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ENDEMIC FLUOROSIS AND ITS RELATION TO DENTAL CARIES[1]

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INTRODUCTION

The first thorough study of mottled enamel, that of Black (*1*) and McKay (*2*) at Colorado Springs and including the Pike's Peak watershed, reported as early as 1916 that, in regard to caries, the teeth of these Colorado children compare favorably with those of other communities where endemic mottled enamel is unknown. Black also wrote of the difficulty of successfully filling carious mottled enamel teeth and stated that, though the percentage of carious teeth is less than in non-endemic areas, probably a greater proportion of filled teeth are eventually lost because of the difficulty of retaining fillings in the hypoplastic tooth structure.

Workers in other countries have also commented on the qualitative aspects of this phenomenon. Mottled enamel is endemic in the southwestern part of the Japanese Archipelago. Masaki (*3*) reported 18 endemic areas in the Prefectures of Hyogo, Fukuoka, Ehime, Hiroshima, and Aichi, 12 of the 18 being located in Hyogo and Fukuoka. In an English abstract of his original report, this investigator states that "It is also remarkable that the percentage of dental caries is comparatively small among those who suffer this abnormality." The number of observations upon which this generalization was based is not stated in the abstract.

Ainsworth has commented on the lessened prevalence of dental caries among children in the endemic areas of Maldon and Heybridge, Essex County, England. In connection with the studies of the Committee for the Investigation of Dental Diseases, of the Medical Research Council, this investigator (*4*) examined approximately 4,000 children in the public elementary schools in various parts of England and Wales. He states (*5*) that the condition of the teeth in the Council schools at Maldon and Heybridge [2] was generally good, being well above the average for Council schools. He specifically notes that "there was relatively little caries: 7.9 percent of the permanent teeth were carious, as compared with an average in all

[1] From the Division of Infectious Diseases, National Institute of Health.
[2] A total of 214 children was examined in the 2 schools.

districts examined of 13.1 percent; and 12.9 percent of deciduous teeth were carious against 43.3 percent in all districts." The percentages just quoted are reported as corrected for age distribution (*4*) in the different schools.

Erausquin (*6*) who has studied mottled enamel extensively in the Argentine Republic records that there appears to be an inverse variation between dental caries and "dientes veteados," the name by which endemic dental fluorosis is known in the Argentine. He stated, however, that the findings were not conclusive on the basis of the limited number of areas studied.

Probably the first attempt to study specifically the relationship of mottled enamel to dental caries was made by McKay (*7*) who, in 1929, attacked the hypothesis that dental decay might be superinduced by "defective" enamel structure, by citing as evidence the observation that mottled enamel teeth, which probably constitute "the most poorly constructed enamel of which there is any record in the literature of dentistry," do not appear to show any greater liability to dental caries than do normally calcified teeth.

His report refers to studies made at Bauxite (Ark.), Minonk (Ill.), Towner (Colo.), Bruneau (Idaho), and the Pima Indian School at Sacaton (Ariz.). Certain tabulated data from the last three named places are included in McKay's report. Table 1 has been compiled from certain of these data.

TABLE 1.—*Variation in prevalence of dental caries in normal and mottled enamel teeth of three endemic areas according to McKay*

Locality	Number of children examined	Total number of permanent teeth examined	Number of teeth examined and percentage with dental caries				
				All teeth		Molar teeth	
				Number examined	Percent carious	Number examined	Percent carious
Towner, Colo. (Pop. 154 in 1930).	[1] 55	1,264	Normal teeth	879	11	254	46
			Mottled enamel teeth	385	9	101	42
Bruneau, Idaho (Pop. 481 in 1930).	[1] 54	1,142	Normal teeth	356	16	126	64
			Mottled enamel teeth	797	8	213	33
Pima Indian School, Sacaton, Ariz. (Pop. unstated).	[1] 78	2,178	Normal teeth	283	22	99	81
			Mottled enamel teeth	1,895	14	529	58

[1] Age, sex, color, continuity of residence, and constancy of exposure to the mottled enamel-prodncing waters not recorded in the report.

In 1933–34 a study was begun by the United States Public Health Service to determine the minimal threshold of toxicity of chronic endemic dental fluorosis. In this study (*8*) (*9*) (*10*) consecutive monthly water samples were received from each of the cities surveyed, which permitted the computation of an arithmetic mean annual fluoride (F) content of the communal water supply. The clinical examinations in these cities were limited to those children who were

born in the community, had always resided there, and had continuously used the common water supply for both drinking and cooking.

In certain of these cities, in addition to recording the degree of severity of mottled enamel, each child was examined for other defects of the enamel, such as present caries, past caries (fillings or extractions), pits and fissures, hypoplasias, etc. The examinations were made in a good light with the child seated facing a window. Mouth mirrors free from blemishes and new explorers were used. For each child examined in connection with the caries aspects of the study, the facts with respect to residence and continual use of the common water supply were verified by an interview with the child's parent or guardian.

The amount of caries recorded may appear somewhat higher than usual; for, in addition to definite cavitation, defects in the enamel on caries-susceptible surfaces showing either a discoloration or opacity around the edges and in which an explorer would cling, were counted as caries. All examinations were made, however, by one individual, the writer.

An analysis of these data indicates that a higher percentage of caries-free children is found in cities whose water supplies contain relatively toxic amounts of fluorides than in those communities with water supplies not so affected. Since in certain cities only the nine year old children were examined, comparisons will be limited to children of this age. It was decided also to omit cities where less than 25 children were examined at this age. Table 2 presents the pertinent data.

TABLE 2.—*Percentages of caries-free children, 9 years of age, in 6 selected cities classified according to their continuous use of water of different fluoride (F) concentration*

Locality	Actual community mottled enamel index	Domestic water supply [1]		Number of children examined
		Fluoride (F) content	Total hardness	
		p. p. m.	*p. p. m.*	
Pueblo, Colo.	Negative	0.6	303	49
Junction City, Kans.	Negative	0.7	277	30
East Moline, Ill.	Border-line	[2] 1.5	242	35
Monmouth, Ill.	Slight	1.7	288	29
Galesburg, Ill.	Slight	1.8	237	39
Colorado Springs, Colo.	Slight	2.5	20	54

[1] For detailed mineral analyses of these waters, see ref. (*8*) (*10*).
[2] Subject to possible correction to 1.3 p. p. m.

TABLE 2.—*Percentages of caries-free children, 9 years of age, in 6 selected cities classified according to their continuous use of water of different fluoride (F) concentration*—Continued.

Locality	Caries-free children					
	All teeth		Permanent		Deciduous	
	Number	Percent	Number	Percent	Number	Percent
Pueblo, Colo.	3	6	18	37	4	9
Junction City, Kans.	0	0	8	26	1	3
East Moline, Ill.	2	6	4	11	8	23
Monmouth, Ill.	6	21	16	55	6	21
Galesburg, Ill.	8	20	22	56	11	28
Colorado Springs, Colo.	13	24	22	41	21	40

Fluoride content in p. p. m.	Number examined	Composite sample of above 9-year-old children classified on the basis of exposure to domestic water of lower and higher fluoride (F) concentration					
0.6–1.5	[3] 114	5	4	30	26	13	11
1.7–2.5	[4] 122	27	22	60	49	38	31

[3] Of this group, 51 were boys and 63 girls; by color, 108 white and 6 colored.
[4] In this group 59 were boys and 63 girls; classified according to color, 116 white, 4 colored, and 2 Mexican.

The data shown in table 2 indicate a greater freedom from dental caries in the 122 children exposed to domestic waters of higher fluoride (F) concentration, both with respect to permanent and deciduous teeth. It is a well known fact that deciduous teeth are seldom affected with mottled enamel; in this particular group, only 3 children, all of Colorado Springs, showed even the mildest forms of mottled enamel in their deciduous teeth, generally in the second deciduous molars. Of the 122 children in the group, 60 were caries-free with respect to the permanent teeth. Of these 60, 33, or 55 percent, were affected with mottled enamel. In the whole group (122), the incidence of mottled enamel was 53 percent. These observations suggest that the limited-immunity-producing factor present in the water is operative whether or not the tooth is affected by mottled enamel. Whether this mechanism functions locally, systemically, or both ways, is not known.

RELATION OF ENDEMIC FLUOROSIS TO DENTAL CARIES IN LARGE POPULATION GROUPS

Source of data.—The disclosure of an inverse relation between the prevalence of dental caries and the fluoride concentration of the domestic water supply, as shown in table 2, raises the question of the kind of relationship between these two variables in other and larger population groups. The requisite data on dental caries are provided by the dental survey of school children 6–14 years of age, made in 26

States in 1933–34 (*11*) under the direction of the United States Public Health Service. This survey included a total of 34,283 examinations of white children in South Dakota, 15,465 in Colorado, and 48,628 in Wisconsin, made by dentists reported as using a mouth mirror and explorer in making the examinations. Furthermore, these examinations were made on a standard examination form and largely for the purpose of recording the amount of dental caries in the school population; the marked differences, therefore, in the amount of caries noted in groups using domestic waters of different mineral composition takes on an added significance.

Data on mottled enamel, on the other hand, are furnished by a recent (1938) survey of South Dakota made by the writer. During this survey (April–May 1938), approximately 3,300 school children in 51 communities were examined for mottled enamel, and endemic mottled enamel was demonstrated in 35 communities, each having a common water supply. A comparable degree of mottled enamel was widely prevalent in the surrounding rural districts in certain of the counties, ascribable to the general custom of farmers of obtaining their domestic water supply from artesian wells in the Dakota sandstone. Moreover, the examination of school children with discontinuities in their residence pointed to 21 other places in the State, not as yet surveyed, but where, on the basis of clinical signs present in the children, mottled enamel is endemic.

Method of analysis.—All South Dakota counties listed in Bulletin No. 226 (*11*) in which 35 percent or more of the estimated population of ages 6–14 years had been examined, were selected for study. On the basis of the mottled enamel data, these counties were divided into three groups: (*a*) Counties where mottled enamel is prevalent, (*b*) counties where mottled enamel distribution is uneven, and (*c*) counties which, so far as we know, are entirely free from mottled enamel. Both the 1933–34 dental needs survey and the 1938 mottled enamel studies were made in those South Dakota counties lying east of the Missouri River.

In computing an index which might point out differences in dental caries in the several counties, it was decided to express the amount of caries (severity) in terms of the number of carious permanent teeth per 100 children. In order to study that age group with the maximum number of permanent teeth in the mouth, the 12–14 year group was selected for study. All children referred to in the tables to follow are white. The amount of caries was determined by combining the data associated with the following items: "Caries, permanent teeth," "Extraction indicated, permanent teeth," "Filled permanent teeth," and "Extracted permanent teeth." For each of these items, the bulletin gives the number of carious permanent teeth per 100 children. Adjustment was made for sex,

and the amount of caries for each county was expressed in terms of the number of carious permanent teeth per 100 children. In Public Health Bulletin No. 226, examinations from communities with a population under 5,000 were combined with examinations from the rural areas and designated "balance of county."

The South Dakota counties selected from the bulletin were classified solely on the basis of the prevalence of or freedom from endemic mottled enamel as shown by the mottled enamel study. The cities of Aberdeen, Huron, and Sioux Falls were classified on the basis of whether or not the common water supply was producing mottled enamel. The results of these computations are contained in table 3. Similar computations were also made for four Colorado cities and eight Wisconsin cities; the results are shown in table 4.

DISCUSSION

This paper, after reviewing the findings of the earlier workers in the field, submits evidence that furnishes support to the hypothesis that a limited immunity from dental caries is operative among school children residing in endemic mottled enamel areas. This evidence may be summarized as follows:

Prevalence.—Observations made on a selected sample of 9-year old children continuously exposed to waters of different fluoride concentrations, with the history of exposure personally verified in each instance by an interview with the child's parent, indicate that a high percentage of children are caries-free in those places where the common water supply contains appreciable amounts of fluorides. For instance, of the 114 children who had continuously used a domestic water comparatively low in fluorides (0.6 to 1.5 p. p. m.), only 5, or 4 percent, were caries-free. On the other hand, of the 122 children of comparable age who had continuously used domestic waters containing 1.7 to 2.5 p. p. m. of fluorides, 27, or 22 percent, were caries-free. In other words, within the range of these observations, limited to a total of 236 nine-year old children, the percentage of caries-free children in areas with domestic waters containing appreciable amounts of fluorides was over 5 times the corresponding percentage in areas with domestic water containing lower fluoride concentrations.

Severity, or amount of caries.—To provide additional evidence of the relation of dental caries to endemic fluorosis, a computation of the dental caries attack rate on the permanent teeth of 12–14-year old children was made with the use of data collected in 1933–34 (Public Health Bulletin No. 226) and correlated with the data subsequently obtained on the geographical distribution of mottled enamel in South Dakota. Briefly, this study shows that, in the group of counties [3]

[3] The city of Aberdeen is included in this group of counties as this city was using a deep well water supply at the time of the dental caries survey. This water supply produced a mild degree of mottled enamel in about 20 percent of the children continuously using the water.

