there is much variation in individual and species susceptibility to seleniferous vegetation and grain in livestock, as has been our experience with laboratory animals. Smith, Franke, and Westfall (*11*) reported data on selenium analysis of urines of one colt and three horses in various stages of "alkali" disease and found a concentration varying from 33 to 170 micrograms percent. Similar analysis of urines from 5 cows in various stages of "alkali" disease, obtained by the author in the course of a field investigation, disclosed a variation of from 61 to 104, and, in one instance, 300 micrograms percent. The 5 cows were selected from a herd of about 50 head of cattle as the only ones that had showed some degree of involvement. On the basis of the data reported by Smith, Westfall, and Stohlman (*12*) it may be estimated that these animals were probably not absorbing any more than 0.3 mg. of selenium per kilo per day, and in most cases

probably much less. Evidently some other factor than the selenium as such must be looked for in explanation of the greater susceptibility of these few animals as compared with the rest of the herd.

The results of the present experiments, it is believed, have an important bearing on the question of the selenium health hazard to man and its control. In 1937 it was reported by Smith and Westfall (*13*) that humans in selenium-endemic rural areas are exposed to the more or less continued ingestion of food selenium up to possibly as much as 0.2 mg. per kilo per day. Additional evidence obtained in this laboratory since that time indicates that this is true not only of the farming population living on seleniferous soil, but also to some extent of the population in general, including town dwellers living within selenium-endemic areas. No convincing evidence of serious chronic selenium poisoning from this source in man has ever been reported. A comprehensive study of the probable sources of selenium to which man is exposed showed that locally produced eggs, meats, and milk, next to cereals and vegetables, were of greatest importance (*13*). The highest concentration of selenium found in many specimens of eggs, a few meats, and many samples of milk showed 914, 800, and 127 micrograms percent, respectively. This gives a protein-selenium ratio of about 1:70, 1:50, and 1:40, respectively. Usually the selenium content of such foodstuffs was much lower, and consequently the protein-selenium ratio much more favorable than in the few extreme instances cited. Since naturally occurring selenium in foodstuffs is now well known to be associated with proteins, it would seem that nature has indeed provided her own, if only a partial, solution to this problem. It would also seem that the greatest selenium hazard to man would come from the ingestion of foods with high selenium and low protein content, as may be the case with occasional samples of cereal grains and vegetables. The writer had occasion on one of his field investigations to obtain 9 specimens of milled products, picked at random, in a milling plant located within a selenium-endemic area in one of the Great Plains States. These included various wheat and corn flours. The selenium content of these flours varied from 40 to 175 micrograms percent in 7 of the specimens, 2 containing none. The most unfavorable protein-selenium ratio in such flours might be 1:20, and this is scarcely likely to cause serious harm. The writer was informed, however, that grain from areas known to produce cereals with high selenium content was assiduously avoided. Whether this was done by actual analysis for selenium or by general knowledge of local conditions could not be ascertained.

It would thus appear that as long as those of the population who are heavily exposed to selenium refrain from consuming highly contaminated cereals and vegetables, the selenium hazard is probably

not serious even if small amounts are more or less regularly ingested with food products of animal origin.

CONCLUSIONS

Experiments made on rats show that the toxicity of naturally occurring food selenium is largely determined by dietary factors. A level of intake of selenium which is highly toxic and tissue-damaging when fed in a diet of low protein and high carbohydrate content is only slightly harmful, if at all, when fed in a diet of high protein and low carbohydrate content. The same level of selenium intake in a low protein and high fat diet causes stunted growth and extensive loss of hair, but no other demonstrable tissue damage except some fatty degeneration of the polygonal cells of the liver. Such animals are quickly restored to a normal condition when returned to a balanced non-seleniferous diet.

All the available evidence indicates that the effects and toxicity of naturally occurring food selenium are determined within certain limits not so much by the level of intake as hitherto supposed, as by the protein-selenium ratio in the diet. At a level of 10 parts per million of selenium, a ratio of 1 percent of protein in the diet to about 30 micrograms or less of selenium per 100 grams of diet is of little, if any, toxicity. A ratio of 1 percent protein to 100 micrograms of selenium per 100 grams of diet under the same conditions is dangerously toxic.

Whether the quality as well as the quantity of protein in the diet plays a role in determining the toxicity of selenium is not known at present. This and many other questions will have to be worked out in the future.

ACKNOWLEDGMENTS

I am indebted to Dr. R. D. Lillie for the microscopic examination of the tissues, to Dr. B. B. Westfall for the selenium analyses and to Mr. E. F. Stohlman for the hematological studies.

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COURT DECISION ON PUBLIC HEALTH

Judgment for employer in action brought by employee because of contraction of echinococcosis.—(Nebraska Supreme Court; *Russo* v. *Swift and Co.*, 286 N. W. 291; decided June 2, 1939.) The plaintiff entered the employ of the defendant company as a beef washer in 1925. He continued in such employment until December 1935, when he became totally and permanently disabled as a result of echinococcosis. He brought an action against the employer, charging the latter with negligence. As stated by the court the plaintiff alleged that he "contracted said echinococcosis, not as a result of any negligence on his part, but through the sole and proximate negligence on the part of defendant and its agents, in purchasing cattle which they knew, or, with the exercise of reasonable care, should have known, were infected with the disease; in negligently failing to discover that said cattle were so infected, and negligently failing to destroy and otherwise place said infected cattle in a position where the disease could not spread to this plaintiff; in failing to warn the plaintiff that such cattle were infected; in permitting plaintiff to unknowingly handle cattle which the defendant knew, or should have known, were infected with a dangerous and contagious disease; in failing to provide plaintiff with gloves or suitable apparatus which would protect him from the ravages of a dangerous and contagious disease, and in failing to provide ventilators, disinfectants, or any other proper method of exterminating said disease." The employee fixed the time of contracting the disease as sometime during the year 1933, when, he alleged, he gradually absorbed the disease through the pores and skin of his hands and through his lungs. The defendant company, by demurring, admitted the truth of all such facts as were well pleaded by the plaintiff and all intendments and inferences that could fairly and reasonably be drawn therefrom, but challenged that his petition alleged facts sufficient to constitute a cause of action.