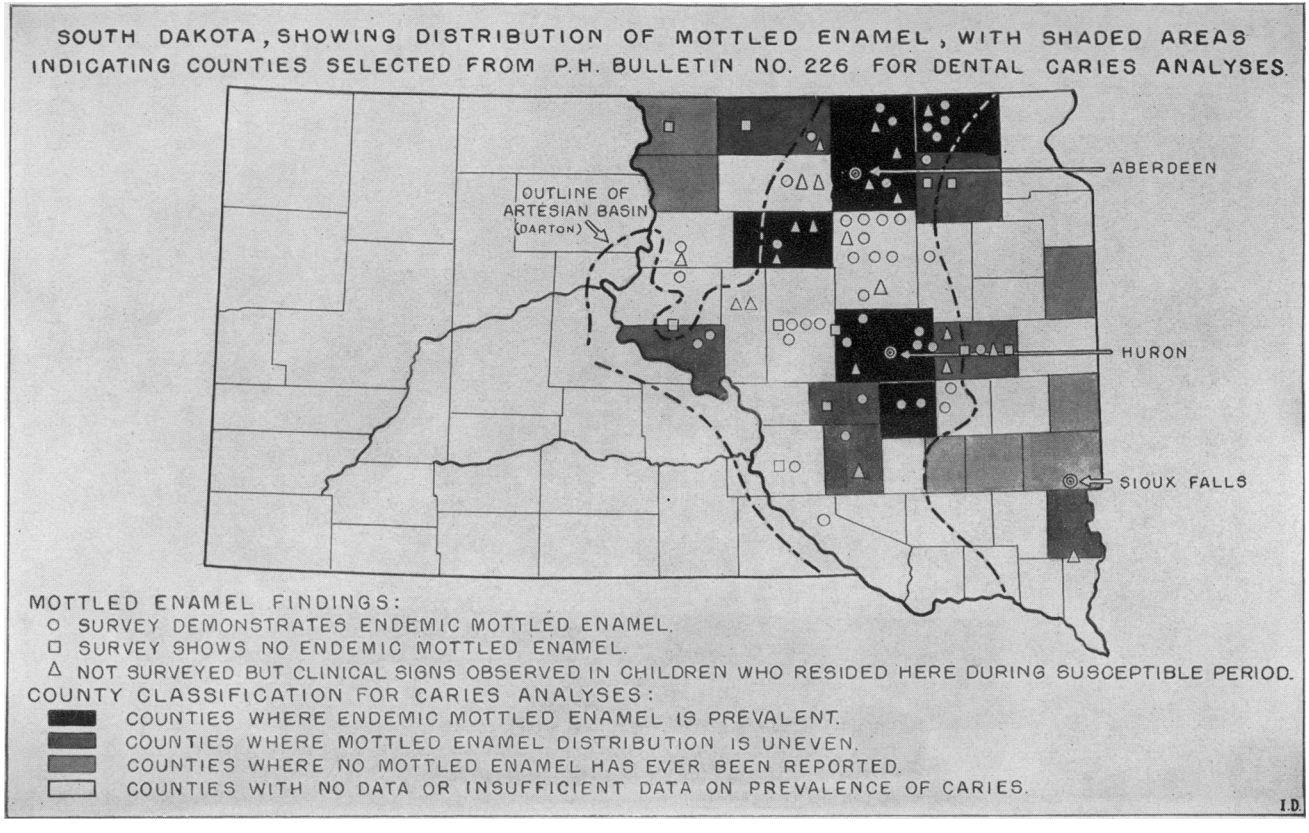
SOUTH DAKOTA, SHOWING DISTRIBUTION OF MOTTLED ENAMEL, WITH SHADED AREAS INDICATING COUNTIES SELECTED FROM P.H. BULLETIN NO. 226 FOR DENTAL CARIES ANALYSES.
OUTLINE OF ARTESIAN BASIN (DARTON)
ABERDEEN
HURON
SIOUX FALLS
MOTTLED ENAMEL FINDINGS:
○ SURVEY DEMONSTRATES ENDEMIC MOTTLED ENAMEL.
□ SURVEY SHOWS NO ENDEMIC MOTTLED ENAMEL.
△ NOT SURVEYED BUT CLINICAL SIGNS OBSERVED IN CHILDREN WHO RESIDED HERE DURING SUSCEPTIBLE PERIOD.
COUNTY CLASSIFICATION FOR CARIES ANALYSES:
COUNTIES WHERE ENDEMIC MOTTLED ENAMEL IS PREVALENT.
COUNTIES WHERE MOTTLED ENAMEL DISTRIBUTION IS UNEVEN.
COUNTIES WHERE NO MOTTLED ENAMEL HAS EVER BEEN REPORTED.
COUNTIES WITH NO DATA OR INSUFFICIENT DATA ON PREVALENCE OF CARIES.
I.D.

FIGURE 1.

TABLE 3.—*Dental caries attack rates in permanent teeth of 12–14 year old white children in selected South Dakota counties and cities classified according to the prevalence of mottled enamel*

County	Number of children examined (12–14 years)	Number of carious permanent teeth per 100 children	Remarks
(a) COUNTIES WHERE MOTTLED ENAMEL GENERALLY IS PREVALENT			
Beadle (less Huron)	332	256	Mottled enamel general throughout county. Areas include Hitchcock, Wolsey, Virgil, Yale, Cavour, and rural districts.
Brown (city, Aberdeen)	653	203	On basis of clinical examinations, old city deep well water contained fluorides in excess of minimal threshold.
Faulk	266	149	Mottled enamel general throughout county. Areas include Faulkton, Orient, Cresbard, Chelsea, and rural districts.
Marshall	391	251	Mottled enamel severe in western half of county, including Kidder, Britton, Langford, Newark, Amherst, and rural districts. No information on eastern half of county.
Sanborn	260	[1] 103	Mottled enamel prevalent in county including Artesian and numerous rural districts.
Total	1,902	201	
(b) COUNTIES WHERE MOTTLED ENAMEL DISTRIBUTION IS UNEVEN			
Jerauld	295	294	Alpena and Wessington Springs are negative; some mottled enamel in and around Lane.
Aurora	340	227	Mottled enamel around Stickney and rural districts in northern part of county.
Kingsbury	398	330	Distribution varied, Iroquois, Bancroft, Esmond, and Lake Preston are endemic. DeSmet and Arlington, two largest communities in county are negative.
Day	666	369	Some mottled enamel in extreme western part of county around Pierpont. Bristol and Andover are negative by survey. No indications of mottled enamel in any other section of county.
Hughes	184	206	Blunt negative for mottled enamel; cases being developed in rural district around Harrold.
McPherson	346	394	Some mottled enamel in extreme eastern part of county around Leola. Eureka surveyed and negative. County generally free of mottled enamel.
Lincoln	536	284	Some mottled enamel observed from Beresford; no other record of mottled enamel in county.
Total	2,765	314	
(c) COUNTIES WHERE NO MOTTLED ENAMEL HAS EVER BEEN REPORTED			
Beadle (city, Huron)	436	398	Negative for mottled enamel; obtains city water from James River with deep well as a reserve.
Campbell	264	368	No record of mottled enamel in this county. Herreid negative by survey.
Deuel	212	218	No reports of mottled enamel in this county.
Hanson	271	382	Do.
McCook	344	407	Do.
Minnehaha:			
City, Sioux Falls	608	451	No reports of mottled enamel in this city; State chemist reports 0.4 p. p. m. F in treated city water.
Balance of county	584	476	No reports of mottled enamel in this county.
Moody	433	498	Do.
Walworth	329	355	Do.
Total	3,481	415	

[1] Only "caries and extractions indicated" of the permanent teeth are listed in Bulletin No. 226 for this county; adjusted for sex shows 68.6 per 100 children. This figure was raised to 103.1 per 100 children in order to compensate for the "unknown" filled and extracted permanent teeth per 100 children. The increase was based on the average ratio that these two missing items bear to the "caries and extraction indicated" reported in the 4 counties adjoining Sanborn County. The percentages that these items bear to the whole were as follows: Jerauld, 31; Kingsbury, 34; Beadle (less Huron) 34, and Aurora, 35.

where mottled enamel is generally prevalent, an examination of 1,902 white children, 12–14 years of age, disclosed 201 carious permanent teeth per 100 children. In the intermediate group of seven counties where the mottled enamel distribution was uneven, and at times sporadic, the examination of 2,765 children showed 314 permanent teeth affected per 100 children; and in the third group of counties and the cities of Huron and Sioux Falls, where no endemic mottled enamel areas are known to exist, an examination of 3,481 children showed a dental caries attack rate of 415 permanent teeth per 100 children. These data indicate that the dental caries attack rate in this particular population is inversely proportional to the prevalence of mottled enamel.

TABLE 4.—*Dental caries attack rates in permanent teeth of 12–14-year old white children of ALL Colorado and Wisconsin cities listed in Public Health Bulletin No. 226*

City	Number of children examined (12–14 years)	Number of carious permanent teeth per 100 children	Fluoride (F) content of common water supply (p. p. m.)	Reference
	Colorado			
Colorado Springs	208	162	2.5	(8)
Pueblo	411	[1] 194	0.6	(8)
Denver	637	342	0.5	(12)
Fort Collins	207	296	None	(12)
	Wisconsin			
Green Bay	687	275	[2] 2.3	----
Sheboygan	244	710	0.5	(13)
Manitowoc	661	682	0.35	(13)
Two Rivers	382	646	0.3	(13)
Milwaukee	2,645	917	0.3	(13)
West Allis	160	831	0.3	(13)
Baraboo	119	733	0.2	(13)
La Crosse	47	731	0.12	(13)

[1] "Extraction indicated" for boys "Unknown"; 4.2 rate for girls used in this adjustment.—Author.
[2] Determination made by Senior Chemist E. Elvove, Division of Chemistry, National Institute of Health. Approximately the same amount has been reported by DeWitt and Nichols (J. Am. Water Works Assoc., 29:980–984 (July 1937).

NOTE.—For the mineral constituents, other than fluorine, of these Wisconsin waters, see Public Water Supplies of Wisconsin, Wisconsin State Board of Health, July 1935.

Similar comparisons made among four cities of Colorado show that in the non-endemic communities, Pueblo, Fort Collins, and Denver, the dental caries attack rate is 194, 296, and 343, respectively; on the other hand, in the endemic area, Colorado Springs, only 163 permanent teeth per 100 children were affected. Applying the same methods of study to eight Wisconsin cities, it was found that in the seven where no endemic mottled enamel is known to exist and where the fluoride

(F) content of the communal water supplies ranges from 0.1 to 0.5 p. p. m., the severity of dental caries showed rates from 646 to 917 carious permanent teeth per 100 children. But in the city of Green Bay, where the city water contains 2.3 p. p. m. of fluoride (F), only 275 carious permanent teeth per 100 children were recorded.

It is fully realized, of course, that the causes of dental caries are, as Rosenau states, "complex and perhaps multiple." Mill's recent analysis of Public Health Bulletin No. 226,[4] moreover, indicates that the dental caries attack rates (amount of dental caries) vary markedly in different geographical regions.

An inspection of the data included in this paper brings out the fact that, regardless of the dental caries attack rate of the region, the use of a domestic water which in itself is capable of producing mottled enamel is concomitant with a lower amount of dental caries.

Relation to dental caries research.—The relationship of chronic endemic dental fluorosis (mottled enamel), a water-borne disease, to dental caries raises several questions, for example:

1. What role, if any, does the physical structure of a tooth play in either susceptibility to or immunity from dental caries?

2. Is the higher fluoride content of the enamel of a mottled-enamel tooth the immunity-producing factor?

3. Is the limited immunity due, directly or indirectly, to the well-known inhibitory action of fluorine on enzymatic processes?

4. While on the basis of our present knowledge it appears justifiable to associate the observed results with the presence of fluorides in the domestic water, the possibility should not be overlooked that other elements of comparatively rare occurrence in water or ordinary constituents of drinking water present in unusually large concentration may directly or through a synergistic action with the fluoride, produce the observed effects. For this reason, it appears essential to obtain as complete chemical analyses as possible of the domestic water of communities which are under investigation for dental caries.

SUMMARY

1. Examinations of 236 nine-year old children with verified continuity of exposure showed that a higher percentage of children is caries-free in those communities where the domestic water supplies contain higher concentrations of fluorides (F) in comparison with communities using waters of lower fluoride concentrations. This limited immunity to dental caries seemed operative with respect to the deciduous teeth as well as the permanent teeth.

2. An analysis of dental caries attack rates in a relatively large number of children in the three States thus far studied (South

[4] J. Dent. Res., *16*: (Oct.) 1937.

Dakota, Colorado, and Wisconsin) indicates that the severity of dental caries is, in general, lower in mottled enamel areas as compared with normal areas in the same State.

3. Inasmuch as it appears that the mineral composition of the drinking water may have an important bearing on the incidence of dental caries in a community, the possibility of partially controlling dental caries through the domestic water supply warrants thorough epidemiological-chemical study.

ACKNOWLEDGMENT

The outline of the artesian basin in eastern South Dakota shown in figure 1 was taken from plate LXIX, by N. H. Darton, in part 2 of the Seventeenth Annual Report of the United States Geological Survey, 1895–96.

The writer desires to express his indebtedness to Senior Statistician Wm. M. Gafafer and Senior Chemist E. Elvove, National Institute of Health, for many helpful suggestions and criticisms in the preparation of this paper, to the Wisconsin State Board of Health for supplying information on the fluoride content of the water supplies of the seven Wisconsin cities with high dental caries attack rates, and to Principal Statistician Selwyn D. Collins, National Institute of Health, for a review of the paper.

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SILICOSIS AND SIMILAR DUST DISEASES

Medical Aspects and Control

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The pneumoconioses represent a class or type of diseases of the lung which develop slowly as a result of occupational exposure. The term "pneumoconiosis" may be applied to any pathological condition of the lung produced by the inhalation of dust. It is common further to classify pneumoconiosis according to the chief constituents of the dust producing the condition; for example, silicosis produced by silica, asbestosis produced by asbestos, siderosis produced by iron, anthracosis produced by carbon particles, etc. Of these, silicosis and asbestosis are by far the most important from the viewpoint of number of workers exposed and the disability associated with their development.

Since silicosis is the most important, and control measures effective in preventing silicosis are applicable in the prevention of other forms of pneumoconiosis, this discussion will be limited chiefly to reporting a summary of our knowledge concerning the cause and prevention of silicosis.