The trial court sustained the demurrer and, on appeal by plaintiff, the supreme court affirmed the judgment of the trial court. The ap-

pellate court, after quoting from medical and other authorities, stated that the plaintiff in his occupation as a beef washer was not suffering from an occupational disease. The court then discussed the duty owed by an employer to an employee and quoted, among other things, the following from an Iowa case: "The master is required to anticipate and foresee or guard against what usually happens, or is likely to happen, but is not required to anticipate or foresee and guard against that which is unusual and not likely to happen. * * * The test is not whether the injurious result or consequence was possible but whether it was probable." In deciding against the employee, the appellate court said:

> Are we to say, in considering the duty of the defendant (employer) toward the plaintiff (employee), that the defendant was guilty of negligence towards the plaintiff in failing to exercise reasonable care, in failing to use the intelligence, ordinarily required in the industry, in furnishing a safe place to work and proper appliances, and in failing to warn the plaintiff? The very nature of the disease of echinococcosis and the manner in which it is contracted are indicative of the fact that an exceptional and unusual standard of care would be necessary on the part of the defendant industry to know or anticipate its existence, and such duty is not required of an employer. We repeat: Under the circumstances as pleaded by the plaintiff, is the disease one which can fairly and reasonably be charged to the knowledge of the defendant? There must be reasonable ground on which to charge the defendant with notice of the existence of the disease. Again referring to the definition of the disease, it is apparent that the term "echinococcus" refers to a parasite, which has its own life cycle, acquired by ingestion and not through the pores and the skin, and in extremely rare instances by inhalation. None of the cases cited by the plaintiff goes to the extent, as contended by him, if [of?] establishing a cause of action under the averments of the second amended petition.

It was also held that the plaintiff's petition, wherein he sought to allege a cause of action under certain health and safety statutes, was defective in failing to allege sufficient facts to show a violation of the said statutes.

DEATHS DURING WEEK ENDED JULY 15, 1939

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

	Week ended July 15, 1939	Corresponding week, 1938
Data from 87 large cities of the United States:		
Total deaths	7,582	[1] 7,731
Average for 3 prior years	[2] 9,526	
Total deaths, first 28 weeks of year	241,830	234,686
Deaths under 1 year of age	466	[1] 515
Average for 3 prior years	[2] 572	
Deaths under 1 year of age, first 28 weeks of year	14,255	14,649
Data from industrial insurance companies:		
Policies in force	67,044,842	69,130,363
Number of death claims	11,529	11,499
Death claims per 1,000 policies in force, annual rate	9.0	8.7
Death claims per 1,000 policies, first 28 weeks of year, annual rate	10.9	9.6

[1] Data for 88 cities.
[2] Data for 85 cities.

PREVALENCE OF DISEASE

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

UNITED STATES

CURRENT WEEKLY STATE REPORTS

These reports are preliminary, and the figures are subject to change when later returns are received by the State health officers.

In these and the following tables, a zero (0) indicates a positive report and has the same significance as any other figure, while leaders (----) represent no report, with the implication that cases or deaths may have occurred but were not reported to the State health officer.

Cases of certain diseases reported by telegraph by State health officers for the week ended July 22, 1939, rates per 100,000 population (annual basis), and comparison with corresponding week of 1938 and 5-year median

Division and State	Diphtheria				Influenza				Measles			
	July 22, 1939, rate	July 22, 1939, cases	July 23, 1938, cases	1934–38, median	July 22, 1939, rate	July 22, 1939, cases	July 23, 1938, cases	1934–38, median	July 22, 1939, rate	July 22, 1939, cases	July 23, 1938, cases	1934–38, median
NEW ENG.												
Maine	6	1	4	1	----	----	5	----	151	25	12	14
New Hampshire	0	0	0	0	----	----	----	----	20	2	4	3
Vermont	0	0	0	0	----	----	----	----	389	29	23	13
Massachusetts	6	5	1	8	----	----	----	----	243	207	167	126
Rhode Island	0	0	0	0	----	----	----	----	221	29	4	16
Connecticut	3	1	2	2	3	1	1	1	145	49	14	41
MID. ATL.												
New York	4	11	10	16	[1] 2	[1] 3	[1] 2	[1] 3	197	491	673	660
New Jersey	1	1	8	8	4	3	2	----	18	15	65	183
Pennsylvania	5	10	13	28	----	----	----	----	31	61	275	553
E. NO. CEN.												
Ohio	10	13	8	8	2	2	----	3	5	7	58	173
Indiana	3	2	10	8	12	8	----	10	12	8	16	20
Illinois	12	19	18	18	1	2	7	7	10	15	58	167
Michigan [2]	5	5	10	11	----	----	----	----	77	73	329	115
Wisconsin	4	2	2	3	4	2	13	14	218	124	384	384
W. NO. CEN.												
Minnesota	0	0	8	5	2	1	1	----	33	17	79	25
Iowa	2	1	1	6	----	----	----	----	130	64	41	18
Missouri	6	5	5	10	----	----	1	11	1	1	8	35
North Dakota	37	5	2	0	----	----	----	----	15	2	28	13
South Dakota	0	0	2	1	----	----	----	----	60	8	----	1
Nebraska	4	1	0	2	----	----	----	----	8	2	12	7
Kansas	6	2	2	3	3	1	3	3	59	21	17	17

See footnotes at end of table.

Cases of certain diseases reported by telegraph by State health officers for the week ended July 22, 1939, rates per 100,000 population (annual basis), and comparison with corresponding week of 1938 and 5-year median—Continued

Division and State	Diphtheria				Influenza				Measles			
	July 22, 1939, rate	July 22, 1939, cases	July 23, 1938, cases	1934–38, median	July 22, 1939, rate	July 22, 1939, cases	July 23, 1938, cases	1934–38, median	July 22, 1939, rate	July 22, 1939, cases	July 23, 1938, cases	1934–38, median
SO. ATL.												
Delaware	0	0	0	0	----	----	----	----	79	4	5	3
Maryland [3]	3	1	5	5	3	1	6	2	31	10	25	33
Dist. of Col	8	1	6	6	----	----	----	----	113	14	7	7
Virginia	19	10	8	8	37	20	----	----	88	47	59	54
West Virginia	24	9	2	3	27	10	9	9	8	3	11	17
North Carolina [3]	16	11	17	11	6	4	----	----	47	32	115	75
South Carolina [3]	8	3	9	3	300	110	68	44	8	3	13	13
Georgia [3]	13	8	18	7	42	25	----	----	10	6	----	----
Florida [3]	12	4	5	5	12	4	----	----	21	7	14	7
E. SO. CEN.												
Kentucky	3	2	7	3	----	----	3	3	7	4	53	53
Tennessee [3]	4	2	5	5	35	20	14	8	5	3	33	33
Alabama [3]	12	7	11	13	12	7	19	7	46	26	5	6
Mississippi [3] [5]	18	7	15	8	----	----	----	----	0	0	----	----
W. SO. CEN.												
Arkansas	7	3	6	3	25	10	12	3	20	8	22	4
Louisiana [3]	17	7	24	11	34	14	6	6	87	36	4	4
Oklahoma	2	1	3	4	8	4	24	13	10	5	9	7
Texas [3]	8	10	23	23	26	31	74	39	45	54	20	55
MOUNTAIN												
Montana	0	0	0	1	28	3	----	----	393	42	33	4
Idaho	0	0	1	0	----	----	2	----	20	2	9	9
Wyoming	0	0	1	0	----	----	----	----	87	4	1	6
Colorado [4]	63	13	4	4	29	6	----	----	43	9	24	24
New Mexico	25	2	0	1	----	----	----	----	12	1	8	8
Arizona	0	0	3	1	159	13	11	6	74	6	38	7
Utah [3]	0	0	0	0	----	----	----	----	179	18	55	23
PACIFIC												
Washington	3	1	1	0	----	----	----	----	644	209	11	36
Oregon	20	4	3	2	30	6	7	8	179	36	8	8
California	19	23	18	23	6	7	23	12	258	315	277	277
Total	8	213	301	310	15	318	313	238	87	2,154	3,126	3,126
29 weeks	15	10,972	13,097	13,995	245	150,548	44,716	103,000	490	344,403	754,176	660,952

Division and State	Meningitis, meningococcus				Poliomyelitis				Scarlet fever			
	July 22, 1939, rate	July 22, 1939, cases	July 23, 1938, cases	1934–38, median	July 22, 1939, rate	July 22, 1939, cases	July 23, 1938, cases	1934–38, median	July 22, 1939, rate	July 22, 1939, cases	July 23, 1938, cases	1934–38, median
NEW ENG.												
Maine	0	0	0	0	0	0	0	0	12	2	5	5
New Hampshire	0	0	0	0	0	0	0	0	10	1	0	1
Vermont	0	0	0	0	0	0	0	0	27	2	3	3
Massachusetts	0	0	0	1	1.2	1	1	5	36	31	61	54
Rhode Island	8	1	0	0	0	0	0	0	23	3	9	8
Connecticut	0	0	1	0	3	1	1	1	42	14	12	7
MID. ATL.												
New York	0.4	1	4	4	2.8	7	2	8	30	75	84	136
New Jersey	2.4	2	1	1	1.2	1	0	1	29	24	10	28
Pennsylvania	3	6	4	2	2.5	5	1	1	48	94	77	114

See footnotes at end of table.

Cases of certain diseases reported by telegraph by State health officers for the week ended July 22, 1939, rates per 100,000 population (annual basis), and comparison with corresponding week of 1938 and 5-year median—Continued

Division and State	Meningitis, meningococcus				Poliomyelitis				Scarlet fever			
	July 22, 1939, rate	July 22, 1939, cases	July 23, 1938, cases	1934–1938, median	July 22, 1939, rate	July 22, 1939, cases	July 23, 1938, cases	1934–1938, median	July 22, 1939, rate	July 22, 1939, cases	July 23, 1938, cases	1934–1938, median
E. NO. CEN.												
Ohio	0	0	0	2	1.5	2	0	2	31	40	39	67
Indiana	1.5	1	1	1	0	0	1	1	39	26	14	22
Illinois	1.3	2	2	8	4	6	1	2	41	63	87	102
Michigan [3]	2.1	2	0	1	18	17	6	2	80	76	81	86
Wisconsin	0	0	0	0	0	0	0	0	53	30	43	63
W. NO. CEN.												
Minnesota	0	0	0	0	1.9	1	0	0	37	19	29	29
Iowa	0	0	0	1	0	0	1	1	18	9	13	19
Missouri	0	0	0	1	2.6	2	0	0	13	10	11	21
North Dakota	0	0	0	0	7	1	0	0	22	3	9	7
South Dakota	0	0	0	0	0	0	2	0	128	17	3	4
Nebraska	0	0	0	0	4	1	1	0	11	3	4	4
Kansas	2.8	1	2	2	0	0	0	0	50	18	21	17
SO. ATL.												
Delaware	0	0	0	0	0	0	0	0	39	2	1	1
Maryland [3]	0	0	2	2	0	0	0	0	31	10	3	12
Dist. of Col.	0	0	0	0	0	0	0	0	0	0	1	3
Virginia	0	0	1	2	1.9	1	2	2	28	15	11	13
West Virginia	0	0	2	2	5	2	0	0	35	13	11	11
North Carolina [3]	1.5	1	1	2	4	3	0	2	29	20	9	10
South Carolina [3]	0	0	2	1	33	12	0	0	0	0	2	2
Georgia [3]	1.7	1	1	1	8	5	2	2	22	13	10	4
Florida [3]	0	0	0	0	9	3	1	0	9	3	9	3
E. SO. CEN.												
Kentucky	1.7	1	3	2	1.7	1	1	2	17	10	13	12
Tennessee [3]	4	2	1	2	1.8	1	2	3	21	12	9	11
Alabama [3]	4	2	3	2	0	0	1	1	5	3	8	8
Mississippi [2 3]	2.5	1	0	0	2.5	1	3	3	0	0	6	7
W. SO. CEN.												
Arkansas	2.5	1	0	0	2.5	1	0	0	0	0	2	2
Louisiana [3]	0	0	3	1	2.4	1	3	3	15	6	5	4
Oklahoma	0	0	0	1	0	0	0	0	2	1	12	11
Texas [3]	0.8	1	2	1	6	7	2	2	12	15	23	31
MOUNTAIN												
Montana	0	0	0	0	0	0	0	0	103	11	4	4
Idaho	0	0	1	1	0	0	0	0	0	0	3	3
Wyoming	0	0	0	0	0	0	0	0	0	0	1	3
Colorado [4]	0	0	3	1	10	2	0	0	82	17	23	9
New Mexico	0	0	0	0	12	1	1	0	86	7	5	6
Arizona	0	0	0	0	0	0	0	1	49	4	3	3
Utah [3]	0	0	0	0	0	0	0	0	99	10	9	9
PACIFIC												
Washington	0	0	3	0	0	0	0	0	31	10	16	11
Oregon	0	0	0	0	0	0	0	1	20	4	12	12
California	0.8	1	2	2	42	51	8	21	56	68	73	73
Total	1.1	27	45	50	5	137	43	227	32	814	899	1,131
29 weeks	1.7	1,259	2,008	3,860	1.6	1,157	668	1,599	156	113,489	133,844	161,216