The Committee on Pneumoconiosis of the Industrial Hygiene Section of the American Public Health Association (*1*), defined silicosis as follows:

> A disease due to breathing air containing silica (SiO_2), characterized anatomically by generalized fibrotic changes, and the development of miliary nodulation in both lungs, and clinically by shortness of breath, decreased chest expansion, lessened capacity for work, absence of fever, increased susceptibility to tuberculosis (some or all of which symptoms may be present), and by characteristic X-ray findings.

This definition of the disease seems to be satisfactory from a medical point of view; however, in a law recently passed in West Virginia, for the purpose of the act silicosis was defined as an insidious fibrotic disease of the lung or lungs, due to prolonged inhalation and accumulation, sustained in the course of and resulting from employment, of minute particles of dust containing silicon dioxide (SiO_2) over such a period of time and in such amounts as result in the substitution of fibrous tissue for normal lung tissues; and the term silicosis shall also include silicosis accompanied by tuberculosis of the lungs evidenced by the presence of tubercle bacillus in the sputum.

While the foregoing definitions of silicosis were obtained from sources that might be expected to have somewhat different views, all have one thing in common—that the cause is silica or quartz.

Although other dusts, when inhaled in sufficient concentrations over a long period of time, have been shown to be capable of producing a definite pulmonary fibrosis, nevertheless, the pneumoconiosis char-

acterized by nodular fibrosis has to date been shown clinically and experimentally to be associated only with the inhalation of dusts containing silica.

According to Dorland's Medical Dictionary, "Pneumoconiosis is a lung disease due to the inhalation of minute particles of dust. It is attended by fibroid induration." Pneumoconiosis is a general term, and the lung disease caused by any one dust usually takes the name of that dust, as anthracosis, silicosis, chalicosis, siderosis, and asbestosis.

SILICA IN NATURE

Silica is the most abundant constituent of the minerals and rocks that make up the crust of the earth. It occurs in two forms, free and combined. The free silicas as a group are definite compounds in the form of SiO_2. The combined forms are silicates. Of free silicas which occur in nature, that known as quartz is by far the most common. Quartz is hard mineral and chemically resistant to reagents. It is an abundant constituent of granite, schist, and other rocks, and the chief component of sandstone and quartzite. Many ores are deposited in veins that consist nearly wholly of quartz. Probably the next most common form in which free silica exists in nature is the amorphous hydrated form known as opal (SiO_2–H_2O). Opal is a silica of colloidal origin and occurs abundantly in the diatomaceous earths. It is less resistant to reagents than quartz. Another type of free silica frequently found is flint; and with flint is found chalcedony, a waxy, translucent form of silica interpreted as consisting of fibers of quartz with a small amount of interstitial opal. Other forms of free silica occurring less abundantly in nature are tridymite, cristobalite, and siliceous glass, or vitreous silica.

OCCUPATIONAL EXPOSURE TO SILICA

Owing to the fact that the earth's crust contains so great an amount of silica, it is obvious that those occupations concerned with the driving of tunnels, development of highways, and mining are frequently associated with a silicosis hazard. A second class of occupations exposing the workers to this hazard are those connected with industries that have to do with the processing and industrial use of mineral products, such as the smelting and refining of ores, the use of sand and gravel for structural purposes, the carving of stone, particularly granite, the manufacture and use of certain abrasives, and the processing of the various forms of free silica. According to Knopf (*2*), the most common forms of free silica used industrially are massive crystalline quartz, quartzite, sandstone, flint, tripoli, diatomaceous earths, and silica sand. Table 1, from Ladoo (*3*), illustrates the great variety of uses to which silica is put in industry and shows the kind of silica adapted to each purpose.

TABLE 1.—*Uses of silica*

Uses of silica	Types of silica used
Abrasive uses:	
In scouring and polishing soaps and powders	Quartz, quartzite, flint, chert, sandstone, sand, tripoli, and diatomaceous earth; all in finely ground state.
In sandpaper	Quartz, quartzite, flint, sandstone, and sand; coarsely ground and closely sized.
In sand-blast work	Quartz, quartzite, sandstone, and sand, crushed into sharp angular grains uniform in size.
Metal buffing, burnishing, and polishing	Ground tripoli and other forms of ground silica.
For sawing and polishing marble, granite, etc.	Sharp, clean sand graded into various sizes.
As whetstones, grindstones, buhrstones, pulpstones, oilstones, etc.	Massive sandstone from very fine to moderately coarse grained.
Tube-mill lining	Chert, flint, and quartzite in dense, solid blocks.
Lithographers' graining sand	Medium to fine sand or rather coarsely ground silica and tripoli.
Tube-mill grinding pebbles	Rounded flint pebbles.
In tooth powders and pastes	Various forms of pure silica finely ground.
Wood polishing and finishing	All forms of silica ground to medium fineness.
Refractory uses: In making silica firebrick and other refractories.	Fairly pure quartzite known as gannister; not less than 97 percent SiO_2 nor more than 0.40 percent alkalies, tightly interlocking grains desired.
Metallurgical uses:	
In making silicon, ferrosilicon, and silicon alloys of other metals, such as copper.	Moderately pure sand, massive crystalline quartz, sandstone, quartzite, or chert.
As a flux in smelting basic ores	Massive quartz and quartzite.
Foundry-mold wash	Ground sandstone, quartz, and tripoli.
Foundry parting sand	Fine sand and ground tripoli.
Chemical industries:	
As a lining for acid towers	Massive quartz or quartzite.
As a filtering medium	Massive diatomaceous earth and tripoli, sand, finely granular quartz or quartzite, finely ground tripoli, diatomaceous earth, and other forms of silica.
In the manufacture of sodium silicate	Pure pulverized quartz sand, pure tripoli, and diatomaceous earth.
In the manufacture of carborundum	Pure quartz sand.
Paint: As an inert extender	Finely ground crystalline quartz, quartzite, and flint; also finely ground sandstone, sand, and tripoli.
Mineral fillers: As a wood filler	Finely ground crystalline quartz, quartzite, flint, tripoli, and other types of ground silica.
In fertilizers	
In insecticides	
As a filler in rubber, hard rubber pressed and molded goods, phonograph records, etc.	Finely ground silica of all types.
In road asphalt surfacing mixtures	
Ceramic uses: In the pottery industry as an ingredient of bodies and glazes.	Flint, tripoli, and chert, and other amorphous silica preferred; also all other forms of very pure silica, all finely ground.
In the manufacture of ordinary glass	Pure quartz sand.
In the manufacture of fused-quartz chemical apparatus such as tubes, crucibles, and dishes.	Very pure massive quartz preferred.
Decorative materials: In the manufacture of gems, crystal balls, table tops, vases, statues, etc.	Rock crystal, amethyst, rose quartz, citrine quartz, smoky quartz, chrysoprase, agate, chalcedony, opal, onyx, sardonyx, jasper, etc.
Insulation:	
Heat insulation for pipes, boilers, furnaces, kilns, etc.	Massive and ground diatomaceous earth.
Sound insulation in walls, between floors, etc.	Do.
Structural materials: Sand-lime brick	Moderately pure, sharp, angular sand, preferably finer than 20-mesh, together with a small percentage of finely pulverized silica.
Optical quartz: For the manufacture of lenses and accessories for optical apparatus.	Clear, colorless, flawless rock crystal or massive crystallized quartz.

In a recent survey (*4*) carried on in a large manufacturing center, it was found that about 9 percent of the industrial workers were employed in occupations where the silica hazard required consideration. According to the census for 1930, there were gainfully employed in the manufacturing and mechanical industries in this country approximately 14 million persons. If the above mentioned survey can be accepted as representative of the occupational distribution of these workers, it appears that there are nearly 1,200,000 individuals potentially exposed to a silicosis hazard in the manufac-

turing and mechanical industries alone. Lanza and Vane (*5*), in their discussion concerning the prevalence and effect of silicosis, state: "Our very rough, but obviously conservative, estimate of the number of workers exposed to silica dust to a harmful degree in the United States is, therefore, upwards of 500,000."

FACTORS INFLUENCING THE ACTION OF SILICA DUST PARTICLES AS THE EXCITING CAUSE OF SILICOSIS

Although it has been accepted that silica is the exciting cause of silicosis, there are certain factors which must be considered as influencing its action.

Tissue reaction to dust.—Early workers were inclined to consider that the injury caused by the dust particle was due to mechanical irritation produced by its hard and cutting edges. Gardner (*6*) has shown experimentally that the inhalation of finely divided carborundum dust having sharp edges, of a greater hardness than silica particles, does not produce the nodular reaction characteristic of silicosis. Collis (*7*) was one of the early workers to draw our attention to the chemical action of dust. Gye and Kettle (*8*) have shown that silica in solution or noncrystalline form exerts a toxic action upon the tissues which leads to the proliferation of fibroblastic cells. Lately, Miller and Sayers (*9*) have reported results of experimental studies which illustrate clearly the reaction of peritoneal tissues to certain dusts. Only the silica-containing dusts have uniformly produced a proliferative reaction. Other dusts have been either completely absorbed, leaving no scar tissue, or have remained unaltered in the form in which they were injected. These latter reactions are classed as "absorptive" or "inert."

Size of dust particles.—Since dust, to exert its harmful action, must enter the finer divisions of the lung, the particle size of the atmospheric dust bears a definite relationship to the injurious effect produced. The silica must be present in the air in particles small enough to enter the finer air spaces and of such dimensions that the phagocytic cells may engulf them. The natural defenses of the respiratory tract probably prevent many particles larger than 10 microns from ever reaching the finer divisions of the lung, and such as do are likely to be expelled with the bronchial secretions. The soluble silica plays a definite part in the production of the disease, and the size of the particle also affects the rate of solution, due to the fact that the smaller the particles the greater the total surface area exposed to the action of solvents.

Table 2 shows the size distribution of various industrial dusts as compared with the dust particles observed in the outdoor air in the general atmosphere. Apparently about 70 percent of the particles found in industrial dusts generally are between 0.5 and 3 microns in

TABLE 2.—*Size-frequency distribution of various industrial dusts as compared to outdoor dust* [1]

[Average frequency in percent]

Kind of dust	Number of samples	Median	Size group in microns											
			0–0.49	0.5–0.99	1–1.49	1.5–1.99	2–2.49	2.5–2.99	3–3.49	3.5–3.99	4–4.49	4.5–4.99	5–5.49	5.5–5.99
Outdoor dust	179	0.5	56.0	41.0	2.5	0.5								
Sandblasting	9	1.4	1.4	19.7	34.7	20.3	12.6	5.2	2.8	1.6	1.1	0.2	0.2	0.2
Granite cutting	4	1.4	2.0	19.0	33.6	24.5	10.4	4.6	3.1	.6	.9	.3	1.0	
Trap-rock milling:														
Crusher house	1	1.4		18.0	39.0	33.0	10.5	2.5	2.0					
Screen house	1	1.3	2.0	31.5	33.0	16.0	10.0	4.5	2.5	.5				
Disk crusher	1	.9	10.0	48.0	31.0	6.0	3.0	1.0	1.0					
Foundry parting compound	2	1.4	.5	22.0	42.0	17.3	9.2	5.0	1.5	2.0	.5			
General foundry air	1	1.2		26.0	48.0	17.0	8.0	1.0						
Talc milling	1	1.5		16.0	32.0	20.0	13.0	7.0	3.0	2.0	2.0	2.0		1.0
Slate milling	1	1.7	1.0	13.0	29.0	17.0	14.0	14.0	6.0	4.0	1.0		1.0	
Marble cutting	1	1.5		12.0	37.0	21.0	10.0	11.0	3.0		1.0	2.0	2.0	1.0
Soapstone	2	2.4	1.2	16.0	19.0	13.0	11.0	6.0	6.5	4.5	5.5	3.3	2.5	11.5
Aluminum dust	1	2.2	3.0	8.0	20.5	14.0	11.5	9.0	6.5	3.0	3.5	4.0	7.0	10.0
Bronze dust	1	1.5	1.0	12.0	33.5	25.0	21.0	6.0	1.5					
Anthracite-coal mining:														
Breaker air	2	1.0	7.0	51.0	26.0	8.0	3.0	3.0	2.0					
Mine air	1	.9	11.0	60.0	17.0	7.0	3.0	1.0	1.0					
Coal drilling	1	1.0	1.0	55.0	34.5	7.5	1.5		.5					
Coal loading	3	.8	11.5	56.3	24.3	5.5	1.5	.7	.2					
Rock drilling	1	1.0	4.0	49.0	25.5	12.5	5.5	1.5	.5	1.5				

[1] From Public Health Bulletin No. 217, Determination and Control of Industrial Dusts, by J. J. Bloomfield and J. M. DallaValle. 1935, p. 52.

diameter. There are no doubt many times as many particles too small to count by the method used, but experimentally it has been shown that a great percentage of such submicroscopic particles are not retained in the lungs but pass out with the expired air. Sayers (*10*) has shown that less than 15 percent is retained when the finer particulate matter, such as lead in the form of fumes, is inhaled. The greater majority of particles found upon microscopic examination of the lung also fall within the limits of from 1 to 3 microns.