See footnotes at end of table.

Cases of certain diseases reported by telegraph by State health officers for the week ended July 22, 1939, rates per 100,000 population (annual basis), and comparison with corresponding week of 1938 and 5-year median—Continued

Division and State	Smallpox				Typhoid and paratyphoid fever				Whooping cough		
	July 22, 1939, rate	July 22, 1939, cases	July 23, 1938, cases	1934–38, median	July 22, 1939, rate	July 22, 1939, cases	July 23, 1938, cases	1934–38, median	July 22, 1939, rate	July 22, 1939, cases	July 23, 1938, cases
NEW ENG.											
Maine	0	0	0	0	24	4	2	2	36	6	31
New Hampshire	0	0	0	0	0	0	1	1	51	5	0
Vermont	0	0	0	0	0	0	0	0	402	30	20
Massachusetts	0	0	0	0	2	2	2	4	146	124	89
Rhode Island	0	0	0	0	23	3	1	0	115	15	6
Connecticut	0	0	0	0	0	0	1	1	151	51	73
MID. ATL.											
New York	0	0	0	0	4	10	12	12	142	356	624
New Jersey	0	0	0	0	4	3	13	5	346	291	330
Pennsylvania	0	0	0	0	8	16	17	17	314	618	327
E. NO. CEN.											
Ohio	3	4	0	0	7	9	6	10	79	103	93
Indiana	6	4	10	0	16	11	11	9	281	189	8
Illinois	3	4	10	10	7	11	25	22	238	363	499
Michigan [2]	3	3	1	0	6	6	2	4	284	269	489
Wisconsin	2	1	3	5	2	1	0	1	460	262	335
W. NO. CEN.											
Minnesota	0	0	9	4	2	1	0	1	43	22	76
Iowa	16	8	13	9	8	4	7	2	67	33	15
Missouri	4	3	11	0	28	22	21	21	63	49	33
North Dakota	29	4	4	0	0	0	0	1	51	7	29
South Dakota	23	3	1	1	8	1	0	0	15	2	21
Nebraska	8	2	1	2	0	0	0	0	141	37	21
Kansas	0	0	4	4	6	2	9	10	42	15	115
SO. ATL.											
Delaware	0	0	0	0	20	1	0	1	59	3	6
Maryland [2]	0	0	0	0	19	6	7	11	176	57	40
Dist. of Col	0	0	0	0	24	3	3	2	299	37	13
Virginia	0	0	0	0	30	16	18	28	201	107	76
West Virginia	0	0	6	0	32	12	15	15	70	26	24
North Carolina [3]	0	0	0	1	37	25	27	25	349	239	334
South Carolina [3]	0	0	0	0	82	30	23	23	68	25	104
Georgia [3]	0	0	0	0	33	20	52	52	139	84	52
Florida [3]	0	0	0	0	15	5	5	3	112	37	19
E. SO. CEN.											
Kentucky	0	0	7	0	52	30	41	41	80	46	46
Tennessee [3]	0	0	1	0	49	28	39	39	106	60	48
Alabama [3]	0	0	0	0	11	6	15	16	125	71	49
Mississippi [2 3]	0	0	0	0	23	9	12	17	----	----	----
W. SO. CEN.											
Arkansas	0	0	2	0	64	26	26	26	37	15	15
Louisiana [3]	0	0	0	0	104	43	17	25	63	26	43
Oklahoma	4	2	0	0	48	24	24	27	4	2	19
Texas [3]	1	1	3	1	43	52	63	63	66	80	157
MOUNTAIN											
Montana	0	0	4	4	0	0	0	1	56	6	54
Idaho	0	0	8	2	0	0	1	1	31	3	6
Wyoming	44	2	1	0	44	2	0	0	0	0	7
Colorado [4]	10	2	1	1	24	5	10	3	135	28	48
New Mexico	0	0	1	0	37	3	3	6	235	19	13
Arizona	12	1	1	0	25	2	2	2	209	17	----
Utah [3]	0	0	0	0	0	0	7	1	477	48	50
PACIFIC											
Washington	0	0	10	5	6	2	2	2	68	22	73
Oregon	10	2	2	2	10	2	0	3	114	23	28
California	0	0	15	1	6	7	6	7	109	133	240
Total	2	46	123	85	18	465	548	647	164	4,051	4,798
29 weeks	12	8,500	12,393	5,866	7	5,066	5,816	5,816	158	113,405	125,842

[1] New York City only.
[2] Period ended earlier than Saturday.
[3] Typhus fever, week ended July 22, 1939, 82 cases as follows: North Carolina, 12; South Carolina, 3; Georgia, 35; Florida, 5; Tennessee, 1; Alabama, 13; Mississippi, 1; Louisiana, 4 (delayed report); Texas, 8.
[4] Colorado tick fever, Colorado, 1 case.

ROCKY MOUNTAIN SPOTTED FEVER

Cases reported by States, Feb. 26 to July 29, 1939

	Feb. 26 to Mar. 25	Mar. 26 to Apr. 22	Apr. 23 to May 20	May 21 to June 17	June 18 to July 15	Week ended July 22	Week ended July 29
Eastern:							
New York				3	3		
New Jersey				4	8	1	2
Pennsylvania				6	3	1	
Delaware				3			
Maryland			7	13	11	5	2
District of Columbia			2	2	2	1	1
Virginia			1	13	10	1	4
North Carolina				3	13	5	2
Georgia					1		1
Central:							
Ohio				3	2		2
Indiana				2	1		
Illinois			1	1	5	2	
Tennessee			1		3	3	1
Iowa			1	10	9	2	
Missouri				1			3
Western:							
Montana	[1]2	2	8	5	1		1
Idaho		4	7	4	5		
Wyoming		3	14	16	5	3	
Colorado		2	3	9	4		
Utah		2	5	5	6	2	
Washington		2	3	2			
Oregon		9	16	7	2		

[1] 1 other case was reported in Montana as occurring in February, exact date not given.

SUMMARY OF MONTHLY REPORTS FROM STATES

The following summary of cases reported monthly by States is published weekly and covers only those States from which reports are received during the current week.

State	Meningitis, meningococcus	Diphtheria	Influenza	Malaria	Measles	Pellagra	Poliomyelitis	Scarlet fever	Smallpox	Typhoid and paratyphoid fever
June 1939										
Alabama	8	11	154	662	361	23	3	41	1	27
Arizona	0	13	159	8	48	5	23	44	4	9
California	7	101	92	27	5,695	5	51	454	113	28
Colorado	1	48	33		294		5	107	13	11
Florida	0	20	31	16	238	22	6	28	0	11
Georgia		31	201	247	219	54	10	22	18	72
Hawaii Territory	0	11	4		12		0	0	0	0
Idaho	0	4			159		0	7	1	2
Illinois	8	99	48	18	129	3	6	722	59	35
Indiana	2	19	14	3	33		2	232	49	16
Louisiana	0	29	25	118	130	14	1	25	0	66
Maryland	3	6	10	1	535	5	0	42	0	5
Michigan	2	37	8	4	1,226		6	1,099	26	13
Minnesota	1	14	12	3	613		2	146	18	6
Mississippi	3	25	799	5,397	835	618	1	14	0	22
New York	11	42		18	6,546		8	1,159	30	44
Oklahoma	2	9	67	203	456	31	1	31	77	37
Rhode Island	0	4			464		0	29	0	3
South Dakota	3	1	34		321		0	47	31	0
Tennessee	3	13	70	138	276	19	1	99	104	32

Summary of monthly reports from States—Continued

June 1939	Cases
Actinomycosis:	
California	
Botulism:	
New York	6
Chickenpox:	
Alabama	46
Arizona	8
California	1,737
Colorado	113
Florida	58
Georgia	50
Hawaii Territory	63
Idaho	8
Illinois	1,144
Indiana	133
Louisiana	12
Maryland	143
Michigan	933
Minnesota	307
Mississippi	302
New York	2,367
Oklahoma	35
Rhode Island	53
South Dakota	12
Tennessee	59
Conjunctivitis:	
Georgia	4
Hawaii Territory	44
Oklahoma	5
Dengue:	
Florida	1
Mississippi	4
Diarrhea:	
Maryland	26
Dysentery:	
Arizona (bacillary)	117
California (amoebic)	10
California (bacillary)	60
Colorado	2
Florida (amoebic)	5
Florida (bacillary)	1
Georgia (amoebic)	10
Georgia (bacillary)	163
Illinois (amoebic)	5
Illinois (bacillary)	14
Illinois (amoebic carriers)	24
Louisiana (amoebic)	7
Maryland (unspecified)	6
Maryland (amoebic)	1
Maryland (bacillary)	6
Michigan (amoebic)	4
Michigan (bacillary)	5
Michigan (unspecified)	1
Minnesota (amoebic)	2
Minnesota (bacillary)	6
Mississippi (amoebic)	207
Mississippi (bacillary)	3,006
New York (amoebic)	5
New York (bacillary)	25
Oklahoma (amoebic)	1
Oklahoma (bacillary)	43
Tennessee (amoebic)	3
Tennessee (bacillary)	67
Encephalitis, epidemic or lethargic:	
Alabama	1
California	2
Florida	3
Illinois	1
Indiana	2
Michigan	1
New York	8
Oklahoma	1
Food poisoning:	
California	390
German measles:	
Alabama	3
Arizona	3
California	69
Florida	2
Idaho	15

June 1939—Continued	Cases
German measles—Continued.	
Illinois	24
Maryland	18
Michigan	54
New York	96
Rhode Island	2
Tennessee	4
Glanders:	
South Dakota	1
Granuloma, coccidioidal:	
California	5
Hookworm disease:	
Florida	284
Georgia	1,024
Hawaii Territory	4
Louisiana	86
Mississippi	829
Oklahoma	7
Impetigo contagiosa:	
Hawaii Territory	22
Illinois	1
Maryland	1
Oklahoma	4
Tennessee	8
Jaundice:	
Maryland	1
Minnesota	15
Leprosy:	
Florida	1
Hawaii Territory	1
Mumps:	
Alabama	48
Arizona	29
California	1,824
Colorado	23
Florida	74
Georgia	129
Hawaii Territory	86
Idaho	9
Illinois	463
Indiana	92
Maryland	137
Mississippi	227
Oklahoma	15
Rhode Island	96
South Dakota	8
Tennessee	29
Ophthalmia neonatorum:	
Alabama	1
California	4
Illinois	3
Maryland	1
Minnesota	1
Mississippi	9
New York [1]	7
Tennessee	4
Puerperal septicemia:	
Mississippi	23
Tennessee	1
Rabies in animals:	
Alabama	30
California	118
Illinois	16
Indiana	41
Louisiana	4
Mississippi	11
New York	13
Oklahoma	25
Rhode Island	3
Rabies in man: Michigan	1
Rocky Mountain spotted fever:	
Colorado	5
Georgia	1
Idaho	6
Illinois	1
Indiana	3
Maryland	14
New York	4
Oklahoma	1
Tennessee	2