Another reason for considering the size of the particles as affecting the harmfulness of the dust, is that it is the larger ones that settle out rapidly while the rate of falling for the smaller particles is very slow. Figure 1 illustrates graphically this difference; those under 1 micron

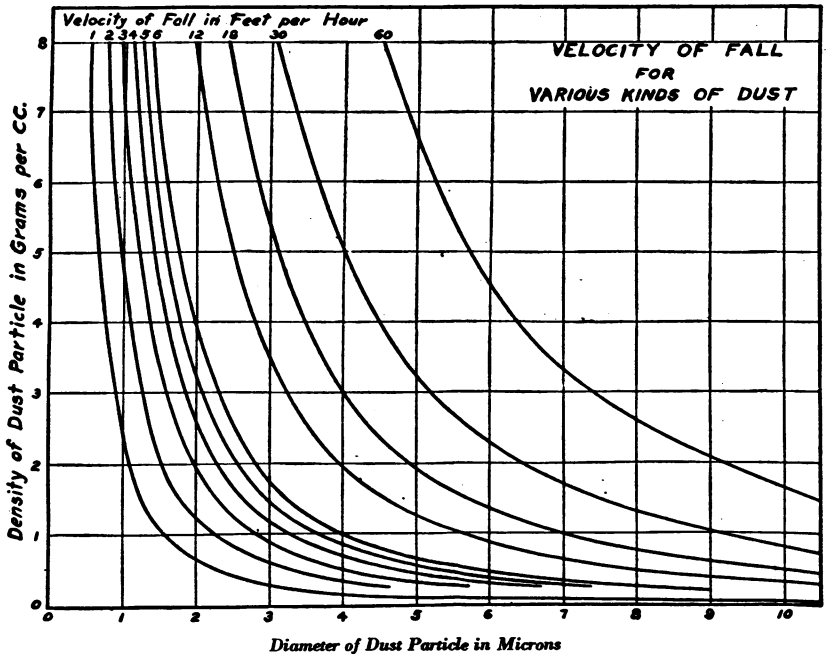

FIGURE 1.—Rapidity of settling in relation to size and density of dust particles.

fall at a rate of from 1 to 3 feet per hour, varying with the specific gravity, while a particle 5 microns in diameter of 7 specific gravity falls about 60 feet per hour. Particles of more than 10 microns would settle out in a relatively short time. The fact that the finer particles remain suspended in the atmosphere for long periods greatly increases their chance of being inhaled.

Thus we may say, from the viewpoint of etiology, that the harmfulness of a given dust containing free silica is directly influenced by the number of particles of free silica it contains less than 10 microns in diameter, and probably the greatest harm is produced by those between 1 and 3 microns.

Concentration of dust in the atmosphere.—The relationship of dust concentration and duration of exposure are closely associated in their etiological significance. The rate at which silicosis will develop, excluding certain factors considered as predisposing, depends upon the dosage of free silica. This dosage is obviously dependent upon the amount of silica in the air inhaled and the duration of exposure.

In 1902, a committee, of which Dr. J. S. Haldane was a member, reported (*11*) upon an investigation made to determine the cause of excessive mortality rates from tuberculosis among the Cornish tin miners. This group decided that it was evident that the inhalation of stone dust by these men in the performance of their tasks as miners was the cause of permanent damage produced upon the lungs. Furthermore, they noted that the condition developed gradually in the case of the ordinary miner, but rapidly in the case of the machine workers who were exposed to greater amounts of dust. Dr. Watkins-Pitchford (*12*) and Dr. Mavrogordato (*13*) and other South African workers, in their discussions relating to the etiology of silicosis, emphasize the relationship of the concentration of dust and duration of exposure to the degree of lung changes produced. Mavrogordato has suggested the advisability of intermittent employment as an aid in lessening the amount of changes produced; but aside from the fact that it does delay the reaction, it has not been shown that it could safely be relied on to prevent silicosis where exposure is to sufficient concentrations. Workers in this country have also shown the relationship of dosage to the severity of the reaction. Reports of the Picher Clinic (*14*), of the Tri-State District of Oklahoma, Kansas, and Missouri lead and zinc mines, which is a cooperative undertaking, by agents of the operators, the Metropolitan Life Insurance Co., the Bureau of Mines, and the Public Health Service have shown clearly the effects of various periods of exposure to silica as met with in the mining processes carried out in the district. Similar studies upon the health of granite workers (*15*) (*16*) likewise stress the duration of exposure necessary to produce definite degrees of silicosis.

Role played by other dusts present with silica.—Most authorities agree that the presence of other inorganic dusts in the silica-containing atmosphere may tend to influence the rate of progressive reaction resulting from inhaled silica. Some of them tend to alter somewhat the radiographic appearance so that the nodular shadows are less discrete.

Some have thought that the relative absence of silicosis in the cement industry was due to the calcium present. However, investigations (*17*) have led to the belief that the absence of evidence of extensive pulmonary fibrosis among employees in the cement industry is due to the fact that there is insufficient total exposure to free silica (considering the percent of free silica in the dust, concentration of dust, and duration of exposure).

No complete reports of pulmonary fibrosis resulting from the inhalation of organic dusts have appeared in the literature. Always when such fibrosis is associated with the breathing of air containing organic dust, either inorganic dust has been shown to be present or at least its absence has not been definitely proved.

In regard to the presence of other irritating dusts, Chapman (*18*), Kessler (*19*), MacDonald (*20*), Kilgore (*21*), and others have reported cases of so-called acute silicosis resulting from the inhalation of air containing high concentrations of silica along with strong alkali in a fine powdered form. Some have suggested that the rapid action of the silica in the production of pulmonary fibrosis is aided by the presence of these alkalis, because silica is more soluble in alkaline solutions. Kettle (*22*) failed to demonstrate such actions experimentally and states that proof is still lacking that any action of the kind may occur. Pathological reports furnish evidence of a definite fibrosis; but the entire picture differs remarkably from that of the typical silicotic, such as has been furnished by Gardner (*23*) in his reports upon the examination of tissues from clinical cases of silicosis as well as his pathological reports made from tissues obtained in his experimental studies.

Sayers, Meriwether, and Lanza (*14*) reported that of the employees in the lead and zinc mines, those who gave a history of previous coal-mining experience developed a definite silicosis in a shorter time than employees of the same age without this experience. This cannot be taken to mean, however, that the inhalation of coal dust hastened the action of silica, for it is doubtful whether any of these coal miners had worked where they were not exposed to some free silica, so that the total dosage of silica was not the same in the two groups.

OCCUPATIONAL HISTORY

An important factor in determining etiological significance of total dust exposure is a complete, carefully taken occupational history.

TABLE 3.—*Occupational record*

Dated: August 8, 1933. Office: Industrial Hygiene.
Name: Kosus Michael. Present age: 49.
Age began work: 15. Number of years worked: 34.

Specific occupation	Specific industry	Number of years in—	
		Hard coal	Non-dusty
Present: Section foreman	Anthracite coal	5	
Preceding present:			
1. Contract miner (chamber)	do	15	
2. Miners' laborer (chamber)	do	3	
3. Mule driver (dry mine)	do	3	
4. Patcher (dry mine)	do	2	
5. Slate picker (dry breaker)	do	2	
6. Farm laborer (Pa.)	Agriculture		1

Remarks: Estimates total time idle during working life—3 years. By recording the complete data, the total dust exposure may be determined and compared to other cases of similar severity.

Table 3a shows the activities and associated dust exposures of chamber miners and laborers. It will be seen that each activity engaged in by those coal miners was studied separately. Experience has shown that the various occupations comprising the process of any dusty occupation are usually associated with dissimilar dust exposure.

TABLE 3A.—*Dust exposure of contract miners and helpers (laborers—Chamber mining*

Activity	Number of samples	Number of hours in activity	Average dust count, millions of particles per cubic foot of air	Millions of particle-hours per cubic foot
Jack-hammer drilling	23	1	575	575
Hand loading	22	2	1,138	2,276
After firing	7	¼	834	209
Taping and wiring	2	½	40	20
Setting up props, and in main airways	8	2¾	15	41
Total	62	6½		3,121

3,121 million particle-hours per cubic foot equals 480 million particles per cubic foot 6½ hours.

It is apparent from this analysis that the hand loading of coal contributes about 73 percent of the total dust exposure of chamber miners and laborers, although this occupation comprises less than one-third of the total working hours. Jack-hammer drilling, which occupied but 1 hour of the 6½-hour miners' day, amounts to 18 percent of the total exposure, while the practice of entering a room too soon after blasting unduly exposes miners to a large amount of dust, this quantity being about 7 percent of the total dust exposure. Table 4 shows the method of analysis used to determine an estimate of the individual's total dust exposure.

TABLE 4.—*Estimation of the individual's total dust exposure*

Occupation	Number of years	Dust concentration in millions of particles per cubic foot (average)	Millions of particle-years per cubic foot
Slate pickers (dry mine)	2	380	760
Patchers (dry mine)	2	71	142
Mule driver (dry mine)	3	71	213
Miner's laborer (chamber)	3	480	1,440
Contract miner (chamber)	15	480	7,200
Section foreman	5	7	35
Total	30		9,790

$\frac{9{,}790 \text{ millions of particle-years per cubic ft.}}{30} = 326$ millions of particle years per cubic foot.

More detailed application of our knowledge of the true cause of silicosis is illustrated in table 5. In this study of the actual duties required by the job, it is revealed that the greatest hazard, so far as molding operations are concerned, is that in connection with the use

of a parting compound. It is generally agreed that the most important silica exposure of molders is due to other silica-generating processes often conducted in the vicinity of molding operations. It is obvious that the risk associated with molding operations can be materially lessened either by using a parting compound containing less silica or in properly safeguarding the user by adequate dust control during this operation. With such occupational histories available, and the knowledge of the percentage of total dust exposure revealed by detailed occupational analysis, it was found possible in a recent study to prophesy in 9 cases out of 10 approximately the conditions that would be found upon physical examination in cases where the total changes were due chiefly to dust inhalation.

TABLE 5.—*Dust exposure of molders*

Activity	Time of exposure in minutes (a)	Average dust exposure in millions of particles per cubic foot (b)	Millions of particle-minutes (a×b)
Use of parting compound	54	63.8	3,445
Remaining task in molding	412	4.4	1,813
Pouring	58	3.1	180
Dumping molds (shake out)	16	32.5	620
Total	540		6,058

$\frac{6{,}058 \text{ million particle-minutes}}{540} = 11$ million particles per cubic foot.

PREDISPOSING CAUSES

Geographical distribution.—Eight countries were represented at the International Conference held in Johannesburg in 1930. A recent bibliography on pneumoconiosis lists references to the literature of 26 countries.

Since the literature of practically all the principal nations of the world contains articles on this subject, it is apparent that no nationality is exempt, and that all races are susceptible is shown by the wide distribution of silicosis.

Age.—Although the data show incidence is higher among the younger miners in districts where the percentage of free silica is high, and among older miners where the percentage of silica is low, age in itself probably is no great factor.

Previous exposure.—Previous occupations of the men are reported to have a definite influence in predisposing to silicosis, if they have been exposed to dust or to other respiratory irritants.

Individual susceptibility.—The factor of individual susceptibility is often mentioned. Generally speaking, if there be any difference in individual susceptibility, it can usually be considered an acquired and not a congenital condition. Ickert (*24*) quotes Bohme and Lucanus

and Schulte-Tigge as stating that it is essential that the individual possess excellent functioning nasal passageways, in order that the self-cleansing mechanism may work efficiently. He calls attention to the fact that Irvine, Simpson, and Strachau report the "classical" type of silicosis to be more common among the robust type of individuals with less respiratory reserve. Ickert found some slight variation in the susceptibility of persons according to their type of body build. As a whole, the group classed as slender individuals developed simple silicosis somewhat slower than the stoutly built persons; but the incidence of advanced silicosis was greater in the former class, being nearly double that developing in the sturdy workers.

Lehmann's (*25*) experiments to determine the functional efficiency of the upper respiratory tract in the removal of dust, suggests that abnormalities of the nasal passageways probably play some part in the rapidity with which silicosis may develop.

It is suggested by some that chronic bronchial asthma may be considered a predisposing factor affecting individual susceptibility. The spasmodic attacks, if frequent, lead to a reduction in the individual's vital capacity. Aside from the pulmonary fibrosis, other pathological manifestations of silicosis such as bronchiectasis, emphysema, and right heart hypertrophy and dilatation, may be aggravated by this chronic condition.

Role of infection.—Since respiratory infection has been shown to be the greatest complicating factor in the development of silicosis, certainly the history of present and past respiratory infections will have to be given consideration in the statistical analysis of records upon which such conclusions are based.

Infections developing along the respiratory tract may be of importance. Sinus infections may act by decreasing the efficiency of the upper respiratory tract in the removal of dust from the air passing to the lungs, and also they may be the source of infections that spread to the lower respiratory tract. Acute pneumonic conditions as well as the more chronic lung changes such as chronic bronchitis, bronchiectasis and bronchiolectasis, emphysema, and pleurisy, all tend to decrease the ability of the lung to rid itself of foreign materials, through lessened lymphatic drainage and decreased power to force the bronchial secretions and foreign matter from the lungs.

Clinical findings and diagnosis.—The subjective and objective signs of silicosis vary according to the rate of development and degree of pulmonary fibrosis and, when infection is present, according to the type, extent, and duration of the complicating pulmonary infection.

Early in the course of the disease, signs of the condition may be absent or slight, except for the changes revealed by X-ray examination. When sufficiently developed, the most common subjective signs are shortness of breath upon exertion, cough, and chest pain. Also,

depending upon degree of development, the chief objective signs are shortness of breath, cough, prolonged expiration, decreased chest expansion, altered breath sounds and rales, impaired resonance, and characteristic radiographic appearance of the lung fields.

Diagnosis is based upon historical data, including the complete occupational history, past and present medical history, clinical, laboratory, and X-ray findings. Except by autopsy, silicosis cannot be diagnosed definitely until examination of the X-ray film reveals characteristic nodular shadows throughout both lung fields. A complete diagnosis should include, in addition to the decision that silicosis is present, a statement as to whether or not there is evidence of a complicating pulmonary infection; the type of infection if possible, and its extent; and, finally, a statement as to whether or not the individual manifests any decreased capacity for doing his usual work and an estimate as to the degree of his disability. Individuals with silicosis and active pulmonary tuberculosis are considered seriously disabled, and for the safety of themselves and others they should be barred permanently from work affording further exposure to a silica-bearing atmosphere, regardless of the manner in which the tuberculous infection may respond to treatment and rest.