June 1939—Continued	Cases
Septic sore throat:	
California	4
Colorado	5
Florida	5
Georgia	78
Illinois	3
Indiana	1
Louisiana	2
Maryland	22
Michigan	25
Minnesota	12
New York	163
Oklahoma	77
Rhode Island	16
South Dakota	4
Tennessee	9
Tetanus:	
Alabama	7
California	4
Georgia	2
Hawaii Territory	2
Illinois	3
Louisiana	2
Maryland	1
Michigan	2
New York	4
Oklahoma	4
Tennessee	2
Trachoma:	
Arizona	69
California	16
Georgia	1
Hawaii Territory	1
Illinois	13
Maryland	1
Michigan	1
Mississippi	8
Oklahoma	5
Tennessee	2
Trichinosis:	
California	3
Georgia	1
Hawaii Territory	2
Illinois	2
New York	6
South Dakota	5
Tularaemia:	
California	4
Georgia	6
Illinois	6
Louisiana	2
Minnesota	4
New York	1
Tennessee	3
Typhus fever:	
Alabama	40
Florida	17
Georgia	68
Hawaii Territory	6
Louisiana	2
Mississippi	1
New York	1
Tennessee	5
Undulant fever:	
Alabama	4
Arizona	6
California	19
Colorado	1
Florida	4
Georgia	12
Idaho	3
Illinois	30
Indiana	5
Louisiana	6
Maryland	7
Michigan	11
Minnesota	9
Mississippi	4
New York	17
Oklahoma	77
Rhode Island	1
Tennessee	1

[1] Exclusive of New York City.

Summary of monthly reports from States—Continued

June 1939—Continued

	Cases
Vincent's infection:	
Florida	7
Illinois	10
Maryland	7
Michigan	17
New York [1]	68
Oklahoma	8
Tennessee	7
Whooping cough:	
Alabama	330
Arizona	131
California	645
Colorado	174
Florida	114
Georgia	146
Hawaii Territory	234
Idaho	21
Illinois	1106
Indiana	258
Louisiana	86
Maryland	208
Michigan	895
Minnesota	141
Mississippi	935
New York	1779
Oklahoma	30
Rhode Island	175
South Dakota	11
Tennessee	242

[1] Exclusive of New York City.

WEEKLY REPORTS FROM CITIES

City reports for week ended July 15, 1939

This table summarizes the reports received weekly from a selected list of 140 cities for the purpose of showing a cross section of the current urban incidence of the communicable diseases listed in the table.

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Data for 90 cities:											
5-year average	110	30	13	1,474	326	487	8	372	63	1,380	----
Current week [1]	71	44	13	1,042	254	252	6	333	64	1,494	----
Maine:											
Portland	0	----	0	2	1	2	0	0	0	6	15
New Hampshire:											
Concord	0	----	0	0	0	0	0	0	0	0	10
Manchester	0	----	0	0	0	0	0	0	0	0	26
Nashua	0	----	0	0	0	0	0	0	0	0	8
Vermont:											
Barre	0	----	0	0	0	0	0	1	0	0	4
Burlington	0	----	0	1	0	0	0	0	0	0	3
Rutland	0	----	0	0	0	0	0	0	0	0	4
Massachusetts:											
Boston	0	----	0	34	8	11	0	6	1	26	187
Fall River	1	----	0	2	1	0	0	1	0	3	36
Springfield	0	----	0	9	0	0	0	3	0	0	27
Worcester	0	----	0	15	3	1	0	2	0	15	50
Rhode Island:											
Pawtucket	1	----	----	1	----	0	0	----	0	1	----
Providence	0	----	0	48	2	0	0	2	0	20	59
Connecticut:											
Bridgeport	0	----	1	7	0	0	0	0	0	0	29
Hartford	0	----	0	9	2	0	0	0	0	10	37
New Haven	0	----	0	15	4	0	0	1	0	5	29
New York:											
Buffalo	0	----	0	23	2	7	0	4	0	22	107
New York	19	6	0	80	36	36	0	80	6	140	1,240
Rochester	2	1	0	24	1	0	0	0	1	4	55
Syracuse	0	----	0	267	1	0	0	1	0	46	52
New Jersey:											
Camden	0	----	0	0	0	4	0	0	2	4	23
Newark	0	----	0	1	1	2	0	2	0	57	83
Trenton	1	----	0	0	2	3	0	2	0	2	30
Pennsylvania:											
Philadelphia	0	2	2	0	12	8	0	14	5	163	453
Pittsburgh	2	----	1	4	9	11	0	6	2	70	133
Reading	0	----	0	1	0	0	0	1	0	0	19
Scranton	0	----	----	0	----	2	0	----	0	1	----
Ohio:											
Cincinnati	0	----	1	0	2	7	0	5	1	3	130
Cleveland	0	4	0	4	3	9	0	8	1	75	175
Columbus	0	----	0	3	3	1	0	1	0	15	67
Toledo	0	----	0	14	3	3	0	1	0	81	65
Indiana:											
Anderson	0	----	0	0	0	1	0	0	0	0	12
Fort Wayne	0	----	0	0	3	0	0	0	0	0	26
Indianapolis	1	----	0	4	6	7	0	2	1	90	101
Muncie	0	----	0	0	0	0	0	2	0	0	8
South Bend	0	----	0	0	0	0	0	1	0	24	12
Terre Haute	0	----	0	0	1	0	0	0	0	0	16

[1] Figures for Boise estimated; report not received.

City reports for week ended July 15, 1939—Continued

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Illinois:											
Alton	0	------	0	0	1	0	0	0	0	0	8
Chicago	12	------	1	10	14	35	0	33	2	112	595
Elgin	0	------	0	0	0	2	0	0	0	8	7
Moline	0	------	0	0	0	0	0	0	0	3	6
Springfield	0	------	0	0	3	0	0	1	1	1	27
Michigan:											
Detroit	3	------	0	20	6	23	0	14	2	98	220
Flint	0	------	0	2	1	0	1	0	0	7	23
Grand Rapids	0	------	0	2	2	5	0	0	0	3	29
Wisconsin:											
Kenosha	0	------	0	0	0	1	0	0	0	0	6
Madison	0	------	0	8	5	0	0	0	0	20	23
Milwaukee	0	------	0	3	4	13	0	3	0	23	91
Racine	1	------	0	1	0	1	0	1	0	6	6
Superior	0	------	0	4	0	0	0	0	0	0	11
Minnesota:											
Duluth	0	------	0	0	1	0	0	0	0	3	16
Minneapolis	0	------	1	7	3	1	0	0	0	5	96
St. Paul	0	------	0	6	4	1	0	3	0	15	64
Iowa:											
Cedar Rapids	0	------	--------	8	-------	0	0	-------	0	2	--------
Des Moines	0	------	0	1	0	2	5	0	0	0	29
Sioux City	0	------	--------	1	-------	0	0	-------	0	1	--------
Waterloo	0	------	--------	1	-------	1	0	-------	0	10	--------
Missouri:											
Kansas City	1	------	1	1	3	2	0	2	0	1	91
St. Joseph	0	------	0	0	1	1	0	1	0	2	35
St. Louis	0	------	0	1	4	2	0	6	3	39	277
North Dakota:											
Fargo	1	------	0	0	0	0	0	0	0	0	4
Grand Forks	0	------	--------	0	-------	0	0	-------	0	0	--------
Minot	0	------	0	0	0	0	0	0	0	1	3
South Dakota:											
Aberdeen	0	------	--------	1	-------	0	2	-------	0	0	--------
Sioux Falls	0	------	0	0	0	4	0	0	0	0	8
Nebraska:											
Omaha	0	------	0	1	2	2	0	0	0	5	55
Kansas:											
Lawrence	0	------	0	1	1	1	0	0	0	0	4
Topeka	0	------	0	0	0	2	0	0	0	5	28
Wichita	0	------	0	13	3	4	0	0	0	5	42
Delaware:											
Wilmington	0	------	0	1	0	2	0	0	1	1	28
Maryland:											
Baltimore	1	5	0	3	3	8	0	14	1	55	185
Cumberland	0	------	0	0	0	1	0	0	0	0	15
Frederick	0	------	0	0	0	0	0	0	0	0	4
Dist. of Col.:											
Washington	5	1	1	35	8	1	0	9	4	33	153
Virginia:											
Lynchburg	1	------	0	5	1	0	0	0	0	28	8
Norfolk	0	------	0	0	2	0	0	1	0	0	19
Richmond	0	------	0	22	1	1	0	3	0	0	63
Roanoke	0	------	0	4	1	0	0	0	0	1	13
West Virginia:											
Charleston	1	------	0	0	5	1	0	1	2	0	22
Huntington	0	------	--------	0	-------	1	0	-------	1	0	--------
Wheeling	0	------	0	0	1	0	1	1	0	5	24
North Carolina:											
Gastonia	0	------	--------	0	-------	0	0	-------	0	0	--------
Raleigh	0	------	0	0	0	0	0	0	0	6	9
Wilmington	1	------	0	1	2	0	0	0	0	0	8
Winston-Salem	0	------	0	0	0	1	0	1	0	1	8
South Carolina:											
Charleston	0	4	0	0	1	0	0	1	0	0	13
Florence	0	------	0	0	2	0	0	0	0	0	9
Greenville	0	------	0	0	2	0	0	0	0	0	25
Georgia:											
Atlanta	0	------	0	2	6	2	0	8	0	6	80
Brunswick	0	------	0	0	1	0	0	0	0	0	4
Savannah	0	1	0	0	0	0	0	2	0	14	35
Florida:											
Miami	1	4	0	0	1	0	0	2	0	5	24
Tampa	0	------	0	0	0	0	0	0	0	0	16
Kentucky:											
Ashland	0	------	0	0	0	0	0	0	0	0	5
Covington	0	------	0	0	0	0	0	6	0	0	17
Lexington	0	------	0	0	0	0	0	2	0	0	17
Louisville	2	------	0	0	4	1	0	5	1	27	2

City reports for week ended July 15, 1939—Continued

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Tennessee:											
Knoxville	0	------	0	0	0	3	0	1	0	0	25
Memphis	0	------	1	0	0	0	0	8	1	76	84
Nashville	0	------	0	0	0	0	0	4	2	15	53
Alabama:											
Birmingham	0	2	0	0	5	0	0	3	1	6	67
Mobile	0	6	0	0	1	1	0	0	0	1	21
Montgomery	0	------	--------	0	-------	0	0	-------	0	1	--------
Arkansas:											
Fort Smith	0	------	--------	0	-------	0	0	-------	0	0	--------
Little Rock	0	------	1	0	9	1	0	6	0	0	16
Louisiana:											
Lake Charles	0	------	0	1	0	0	0	0	0	0	2
New Orleans	0	------	0	6	5	4	0	10	7	21	160
Shreveport	0	------	0	1	6	0	0	1	4	1	45
Oklahoma:											
Oklahoma City	0	------	0	0	3	1	0	0	0	0	45
Texas:											
Dallas	0	------	0	1	7	0	0	2	4	2	62
Fort Worth	0	------	0	2	3	1	0	4	0	0	37
Galveston	0	------	0	0	0	0	0	0	0	0	17
Houston	4	------	0	11	9	2	0	9	4	14	98
San Antonio	1	------	0	1	7	0	0	3	1	0	79
Montana:											
Billings	0	------	0	0	0	1	0	2	0	1	12
Great Falls	0	------	0	9	0	2	0	0	0	0	8
Helena	0	------	0	0	0	0	0	0	0	0	2
Missoula	0	------	0	0	0	1	0	0	0	0	7
Idaho:											
Boise	-------	------	--------	-------	-------	-------	-------	-------	-------	--------	--------
Colorado:											
Colorado Springs	0	------	0	0	0	6	0	1	0	0	7
Denver	4	------	0	7	5	0	0	4	0	14	83
Pueblo	0	------	0	2	0	0	0	0	0	21	3
New Mexico:											
Albuquerque	0	------	0	0	0	1	0	1	2	0	11
Utah:											
Salt Lake City	0	------	0	7	1	2	0	0	2	19	39
Washington:											
Seattle	0	------	0	198	1	1	0	5	0	7	97
Spokane	0	------	0	7	2	1	0	0	2	0	29
Tacoma	0	------	0	2	3	0	0	0	0	0	27
Oregon:											
Portland	1	------	0	3	7	1	0	1	0	3	59
Salem	0	------	--------	2	-------	0	0	-------	0	0	--------
California:											
Los Angeles	6	8	0	76	2	12	0	18	0	15	324
Sacramento	2	1	1	12	3	3	4	1	0	1	32
San Francisco	1	3	1	3	3	2	0	10	0	5	153