As indicative of the affected individual's status under compensation laws, it has been customary to define the disease arbitrarily into stages—first, second, and third (*1*). From a medical point of view, there is no such clear line of demarcation.

First stage.—Chest film indicates early nodular fibrosis, the individual may or may not exhibit such clinical evidence as slight shortness of breath upon exertion, and some cough. The general appearance is that of a healthy individual, and there is no appreciable decrease in capacity to perform usual duties.

Second stage.—More advanced nodulation is shown by X-ray, and there may be some evidence of localized aggregation of nodules and pleural thickening. Definite shortness of breath upon exertion, cough more pronounced, chest movements sluggish, and expansion usually decreased. The individual may be able to continue at his usual job, although less effectively.

Third stage.—Fibrous changes as shown by X-ray film are further accentuated. Nodules are larger and assume conglomerate form, showing large shadows corresponding to areas of dense fibrosis. Shortness of breath is marked and distressing upon slight exertion. Cough is increased, and may be dry, but is usually productive. Chest expansion is decreased even upon forced inspiration. The individual's capacity for work is seriously and permanently impaired.

The stages just discussed would seem to indicate in a way the extent of disability or potential disability. However, those interested in silicosis are tending toward the opinion that it is best to make a

decision as to whether the individual has silicosis or not, and to determine the extent of disability, if any, due to the disease, without reference to stage.

TUBERCULOSIS AND SILICOSIS

In 1905 the Miners' Phthisis Committee of Australia, reported that gold miners there who contracted silicosis died of tuberculosis (*26*).

The increased incidence of tuberculosis among occupational groups exposed to silica has been clearly shown in every instance where this hazard exists. Britten (*27*) summarized the report of the Registrar-General of England and Wales for 1921–23, and showed the occupational mortality rate for the group of trades classed as "Dusty Trades" to be from 3 to more than 10 times as high as the rate for all occupied and retired males.

TABLE 6.—*Standardized mortality from respiratory tuberculosis in occupations with rates above average, males age 20–65, England and Wales, 1921–23*

Occupation	Mortality rate (standardized)
All occupied and retired males	149.6
Tin and copper mines, underground workers not superintending staff (III)	1,886.0
Tin and copper mines, not superintending staff (III)	1,323.5
Grinders in the cutlery tools (IV)	1,178.5
Metal grinders (IV)	636.7
Slate masons and slate workers (III)	512.5
Potters' mill workers, slip makers, potters (III)	411.4

Lanza and Vane (*5*) show by an analysis of the mortality experience of 12 life-insurance companies, for the period of 1915 to 1926, that the actual mortality from respiratory tuberculosis among the silica-exposed persons was about three times that of a non-silica-exposed group. When this comparison is limited to the rates for some of the occupations with a very great silica exposure, such as metal mining, sandstone, and granite quarries, the excess is still more striking, the rate being about 10 times that obtained in the non-silica-exposed group.

Gardner (*28*) has shown that animals exposed to silica when inoculated with a strain of tubercle bacilli of low virulence will develop systemic tuberculosis and die, while control animals not so exposed usually are not seriously affected by injections of such organisms.

PROGRESSIVE TENDENCY OF THE DISEASE

Since infection has been shown to play an important role in the advanced seriousness of silicosis, the possible relationship of this infective element to the progressive tendency of the disease cannot be overlooked.

Irvine (*29*) has stated that it is not so much what the condition of the silicotic is today, as what will it become tomorrow. Irvine

emphasizes the tendency of the fibrosis to progress, even though removed from exposure, and expresses his opinion that it is one of the most serious aspects of the whole silicosis problem. No remedy has been shown to be of value in the elimination of the pulmonary fibrosis although improvement in symptoms may be noted after the victim is removed from exposure. According to the observations made by Bohme-Bochum (*30*), silicosis progressed, after removal from exposure, in 20 percent of the cases diagnosed as having silicosis in grade 1, in 40 percent of the cases in grade 2, and in practically all cases in grade 3.

EFFECTS OF OTHER DUSTS

It seems to be the consensus of opinion that silica dust is the most harmful, and it is the principal one with which industry is concerned. However, asbestos is reported to be the one which produces a condition which is disabling and causes death.

Asbestosis is relatively a new disease in industrial workers, as asbestos has not been in general use for so many years. Asbestos is an anhydrous form of magnesium silicate. The symptoms of asbestosis are insidious in onset and irregular in the course of the disease. Dyspnea and cough are the most prominent, and as the disease progresses the cough becomes productive. Elastic fibers can be found in the sputum in advanced cases, indicating that there is destruction of the pulmonary tissue. Asbestos bodies have been found in the sputum and the finding of them is a diagnostic aid, showing that the worker has been exposed to the dust. These particles of asbestos in the sputum usually show evidence of disintegration. Asbestosis usually develops after exposure to the dust (excessive quantities) for a period of approximately 7 to 11 years (*31*). It has not been shown that asbestosis predisposes to tuberculosis as does silicosis. Death usually results from pneumonia, bronchitis, or influenza and less often from tuberculosis. When tuberculosis complicates asbestosis, the clinical picture is not the fulminating type.

The X-ray characteristics of the chests of asbestos workers differ from those of workers exposed to silica dust. The markings are less distinct and are described by some as having a "ground-glass appearance." The nodular fibrosis of silicosis is conspicuously absent.

Another form of pneumoconiosis occurs incident to exposure to magnesium silicate dust in the form of talc. This dust is a hydrous form of magnesium silicate, $H_2Mg_3(SiO_3)_4$, and is used in a number of industrial processes. The mining and milling of talc entails exposure to excessive amounts of the dust. Dreessen (*32*) reports the results of a study of exposure to talc dust. He states that a fine bilateral fibrosis of the lungs occurs, which is demonstrable by the X-ray; and that while very dusty conditions prevail in the production of talc, it cannot be said that the resultant pneumoconiosis has led to disability.

ANTHRACO-SILICOSIS

Anthraco-silicosis is the term used to describe a certain form of pneumoconiosis, commonly called miners' asthma. It is a chronic condition due to breathing dust incident to coal mining. It is characterized by general fibrotic changes of the lungs with the presence of excessive amounts of carbonaceous and siliceous materials. A compensatory emphysema occurs and often cardiac changes take place in the later stages.

The coal mining employees were grouped occupationally, largely in accordance with the proportion of free silica found in the dust to which they were exposed.

No cases of anthraco-silicosis were found in a control group composed of hard-coal mining employees whose dust exposure averaged less than 5 million particles per cubic foot of air.

The prevalence of anthraco-silicosis among the entire group of employees was found to be about 23 percent. Among men exposed 15 to 24 years to dust containing less than 5 percent free silica, 14 percent of those who had worked where the average dust count was 100 million to 199 million particles per cubic foot, 29 percent of those exposed to 200 million to 299 million particles, and 58 percent of the men who had worked for this period in more than 300 million particles per cubic foot, developed anthraco-silicosis.

Among the men employed for more than 25 years in dust containing less than 5 percent free silica, the proportion of persons found with anthraco-silicosis under different concentrations of dust was as follows: 5 million to 99 million particles, 7 percent; 100 million to 199 million particles, 54 percent; 200 million to 299 million particles, 71 percent; 300 million or more particles per cubic foot, 89 percent.

With the exception of miners, their helpers, and rock workers, about 25 percent of all the men employed underground developed anthraco-silicosis after a working period of more than 25 years. This group was exposed to dust having a quartz content of about 13 percent.

The prevalence of anthraco-silicosis among persons who had been exposed for more than 2 or 3 years to dust of which about 35 percent was free silica, varied from 10 percent among those who had worked in concentrations of less than 200 million particles per cubic foot for less than 15 years, to 92 percent among those who had been employed for more than 25 years in dust concentrations exceeding 300 million particles per cubic foot, more than 2 or 3 years of which time was spent in rock work.

Age *per se* appeared to play only a minor role in the development of anthraco-silicosis.

Analysis of the data for the purpose of determining safe limits of dust exposure indicated that employment in an atmosphere con-

taining less than 50 million dust particles per cubic foot would produce a negligible number of cases of anthraco-silicosis when the quartz content of the dust was less than 5 percent. In the gangways where the silica content of the dust was about 13 percent, a safe limit appeared to be 10 million to 15 million particles per cubic foot. The limit of toleration for rock workers was set tentatively at 5 million to 10 million particles per cubic foot of air.

Pulmonary infection increased with length of service more rapidly among the men in the haulageways than in the control group, and much more rapidly among the regular miners. The highest rates of pulmonary infection, however, were found among the rock workers of more than 15 years' service.

The prevalence of pulmonary tuberculosis among the hard-coal mining employees at ages below 35 was slightly less than that found through studies of tuberculosis among male adults in the general population of the country. In the age group 35 to 44, however, the prevalence of tuberculosis was about twice, at ages 45 to 54 about 5 times, and for the ages above 55 it was about 10 times the rate found in the general population.

The highest prevalence of clinical pulmonary tuberculosis occurred among the rock workers (men who had been employed in rock loading or rock extraction for more than 2 or 3 years). After 20 years' service, of which more than 2 or 3 years were in rock work, 37 percent presented evidence of pulmonary tuberculosis.

In a group of 135 completely disabled former anthracite workers, which did not include any known cases of tuberculosis, 10 percent proved positive for pulmonary tuberculosis.

Pulmonary infection (tuberculous and nontuberculous) was found among 58 percent of the employed men having early anthraco-silicosis, and in 92 percent of the workers in the more advanced stages.

Clinical pulmonary tuberculosis was diagnosed in 15 percent of those with early anthraco-silicosis, and in 43 percent of those in the more advanced stages.

In all groups combined, with the exception of the control group, about 20 percent of the nontuberculous workers were diagnosed as having some respiratory disease other than tuberculosis. In the control group only 6 percent had nontuberculous respiratory disease.

In the control group less than 2 percent were found with moderate or marked physical impairment causing decreased capacity for work as compared with about 10 percent among the regular miners, and with approximately 13 percent among the rock workers. With the exception of the rock workers, no group showed moderate or marked physical impairment in excess of that found among the controls when the period of employment was less than 20 years. However, an excess in the prevalence of slight impairment was found among the regular

miners and among others exposed to dust containing less than 5 percent free silica when they had worked from 10 to 20 years in atmospheres containing more than 100 million particles per cubic foot.

The correlations between exposure to dust and the evidence of constitutional changes left little doubt as to the etiological significance of the dust in the air breathed. Like correlations were found between the silica exposure and the extent of pulmonary changes.

Mortality from respiratory diseases was found to be much greater among anthracite workers than in the general adult male population of the country. The data indicated that underground work in the absence of dust did not predispose to fatal attacks of respiratory disease.

PREVENTION OF SILICOSIS

This phase of the silicosis problem may advantageously be considered under two main divisions—medical and mechanical.

Medical.—Preliminary and periodic physical and roentgenological examination should be made of all employees engaged in industrial processes where there is a potential if not actual exposure to excessive concentrations of silica in the atmosphere. Medical personnel making these examinations should be familiar with the industrial processes in which the individual is to be employed.

In fairness to the individual, his fellow workers, industry, and even the public at large, an individual with evidence of active tuberculosis or extensive healed lesions should not be employed in an atmosphere where there may be exposure to silica dust. The infected individual's condition may become aggravated by such exposure to silica dust; his fellow workers may become infected under conditions which might not be dangerous were it not for the presence of silica dust; industry may eventually bear the burden of compensation for injury to the health of the employee and possibly some of his coworkers; and last, but not least important, the prevention and control of tuberculosis among the general population will be more difficult. In the absence of infection, silicosis is a slowly progressing disease and develops at such a rate that the individual affected may not become disabled during his ordinary working life. Therefore, if infection can be lessened, the silicosis problem may be much more readily controlled.

Periodic physical examination will serve to detect early evidence of infection and abnormal pulmonary changes resulting from dust exposure, which will permit correction of the conditions causing the pulmonary fibrosis and prevent the development of disabling pulmonary disease. Unless active pulmonary infection is diagnosed, the individual in whom a beginning fibrosis or nondisabling silicosis is present may be safely continued at his employment, provided the dusty conditions causing it are eliminated. Usually the physician

familiar with the patient's condition and the occupational environment to which he is exposed, is in the best position to advise his retention or removal from the industry.

Engineering or mechanical control.—Although we have mentioned the medical phase of control first, it is of secondary importance in effectiveness compared to that which is accomplished by engineering methods. Prevention of silicosis from the viewpoint of engineering and mechanical control lies in the efficiency of such methods as may be adopted (1) to prevent the escape of siliceous dust into the workroom atmosphere, (2) efficient removal of the dust from the atmosphere, in the processes where its escape is not preventable, (3) and the use of mechanical personal protective equipment (for emergency use or in operation of such processes as are used only temporarily or for but short intervals of the working day).

Certain processes readily permit trapping of the dust at its source, others may be completely enclosed, preventing the escape of dust at any point during operation. Examples of such are the jack-hammer with dust trap attached and enclosed abrasive processes where the product being ground is passed mechanically in and out without escape of abrasive material used.

The dust in the air of some quarters may be removed by air-filtering arrangement securing a rapid exchange of clean air for dusty air. Water sprays, and other wetting-down methods serve to keep general atmospheres clean or assist in removing the offending silica.

Whenever practicable, dusty processes should be isolated from other divisions of the industry, thus subjecting as few employees as possible to a potential hazard.

Occasionally, even without any decrease in efficiency or increase in cost, materials containing less silica may be found as satisfactory substitutes.