State and city	Meningitis, meningococcus		Poliomyelitis cases	State and city	Meningitis, meningococcus		Poliomyelitis cases
	Cases	Deaths			Cases	Deaths	
Massachusetts:				Minnesota:			
Boston	1	0	0	Minneapolis	0	0	1
New York:				St. Paul	0	0	1
New York	0	0	2	South Carolina:			
Rochester	0	0	1	Charleston	0	0	3
New Jersey:				Greenville	0	0	1
Camden	0	0	1	Tennessee:			
Newark	1	0	1	Nashville	0	0	1
Pennsylvania:				Alabama:			
Pittsburgh	0	0	1	Birmingham	0	0	1
Indiana:				Arkansas:			
Indianapolis	2	0	1	Little Rock	0	0	1
Illinois:				Texas:			
Chicago	0	0	1	San Antonio	0	0	1
Michigan:				California:			
Detroit	0	0	6	Los Angeles	0	0	5
				San Francisco	0	0	1

Encephalitis, epidemic or lethargic.—Cases: Springfield, Ill., 2; Milwaukee, 1; Topeka, 2.
Pellagra.—Cases: Savannah, 1; Louisville, 4; San Francisco, 1.
Typhus fever.—Savannah, 1; Miami, 1; Mobile, 2; Lake Charles, 1; Galveston, 1; Houston, 1.

FOREIGN AND INSULAR

CANADA

Provinces—Communicable diseases—Week ended July 1, 1939.—During the week ended July 1, 1939, cases of certain communicable diseases were reported by the Department of Pensions and National Health of Canada as follows:

Disease	Prince Edward Island	Nova Scotia	New Brunswick	Quebec	Ontario	Manitoba	Saskatchewan	Alberta	British Columbia	Total
Cerebrospinal meningitis		1		1						2
Chickenpox		8	4	64	145	15	11	16	29	292
Diphtheria		1		29	1	3	1			35
Dysentery				2	1					3
Influenza		8			5				29	42
Lethargic encephalitis				1	1					2
Measles		5	6	434	582	68	1	12	2	1,111
Mumps	3			18	55	4			4	84
Pneumonia	2	7			1				7	17
Poliomyelitis					3					3
Scarlet fever			17	62	70	15	9	10	4	187
Smallpox							1			1
Trachoma							10			10
Tuberculosis		16	13	84	73	52		4		242
Typhoid and paratyphoid fever			2	18						20
Whooping cough		19	13	60	98	15	35	6	43	289

JAMAICA

Communicable diseases—4 weeks ended July 8, 1939.—During the 4 weeks ended July 8, 1939, cases of certain communicable diseases were reported in Kingston, Jamaica, and in the island outside of Kingston, as follows:

Disease	Kingston	Other localities	Disease	Kingston	Other localities
Cerebrospinal meningitis		1	Poliomyelitis	1	
Chickenpox	6	27	Puerperal septicemia		2
Diphtheria	8	9	Tuberculosis	44	77
Dysentery	5	2	Typhoid fever	6	76
Leprosy		2			

VIRGIN ISLANDS

Notifiable diseases—April–June 1939.—During the months of April, May, and June 1939, cases of certain notifiable diseases were reported in the Virgin Islands as follows:

Disease	April	May	June	Disease	April	May	June
Chickenpox	155	73	23	Pneumonia	1	7	1
Filariasis	15			Syphilis	6	10	13
Gonorrhea	5	11	9	Tetanus	1		
Hookworm disease		1	3	Tuberculosis	5	3	2
Malaria	1			Whooping cough	1		
Pellagra	2	2					

YUGOSLAVIA

Communicable diseases—4 weeks ended June 18, 1939.—During the 4 weeks ended June 18, 1939, certain communicable diseases were reported in Yugoslavia as follows:

Disease	Cases	Deaths	Disease	Cases	Deaths
Anthrax	26	3	Poliomyelitis	2	1
Cerebrospinal meningitis	44	22	Scarlet fever	202	1
Diphtheria and croup	406	26	Sepsis	8	3
Dysentery	14	1	Tetanus	55	27
Erysipelas	163	4	Typhoid fever	174	14
Favus	5		Typhus fever	38	1
Paratyphoid fever	19				

CHOLERA, PLAGUE, SMALLPOX, TYPHUS FEVER, AND YELLOW FEVER

NOTE.—A table giving current information of the world prevalence of quarantinable diseases appeared in the PUBLIC HEALTH REPORTS for July 28, 1939, pages 1409–1421. A similar cumulative table will appear in future issues of the PUBLIC HEALTH REPORTS for the last Friday of each month.

Cholera

Ceylon—Batticaloa.—During the week ended July 8, 1939, 1 death from cholera was reported in Batticaloa, Ceylon.

Plague

Argentina—Mendoza Province—Santa Rosa.—During the period July 1–15, 1939, 1 case of plague with 1 death was reported in Santa Rosa, Mendoza Province, Argentina.

British East Africa—Nyasaland—Central Shire District.—During the week ended July 22, 1939, plague was reported present in Central Shire District, Nyasaland, British East Africa.

Hawaii Territory—Island of Hawaii—Hamakua District—Paauilo.—A rat found on June 23, 1939, in Paauilo, Hamakua District, Island of Hawaii, Hawaii Territory, has been proved positive for plague.

Smallpox

On vessel—S. S. Atalaya.—On July 25, 1939, 1 case of smallpox was reported on the Brazilian S. S. *Atalaya* at New Orleans, La. Everyone on board the vessel was vaccinated and all precautions were taken.

Yellow fever

Guinea (French).—For the period June 1–10, 1939, 2 cases of yellow fever were reported in French Guinea.

Niger Territory—Konni Circle.—During the period June 11–20, 1939, 3 cases of yellow fever were reported in Konni Circle, Niger Territory.

Senegal.—For the period June 11–20, 1939, yellow fever was reported in Senegal as follows: Bambey, 1 case; Diourbel, 6 cases; Ziguinchor, 10 cases, 5 deaths.

Sudan (French)—Bandiagara.—For the period June 11–20, 1939, 1 case of yellow fever was reported in Bandiagara, French Sudan.

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