Sometimes cleaning of work places may constitute a source of atmospheric pollution with silica dust. Vacuum cleaning and wet methods may satisfactorily solve this problem; when such is not feasible, the cleaning should be done outside working hours when there will be the least number of persons exposed, and for the time involved, those doing the cleaning may be protected by respirators or positive pressure helmets.

In the driving of tunnels and mining which may necessitate the removal of siliceous rock, blasting operations account for a considerable portion of atmospheric pollution. It has been found practicable in most instances to do this blasting after regular working hours or between shifts, allowing sufficient time to elapse for the air to clear. Water sprays released simultaneously with blasting have been used to prevent general dispersion of the dust to other working localities in the vicinity of blasting operations.

The part played by a study of workroom environment by qualified personnel is essential to successful control of a silicosis hazard. Analysis of workroom air prior to the installation of methods for keeping the atmosphere safe serves to indicate the necessity for the institution of methods for dust control and, by comparison, to determine the effectiveness of measures employed thereafter. Periodic inspection of all equipment designed for the prevention of atmospheric contamination should be carried out at intervals found to be satisfactory.

Close cooperation between the engineering and medical personnel is essential for economical and safe control of the dust hazard. No one method or equipment has proved effective and practicable, but the adoption of those most practicable and safe under capable direction may be relied upon to prevent conditions which will cause silicosis.

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STUDIES ON TRICHINOSIS

VII. The Past and Present Status of Trichinosis in the United States, and the Indicated Control Measures [1]

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INTRODUCTION

Some factors regarding trichinosis that have been clarified since the problem was taken up by the Public Health Service in the spring of 1936 are as follows:

1. Abundant evidence indicates quite clearly that trichinosis is probably a greater problem in the United States than in any other country in the world. The evidence to this effect was available about 50 years ago, but was quite generally overlooked, disregarded, or misinterpreted.

* Died May 1, 1938.

2. Human trichinosis rests primarily on a basis of swine trichinosis.

3. Swine trichinosis rests primarily on a basis of uncooked or inadequately cooked pork scraps fed to swine in garbage, table scraps, swill, and similar material.

4. The indicated control of trichinosis in the United States is primarily a matter of keeping such uncooked pork scraps out of the feed of swine, and either cooking such feed as garbage or refraining from feeding any food containing pork scraps.

5. Rats appear to have a very minor role in the production of porcine trichinosis, and at this time may be given only secondary consideration in our control measures.

6. The evidence available indicates that our too casual measures for the control of trichinosis have not lowered the incidence of trichinae or trichinosis in man in this country during the past 50 years. The incidence in swine has decreased only to the extent that the once widespread practice of feeding offal from slaughtered hogs to other hogs has decreased; and this bad practice has been almost entirely abolished purely as a result of improved sanitary conditions in general and not as a definite part of any trichinosis control program.

7. Prompt action looking towards the control of trichinosis is imperative for the protection of the public health on the one hand, and the protection and improvement of the swine industry and the packing industry on the other hand. An extension of the present move by scientists, physicians, and veterinarians to protect the public health must be anticipated by the swine growers and packers to protect their industries from the consequences which may be expected from research within the next few years.

The factors just enumerated will be discussed in the following sections.

PREVALENCE OF TRICHINOSIS

There was abundant evidence 50 years ago that the United States had an extraordinary prevalence of trichinosis, but that evidence was misread, misunderstood, disregarded, or misinterpreted for the most part, and wholly unwarranted conclusions were drawn from it. Various writers, including Mark (*1*), pointed out, in the 1880's, that

[1] Following are the preceding articles of this series:

I. The incidence of trichinosis as indicated by post-mortem examinations of 300 diaphragms. By Maurice C. Hall and B. J. Collins. Pub. Health Rep., 52: 468 (1937). (Reprint 1816.)

II. Some correlations and implications in connection with the incidence of trichinae found in 300 diaphragms. By Maurice C. Hall and B. J. Collins. Pub. Health Rep., 52: 512 (1937). (Reprint 1817.)

III. The complex clinical picture of trichinosis, and the diagnosis of the disease. By Maurice C. Hall. Pub. Health Rep., 52: 539 (1937). (Reprint 1819.)

IV. The role of the garbage-fed hog in the production of human trichinosis. By Maurice C. Hall. Pub. Health Rep., 52: 873 (1937). (Reprint 1836.)

V. The incidence of trichinosis as indicated by post-mortem examinations of 1,000 diaphragms. By M. O. Nolan and John Bozicevich. Pub. Health Rep., 53: 652 (1938). (Reprint 1929.)

VI. Epidemiological aspects of trichinosis in the United States as indicated by an examination of 1,000 diaphragms for trichinae. By Maurice C. Hall. Pub. Health Rep., 53: 1086–1105 (July 1, 1938).

nowhere in the world did swine show such amazing incidences of trichinae, up to 13 and 18 percent, as in this country. Billings (*2*) stated that there was an enormously greater percentage in this country than in Germany, if the German statistics were reliable, and the subsequent developments of the past 50 years show that these statistics were reliable. Billings (1880) and Williams (1901) cite a number of European necropsy findings over the period of 35 years from 1852 to 1887, covering the findings of Delpech, Fiedler, Gribbohm, Leuckart, Mueller, Roth, Rudnew, Scheiber, Sivers, Turner, Wagner and Zenker. An incidence of less than 1 percent was found in Switzerland, Roumania, France, and at Erlangen and Kiel in Germany; of 1 to 2 percent in Scotland and Russia; of 2 to 3 percent at Dresden and Berlin in Germany; of 2.5 to 6 percent at Leipzig in Germany. Where specific figures are cited, there is a total of 8,551 necropsy cases with 34 positive cases, or an incidence of about 0.4 percent.

These figures may be contrasted with the 5 to 14.3 percent reported by Whelpley, Thornbury, and Williams from 1891 to 1901, the 27.6 percent reported by Queen in 1931 at Boston, and the 24 percent found by McNaught and Anderson in 1936 at San Francisco, as summarized by Hall and Collins (*3*), and with our unpublished findings of approximately 17 percent in a large series of cases from many cities in the United States. The lowest of the recent figures for the United States, 3.5 percent at New Orleans, as ascertained by Hinman in 1936, is higher than all but one of the European figures cited. Nevertheless, 50 years ago competent authorities concluded from just such figures as are cited here that trichinosis was a minor public health problem in the United States, and this fiction, once established, has maintained itself in the minds of scientists and medical men for a half century. It can now be definitely branded as fiction, with a notation to the effect that there was an export trade involved, and that the literature shows that a desire to make facts fit the needs of that export trade played a role in the minds of observers who did not lack the mental qualifications to draw sound conclusions from adequate data, provided they had an unprejudiced status in the matter. There is no chance that scientists and the medical and veterinary professions will again fail to carry out their mission in the protection of the public health. We have wasted a half century, at what cost in health and life one hesitates to consider, but now we believe that the more enlightened livestock and packing industries will aid in removing the handicap of trichinosis from their industries and will not ask that we perpetuate the disease yet another half century.

RELATIONSHIP OF TRICHINOSIS TO MEATS OTHER THAN PORK

Such rare items as human trichinosis originating from eating bear meat, of which we have several cases, or dog meat, of which at least one case is on record, or beef, which was the apparent source of a few cases in two or three instances, may be ignored as not warranting consideration in a control program at this time. Our concern is with pork, the customary source of human trichinosis.

CAUSE OF TRICHINOSIS IN SWINE

The evidence to the effect that swine trichinosis results from pork scraps fed to swine, in the vast majority of cases, is overwhelming. The high incidence of trichinae found in garbage-fed hogs and hog-lot hogs fed on table scraps and swill, and the very low incidence or entire absence of trichinae in hogs raised on pasture or in the fields and woods, or even in knackers' yards when fed on cooked meat and corn, in the presence of rats quite generally infested with trichinae, was shown by many workers a half century ago and has recently been verified. This statement could be documented by numerous references, and an adequate sample of such references has been published by Hall (*4*). The prevalence of pork scraps in our American garbage is well established, and follows from an economy of abundance that permits of discarding to the garbage pail meat unsatisfactory from the standpoint of health or esthetic considerations, plus a certain amount of waste.

ALTERNATIVE CONTROL MEASURES

If we inspect the more obvious possibilities as to lines of action we might pursue at this time, they appear to be as follows: (*a*) To go on as we have done for 50 years, stating that trichinosis is a rare disease of man and a minor public health problem so far as the United States is concerned, trusting to casual control measures which statistics show to have accomplished nothing in lowering the incidence of porcine or human trichinosis, and leaving it to the consumer of pork to be guided by the policy of "Caveat emptor;" (*b*) sound a general alarm, warning the public that trichinosis is rife and pork dangerous, a policy which will neither control trichinosis nor help the public, the swine grower, or the packer; (*c*) demand restrictive legislation which will be to a large extent ill-considered, cumbersome, and ineffective; (*d*) inaugurate microscopic inspection of pork at a cost of millions of dollars annually, and with the probability that it will materially interfere with the speed and efficiency of packing-house procedures; (*e*) demand refrigeration of all pork to destroy trichinae, at enormous cost; or (*f*) lay out a campaign calling for the cooperation of the swine grower

and the packer in solving the problem of trichinosis, with the task of keeping pork scraps out of the feed of swine as the indicated solution of the problem. If we inspect these possibilities, the obvious objections to most of them are sufficiently evident.

(*a*) We should not pursue a policy which has failed for half a century to lower the incidence of trichinosis, nor should we continue to sell to the trusting public food that may cause serious disease or death when that food is used by a vast majority who cannot protect themselves.

(*b*) Our objective is control of trichinosis, not public scares and damage to the swine and packing industries by pointless publicity. What is needed is constructive action, not words.

(*c*) The control of disease in such cases may follow the lines of action by an industry to improve conditions in the industry, or the education of the public as to self-protective measures, or governmental action in the way of restrictive legislation and the exercise of the police power. Of these possibilities, improvement within the industry is the one which industry desires, as a rule, and is the most rapid and effective when it can be invoked. Education has been a failure, so far as trichinosis is concerned, and our statistical evidence, showing trichinae present in approximately 17 percent of a large necropsy series, indicates that a large proportion of the American public eats raw or undercooked pork, and expects to have the right to eat pork raw or undercooked, if it has a taste for it, just as it eats beef raw or undercooked, and to do so without incurring the penalty of trichinosis and possible death. It appears to be a legitimate demand that when a man exchanges dollars for pork, he should not do it on the basis that he may be purchasing his death warrant, and that in a day when foods of all sorts are assumed to be, and quite generally are, safe and wholesome as produced and marketed, no one has a right to demand that an exception be made for pork in order that the custom of feeding raw pork scraps to swine may be preserved. The Court of Appeals of New York State (*McSpedon* v. *Kunz, et al.*, 288 N. Y. S.; 271 N. Y. 131, 2 N. E. (2nd) 513) upheld this position in 1936 in sustaining the law of implied warranty as applied to pork, regardless of whether the pork is of a sort customarily cooked by the consumer or not, in a damage suit for trichinosis acquired from eating undercooked pork chops. As parasitologists, medical men, lawyers, or citizens we must admit that meat is diseased either because of a content of pathologic tissue or because of a capacity for transmitting disease. Trichinous pork is diseased meat because the parasites and inflammatory, calcified, or necrotic reaction tissues in it are not normal constituents of meat, and especially because it is capable of transmitting a serious and sometimes fatal disease.

(*d*) The meat-inspection statistics of Germany give convincing evidence that microscopic inspection of pork leads in the direction of eradication of both human and porcine trichinosis in Germany, since the incidences of both have fallen to exceedingly low figures during the past half century. That inspection has fitted into the more leisurely slaughter-house procedures of Germany, and although it now costs many thousands of dollars to find one trichinous pig, that alone is evidence that it is accomplishing its mission in the control of trichinosis. Under the high-speed procedure of American packing plants, microscopic inspection for trichinae would require the training and use of a regiment of inspectors at a cost exceeding our present total cost for meat inspection. It would be especially expensive and difficult if anything approximating the present speed of plants were maintained, and in all probability it would slow down those procedures. If unaccompanied by changes in our methods of swine production, it would mean enormous expenditures for perhaps 50 or 100 years. Unless it were Nation-wide, which would call for legislation by all the States, it would unquestionably be dangerous, since its application by the Federal Government to interstate shipments alone would give a false sense of security in the consumption of a pork supply that was a mixture of inspected and uninspected pork as marketed and served. So long as there is a simple and inexpensive control measure in sight, this alternative may be regarded as one of those last painful measures which should be taken only under stress of necessity.

(*e*) The refrigeration of all pork to destroy trichinae, involving the refrigeration of pork for 20 days at a temperature not exceeding 5° F., would involve enormous costs in storage space and refrigerating equipment. Unless it were applied to all pork, and not just to pork in interstate shipments, it would involve the same dangers that microscopic inspection of the same limited sort would involve. At the moment, it may be set aside on the same grounds as those applying to microscopic inspection.

(*f*) The logical solution of our problem is to have a control campaign so planned that medical men may, in a cooperative way, eliminate the hazard of human trichinosis, and assist the swine grower and packer to remove the stigma of trichinosis from their products. It is unnecessary to feed raw pork scraps to swine. Garbage feeders can cook garbage. In spite of all objections or statements to the contrary, it can be done and is done in many places, and it is compulsory in Canada for all garbage not originating on the premises where it is fed, and in England. Whether every detail of cost, feed values, and other items meets with the entire approval of everyone concerned or interested is a minor matter compared with the public health as

affected by garbage-fed hogs, of which about 5 percent are trichinous. The farmer can feed his table scraps to his poultry, or cook them, but uncooked pork scraps must be kept out of feed for swine.

The swine grower who follows the Bureau of Animal Industry system of swine sanitation, a procedure which the writer pointed out years ago as our best control measure for trichinosis, has a legitimate complaint against the swine grower or feeder who maintains a filthy hog lot or garbage-feeding plant to advertise to the world that, in spite of the general prevalence of modern business methods of producing and marketing food under clean and sanitary surroundings, there are some swine growers who produce swine, and consequently pork, under filthy conditions. This procedure does the swine industry and the packer an injustice and a disservice, and they should be the first to resent it. As part of a control campaign, it should be the business of the progressive swine grower to educate his less progressive associates, and of the packer to require that the product he buys be raised under such conditions as will give assurance that the packer will not be selling pork that may cause the illness or death of the innocent purchaser, and that will not involve the packer in damage suits for this loss of health or life. By virtue of his position as the purchaser of the swine growers' product, the packer is in a position to require that what he buys meets certain reasonable specifications, and to refuse to buy swine that do not meet those specifications. By virtue of his extensive and well organized forces, and his long and intimate association with the swine growers and dealers, he can carry out such a program. It is to his best interest to do this. The big packing houses have accomplished much in the way of disease control, and will undoubtedly support a program of trichinosis control.

RELATIONSHIP OF THE RAT TO TRICHINOSIS IN SWINE

A large amount of evidence shows that, in spite of very high incidences of trichinae among rats, the rat ordinarily acquires trichinosis from eating other rats or eating pork scraps, but that his role as a donor of trichinosis to swine is a very minor one. The great majority of swine growers testify that swine usually do not eat rats even when these rats are numerous in hog pens or around garbage-feeding plants; and the fact that a few persons have seen an occasional rat eaten by swine indicates that this happens but seldom, perhaps when swine are on a feed deficient in protein, are underfed, or are for some reason compelled to eat rats or are in some way conditioned to eating rats, but that this happens too rarely to be an important factor in causing porcine trichinosis. While rat destruction is always advisable for many reasons, aside from a consideration of trichinosis, rats must be regarded as a minor matter in connection with human or porcine

trichinosis, and can be given only incidental consideration in this connection. Evidence on this point is cited by Hall (*4*).

INEFFECTIVENESS OF PREVIOUS CONTROL MEASURES

From the summarized data published by Hall and Collins (*3*), we note that necropsy studies during the past 45 years show the following incidences for trichinae: Whelpley, 1891, St. Louis, Mo., 5 percent; Thornbury, 1897, Buffalo, N. Y., 14.3 percent; Williams, 1901, Buffalo, N. Y., 5.35 percent; Queen, 1931, Rochester, N. Y., 17.15 percent, and Boston, Mass., 27.6 percent; Riley and Scheifley, 1934, Minneapolis, Minn., 17.09 percent; Hinman, 1936, New Orleans, 3.5 percent; McNaught and Anderson, 1936, San Francisco, 24 percent; Hall and Collins, 1937, Washington, D. C., and Baltimore, Md., 13.67 percent. Nolan and Bozicevich (*5*) have recently reported an incidence of 17.4 percent for 1,000 necropsies at Washington, D. C., and 5 other cities in the East. It is quite evident that these figures show no decline in the incidence in man.

As regards the incidence of trichinae in swine, it appears that the extraordinary incidences, up to 18 percent, which were associated with the feeding to swine of offal from slaughtered swine, have disappeared with the sharp decline in the feeding of offal. Incidences of this sort are cited by Hall (*4*), and are tabulated in table 1. On the other hand, our best indicator for comparison, the incidence in various lots of so-called grain-fed hogs of the Middle West (table 1), shows no perceptible decline when one considers that the earlier figures for these grain-fed hogs included some hogs to which offal was fed. Apparently these hogs have a trichina incidence of about 1.5 percent.

A consideration of the figures for trichinae in man and swine over the past 50 years shows that what we have done to control trichinosis during that time has entirely failed to control it. We have depended on two measures: Warnings to the public to cook pork well, warnings which the public quite generally disregards, as shown by the presence of trichinae in approximately one out of every six necropsy cases in what is now a large series, and evidently a warning which the public will probably continue to disregard; Federal meat inspection regulations requiring that all pork in products of a sort customarily eaten without cooking by the consumer must be so processed as to kill any trichinae that may be present. Of itself, this is a sound procedure, soundly carried out, but since the Federal government inspects only about 70 percent of the meat in the country, and since there is very little inspection of an adequate sort outside of Federal inspection, the shuffle of inspected and processed products with uninspected and unprocessed products, as they appear on the market and in hotels,

restaurants, and elsewhere, results in a failure to protect a public which, as a rule, eats both products indiscriminately. There is no microscopic inspection of pork in this country, and no processing of fresh pork or of pork products customarily eaten after cooking by the consumer.

TABLE 1.—*Incidences of trichinae in swine in the United States*

Author	Date	Place	Percent infested
	Offal-fed swine		
Harding and Robbins	Ante 1884	Dearborn Co., Ind	6.5–16.3.
Mark	1889	Boston, Mass	17.95.
Calvin	1890	Iowa	8.33–10.0.
	Garbage-fed swine		
Hall	1935	Several cities	4.8.
Schwartz	1936	do	5.0 (approximate).
	Grain-fed swine		
Detmers	1883	Chicago, Ill	2.4.
Atwood and Belfield	1886	do	2.0.
Mark	1889	do	2.0 (500 swine).
Bureau of Animal Industry (8,000,000).	1898-1906	In 10 States	1.41 (live).
Hall	1935	Several cities	1.5 (live).
Schwartz	1936	do	1.0 (approximate) (live).
	Southern swine		
Deverson	1881	South	0 (4,146 swine).
		Louisville, Ky	0.83 (241 swine).
		New Orleans, La	0.4 (5,400 swine).
Simpson	Ante 1884	Atlanta, Ga	0 (30 swine).
Steger	do	Nashville, Tenn	0 (180 swine).
Meyers	do	San Antonio, Tex	0.6 (330 swine).
Unpublished	Recent	Georgia and Louisiana	Less than 1.
	Pasture-raised swine		
Calvin	1890	Iowa	0 (300 swine).

PREVENTION OF TRICHINOSIS IN SWINE

At the present time research on trichinosis is being carried on at many places in the United States, and one can predict with some certainty some of the results that will come from these investigations. Necropsy studies will undoubtedly make it certain that the incidence of trichinae in man in this country is very high, probably about 17 percent. Further study of the complex picture of trichinosis will serve to differentiate this disease from the 50 clinical entities with which Hall (*6*) has found it confused. Improvements in diagnostic methods already being made will aid in diagnosis and, simultaneously,

furnish a method by which incidence may be more readily and extensively ascertained. As a result, we shall probably detect thousands of clinical cases annually instead of a few hundred. When this happens, there will be definite and unpleasant repercussions in the market for swine and pork unless effective control measures are carried out before the storm breaks.

The indicated measures for the control of trichinosis are such procedures as will keep raw and undercooked pork scraps out of the feed of swine. This will be most effectively accomplished by the raising of swine on pasture, preferably under the swine sanitation system developed by the Federal Bureau of Animal Industry. Where garbage is fed, it must be required by law that it be cooked to kill any trichinae in pork scraps that may be present. The dirty hog lot should be abolished for sanitary, business, and esthetic reasons. In brief, it will devolve on the swine grower to control trichinosis by abandoning the dangerous and unnecessary practice of feeding raw and undercooked pork scraps to swine. The majority of swine breeders do us the service of breeding swine; a small minority do us the unconscious disservice of breeding trichinosis.

Since the swine industry is not organized on a nation-wide scale, and hence cannot be approached through such an organization, it devolves upon the packers, who do have such an organization, to require of the swine grower that he raise swine under conditions which protect the packer in purchasing swine and in marketing pork. As already noted, the highest court in New York State has held that the law of implied warranty applies to pork, even though it be pork of a sort customarily cooked by the consumer before being eaten, and that the responsibility in suits for damages, in cases in which trichinosis has followed the eating of pork, may be passed to the packer. So far as an incomplete survey of the literature shows, New York State has more recorded cases of clinical trichinosis than any other State in the Union. Ransom's (7) figure of 355 cases up to 1915, plus the available figures from the reports of the New York State Department of Health from 1928 through 1935, or 687 cases, total 1,042 recorded cases, and since it was made a reportable disease in 1930, the figures have risen from 21 cases in 1932 to 243 cases in 1935. A general agreement on this point of law by courts in the United States, at some future time when large numbers of cases of clinical trichinosis were being diagnosed, would make the selling of pork a hazardous occupation for the business man. As a foreseen possibility, this condition should be anticipated by the packer, and action taken to forestall unpleasant possibilities. The efficient and far-flung organization of the packers is competent to undertake the task of buying swine that are free from trichinae, and refusing to buy swine that are likely to have trichinae, or assessing the added cost of processing pork from

such swine against the producer by a correspondingly lower purchase price.

As regards the safer groups of swine, there are in the Middle-West and in some other regions large numbers of pasture-raised swine (table 1) that are either entirely free from trichinae or that certainly have an extremely low incidence of trichinae, and these may be purchased with almost complete safety so far as the likelihood of danger from trichinosis is concerned. In the South, there are large numbers of swine (table 1) that run at large in the woods and fields and are fed only when they are finally fattened for market, being fed on peanuts or other crops, and of these swine definitely less than 1 percent have trichinae. Unpublished studies coming to our attention indicate that the more recently ascertained incidence in Southern swine still lies in the range of some fraction of 1 percent. In addition to these groups, there are the swine raised on cooked garbage, a group that may be regarded as safe provided the garbage is adequately cooked.

As regards the more dangerous groups of swine, those fed on uncooked garbage (table 1) are definitely dangerous from the standpoint of trichinosis, the incidence being about 5 percent, and such animals, if purchased at all, should be purchased only with a view to adequate processing of the pork to destroy trichinae that may be present. The other dangerous group consists of the hog-lot swine, usually in lots of one animal or a few animals, fed on garbage, table scraps, slops, swill, or other material containing raw or uncooked pork scraps. If such swine are purchased at all, the pork from them should be processed to kill trichinae that may be present.

The foregoing considers only the safety of the swine supply immediately available. Selective buying would undoubtedly start improvements to bring the more dangerous elements into line with the safer elements, and an educational campaign would take point from the threat of lost markets against those raising unsafe and, consequently, unmarketable swine. As a result, we could look forward to an extension of the use of the swine sanitation system and a greater use of pastures, the general practice of cooking garbage, and the abolition of the dirty hog lot. When we have eliminated raw and inadequately cooked pork scraps from the feed of swine, we can turn our attention to the rat as a minor matter, ascertain his role, and develop any necessary control measures. The use of the swine sanitation system is an adequate control measure already available.

ORGANIZATION OF A CONTROL PROGRAM

No control program of value can be couched in glittering generalities. It must meet the specifications of a science of control in providing for the following elements: (1) A centralized organization to

provide plans and coordination, and to receive and make reports as a basis for determining where the work makes progress and where it fails; (2) adequate authority to carry out plans; (3) adequate personnel and funds; (4) well-developed and coordinated plans of objectives to be attained, with latitude for individual judgment as to means of attaining them.

(1) Since the control of human trichinosis depends on the control of porcine trichinosis, an interstate problem, it is essential that there be a national control program for the United States, in which the U. S. Public Health Service, as the Federal agency concerned with human trichinosis, and the Federal Bureau of Animal Industry, concerned with porcine trichinosis, could cooperate jointly in assisting with coordination of the work of State health departments and State Livestock Sanitary Boards, respectively.

(2) Authority for action would be that already vested in State, county, and city officials in the control of matters affecting public health and health of livestock, or authority which might be requested from State legislatures and obtained to meet specific needs. The nature of the program would be such that little authority would need to be vested in Federal agencies. For the most part, existing authority is derived from specific laws in regard to health of man and animals, and the general laws in regard to nuisances.

(3) The available personnel and funds include those of State, county, and city health departments and livestock sanitary offices. The program of action falls in large part within the scope of customary and authorized activities.

(4) A control plan includes the simple steps of locating all garbage plants and piggeries where raw or undercooked pork scraps are fed in garbage, swill, slops, table scraps, etc., these surveys using the county as a unit basis, reporting these to State authorities, and taking whatever steps are indicated to eliminate the practice. Measures to educate those concerned may result in individual agreements to discontinue the feeding of garbage and table scraps, or to cook thoroughly any such garbage or table scraps. The buyers and packers can exert considerable influence to this end. Where necessary, the cooking of garbage or table scraps should be provided for legally as something essential in the control of animal diseases, such as hog cholera, foot-and-mouth disease, and porcine trichinosis, as well as to prevent human trichinosis. In this connection some system of licensing may be found desirable in order that more adequate control may be exercised over plants feeding cooked garbage. Licenses could be granted to such existing plants as represent a considerable monetary investment but should be refused for any new ventures of this sort. Most individuals who feed restaurant or hotel garbage on a small scale

usually have little financial stake involved and, if forced by law or regulation to discontinue the practice, would suffer little or no financial loss. The possibilities in the development of methods of dry cooking for garbage, and of having cities cook the garbage supplied to garbage feeders, should be given consideration as a possible substitute for present methods of garbage cooking by feeders.

From time to time, reports of the total number of garbage plants and piggeries and of the number in which the practice of feeding raw or undercooked pork scraps has been abolished, should be made by counties and cities to State boards of health and State livestock sanitary boards, and their consolidated reports forwarded to the Federal agencies interested. By virtue of the fact that the great bulk of the pig crop is slaughtered annually, any improvements in swine practice which may be put into effect in any year will show results the following year. If 5 or 50 percent of the danger areas were cleaned up in any one year, the incidence of swine trichinosis and clinical cases of human trichinosis the following year would reflect the improvement. Potentially, trichinosis could be practically eradicated within 5 years, and this could be done at very little expense. Even with the available personnel, funds, and authority, it should be possible to effect so much improvement in the next 5 years that our predicted increase of diagnosed clinical cases to thousands annually would not occur. Reports of progress showing the present status and any improvements from this time on would soon show definitely what might be expected from the present available forces. The solution of the problem is simple and inexpensive. It requires individual work on the individual cases involved, not regulations passed and unenforced, or educational matter broadcast at random.

If the packing plants operating under Federal inspection were to adopt the practice of processing all pork from hogs fed on garbage, in order to destroy any trichinae present, making the legitimate deductions for added costs from the price of such hogs, the immediate effect would be to drive such animals to the smaller packing plants and slaughtering establishments not operating under Federal inspection. In general, these plants either have no inspection or have a highly inadequate inspection by nonprofessional, untrained inspectors. Steps should be taken through the joint efforts of health departments and livestock sanitary boards to establish nation-wide meat inspection by professional, trained personnel along lines equivalent to the standard maintained by the Federal Bureau of Animal Industry. This development is long overdue. It is unfortunate that such a measure, so important in the protection of public health as is meat inspection, has not been applied to about a third of the meat used in this country.

The elimination of pork scraps from swine feed would result in the control of trichinosis, a marked decrease in hog cholera, and an increased safety from foot-and-mouth disease and other diseases. Nation-wide meat inspection would protect the public against diseased meat and diseases and parasites transmitted in meat, to an extent almost 50 percent greater than it is protected at present. However, such meat inspection must be sound inspection by trained personnel, and not the farce of so-called inspection by untrained appointees. Now is an opportune time to further the national welfare by a distinct forward step in the protection of the public health.

SUMMARY

Our analysis of the trichinosis problem in the United States indicates that human trichinosis rests primarily on a basis of swine trichinosis, and swine trichinosis rests primarily on a basis of raw or inadequately cooked pork scraps in garbage, table scraps, swill, etc., the rat having only a minor and not definitely ascertained role in the production of swine trichinosis.

The United States has the greatest problem of trichinosis in the world, a fact that follows from our high level of prosperity, with a resultant garbage pail rich in pork scraps, which are fed to swine in garbage-feeding plants and in small hog lots. The basic solution of the problem is keeping raw and inadequately cooked pork scraps out of the feed of swine. The control measures invoked in the past 50 years have not visibly lowered the incidence of trichinae in man and swine, and a decrease from such figures as 13 to 18 percent infestation in swine in former years follows from a sharp decline in the feeding of offal, which has resulted from a rising level in sanitation around slaughter houses for reasons other than the control of trichinosis.

The control of trichinosis calls for improvements in methods of raising swine, and these improvements should be specified by the packers as requisites to be met by swine growers before swine can be purchased for marketing as pork and without the need for specific measures to destroy trichinae that may be present in the pork. If the packers and swine growers will take their share of the task of eliminating the dangerous practice of feeding raw and inadequately cooked pork scraps to swine, they can play a large role in controlling trichinosis, eliminating that hazard to the public health, and protecting the swine industry and the packing industry from repercussions that may be expected in the next few years as a result of increased interest in trichinosis, the establishment of a high incidence figure on a wider basis, the development of better diagnostic methods, the detection of many more clinical cases of the disease, and a consequent

publicity that cannot fail to affect unfavorably the market for swine and pork. Useless publicity and ill-considered legislation are not the correct control measures for trichinosis, and at this time the packer and the swine grower should be called in as cooperators in the control of trichinosis.

A planned and directed campaign, coordinated by the United States Public Health Service and the Federal Bureau of Animal Industry, should be carried out by State, county, and city public health and livestock sanitary officers to eliminate the practice of feeding raw and undercooked pork scraps to swine, and to establish Nation-wide meat inspection by professional, trained personnel.

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DEATHS DURING WEEK ENDED JULY 30, 1938

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

	Week ended July 30, 1938	Corresponding week, 1937
Data from 88 large cities of the United States:		
Total deaths	7,017	[1] 7,218
Average for 3 prior years	[1] 7,502	
Total deaths, first 30 weeks of year	251,221	274,540
Deaths under 1 year of age	551	[1] 489
Average for 3 prior years	[1] 534	
Deaths under 1 year of age, first 30 weeks of year	15,960	17,376
Data from industrial insurance companies:		
Policies in force	69,014,251	70,091,298
Number of death claims	12,118	13,217
Death claims per 1,000 policies in force, annual rate	9.2	9.8
Death claims per 1,000 policies, first 30 weeks of year, annual rate	9.5	10.4

[1] Data for 86 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 August 6, 1938, rates per 100,000 population (annual basis), and comparison with corresponding week of 1937 and 5-year median

Division and State	Diphtheria				Influenza				Measles			
	Aug. 6, 1938, rate	Aug. 6, 1938, cases	Aug. 7, 1937, cases	1933–37 median	Aug. 6, 1938, rate	Aug. 6, 1938, cases	Aug. 7, 1937, cases	1933–37 median	Aug. 6, 1938, rate	Aug. 6, 1938, cases	Aug. 7, 1937, cases	1933–37 median
NEW ENGLAND												
Maine	12	2	0	1	12	2	------	------	152	25	------	3
New Hampshire	0	0	0	0	------	------	------	------	10	1	8	8
Vermont	0	0	0	0	------	------	------	------	177	13	------	7
Massachusetts	0	0	6	9	------	------	------	------	95	81	36	65
Rhode Island	8	1	0	0	------	------	------	------	31	4	1	7
Connecticut	3	1	8	2	9	3	------	------	39	13	18	25
MIDDLE ATLANTIC												
New York	6	15	20	24	[1] 1	[1] 1	[1] 3	[1] 1	130	323	151	186
New Jersey	11	9	2	5	1	1	------	1	30	25	73	73
Pennsylvania	11	21	17	18	------	------	------	------	60	117	257	195
EAST NORTH CENTRAL												
Ohio	5	6	6	15	------	------	1	3	108	139	77	77
Indiana	9	6	2	11	------	------	------	14	2	1	33	13
Illinois	12	18	15	20	2	3	1	5	17	25	80	80
Michigan [2]	13	12	7	7	------	------	------	------	169	157	68	47
Wisconsin	7	4	3	2	18	10	28	21	312	175	38	42
WEST NORTH CENTRAL												
Minnesota	4	2	5	3	------	------	1	1	81	41	4	18
Iowa [3]	6	3	2	2	2	1	1	------	43	21	6	6
Missouri	16	12	7	7	30	22	25	18	18	14	1	13
North Dakota	7	1	3	3	7	1	2	2	89	12	------	3
South Dakota	0	0	0	1	------	------	------	------	------	------	------	------
Nebraska	4	1	0	2	19	5	------	------	31	8	2	2
Kansas	6	2	1	4	3	1	------	------	22	8	7	7
SOUTH ATLANTIC												
Delaware	0	0	0	0	------	------	------	------	------	------	------	------
Maryland [2 3]	12	4	4	3	6	2	------	1	40	13	13	16
Dist. of Col.	33	4	3	5	------	------	------	------	42	5	3	3
Virginia [3]	29	15	17	12	------	------	------	------	89	46	13	21
West Virginia	8	3	3	5	34	12	14	14	17	6	24	24
North Carolina [3 4]	40	27	8	14	3	2	------	------	149	100	32	32
South Carolina [4]	25	9	8	4	175	63	42	45	25	9	34	15
Georgia [4]	30	18	17	11	------	------	------	------	------	------	------	------
Florida	6	2	2	7		1	------	------	------	------	8	8

See footnotes at end of table.

Cases of certain diseases reported by telegraph by State health officers for the week ended August 6, 1938, rates per 100,000 population (annual basis), and comparison with corresponding week of 1937 and 5-year median—Continued

Division and State	Diphtheria				Influenza				Measles			
	Aug. 6, 1938, rate	Aug. 6, 1938, cases	Aug. 7, 1937, cases	1933–37 median	Aug. 6, 1938, rate	Aug. 6, 1938, cases	Aug. 7, 1937, cases	1933–37 median	Aug. 6, 1938, rate	Aug. 6, 1938, cases	Aug. 7, 1937, cases	1933–37 median
EAST SOUTH CENTRAL												
Kentucky	7	4	3	3	5	3	1	-------	9	5	21	21
Tennessee [3] [4]	5	3	7	7	11	6	8	6	13	7	7	13
Alabama [4]	34	19	11	13	32	18	5	3	22	12	1	3
Mississippi [3]	28	11	9	9	-------	-------	-------	-------	-------	-------	-------	-------
WEST SOUTH CENTRAL												
Arkansas	25	10	5	5	61	24	4	4	46	18	2	2
Louisiana [4]	20	8	9	9	17	7	10	10	2	1	2	4
Oklahoma	6	3	5	6	59	29	1	5	12	6	6	3
Texas [4]	22	26	31	31	63	74	53	24	3	4	36	36
MOUNTAIN												
Montana [3]	0	0	1	1	-------	-------	-------	-------	348	36	3	3
Idaho [3]	32	3	0	0	42	4	3	1	53	5	4	4
Wyoming [3]	0	0	1	0	-------	-------	-------	-------	67	3	2	3
Colorado	68	14	6	4	-------	-------	-------	-------	63	13	12	12
New Mexico	25	2	5	3	-------	-------	-------	-------	25	2	21	10
Arizona	63	5	0	1	127	10	11	1	114	9	1	1
Utah [3]	0	0	0	0	10	1	-------	-------	352	35	4	4
PACIFIC												
Washington	13	4	1	1	-------	-------	-------	-------	35	11	16	19
Oregon	5	1	1	0	46	9	8	7	76	15	7	7
California	14	16	21	21	8	10	7	9	159	188	21	91
Total	13	327	282	348	16	326	229	248	72	1,752	1,153	1,355
31 weeks	18	13,737	13,093	17,317	73	45,372	274,029	141,283	1,003	758,270	239,151	310,034

Division and State	Meningitis, meningococcus				Poliomyelitis				Scarlet fever			
	Aug. 6, 1938, rate	Aug. 6, 1938, cases	Aug. 7, 1937, cases	1933–37 median	Aug. 6, 1938, rate	Aug. 6, 1938, cases	Aug. 7, 1937, cases	1933–37 median	Aug. 6, 1938, rate	Aug. 6, 1938, cases	Aug. 7, 1937, cases	1933–37 median
NEW ENGLAND												
Maine	0	0	0	0	0	0	13	2	61	10	2	7
New Hampshire	0	0	0	0	0	0	0	0	20	2	1	2
Vermont	0	0	0	0	14	1	0	0	41	3	0	2
Massachusetts	1.2	1	3	3	0	0	12	12	37	31	38	51
Rhode Island	0	0	0	1	8	1	1	1	15	2	2	2
Connecticut	0	0	0	0	3	1	3	1	18	6	13	9
MIDDLE ATLANTIC												
New York	2.8	7	9	9	4	9	17	17	29	71	71	97
New Jersey	0	0	0	2	2.4	2	3	3	23	19	12	23
Pennsylvania	1.0	2	10	4	0.5	1	11	4	54	106	126	106
EAST NORTH CENTRAL												
Ohio	0	0	3	3	5	7	38	3	81	104	35	59
Indiana	1.5	1	2	2	1.5	1	7	1	27	18	24	15
Illinois	1.3	2	7	7	3	5	28	10	54	82	91	91
Michigan [3]	2.2	2	1	1	1.1	1	14	8	65	60	89	60
Wisconsin	0	0	1	0	0	0	6	0	91	51	48	48

See footnotes at end of table.

Cases of certain diseases reported by telegraph by State health officers for the week ended August 6, 1938, rates per 100,000 population (annual basis), and comparison with corresponding week of 1937 and 5-year median—Continued

Division and State	Meningitis, meningococcus				Poliomyelitis				Scarlet fever			
	Aug. 6, 1938, rate	Aug. 6, 1938, cases	Aug. 7, 1937, cases	1933–37 median	Aug. 6, 1938, rate	Aug. 6, 1938, cases	Aug. 7, 1937, cases	1933–37 median	Aug. 6, 1938, rate	Aug. 6, 1938, cases	Aug. 7, 1937, cases	1933–37 median
WEST NORTH CENTRAL												
Minnesota	0	0	0	1	4	2	9	4	49	25	25	25
Iowa [3]	0	0	0	1	4	2	4	0	37	18	26	14
Missouri	0	0	1	1	1.3	1	16	2	35	27	15	16
North Dakota	0	0	0	0	7	1	0	0	59	8	5	4
South Dakota	0	0	1	0	8	1	1	1	60	8	5	4
Nebraska	0	0	0	0	0	0	7	0	15	4	2	4
Kansas	0	0	1	0	6	2	13	2	67	24	23	23
SOUTH ATLANTIC												
Delaware	0	0	0	0	0	0	0	0	0	0	8	0
Maryland [2 3]	0	0	0	0	6	2	3	3	19	6	5	13
Dist. of Col.	0	0	2	1	17	2	0	0	8	1	1	3
Virginia [3]	4	2	1	1	4	2	4	3	17	9	8	13
West Virginia	0	0	0	1	2.8	1	12	1	25	9	18	18
North Carolina [3 4]	3	2	2	2	3	2	4	3	13	9	25	20
South Carolina [4]	6	2	0	0	0	0	1	1	19	7	11	2
Georgia [4]	0	0	3	1	3	2	6	1	19	11	7	7
Florida	6	2	0	0	3	1	0	0	12	4	2	2
EAST SOUTH CENTRAL												
Kentucky	7	4	4	1	0	0	9	4	23	13	20	16
Tennessee [3 4]	0	0	1	1	4	2	3	10	20	11	8	16
Alabama [4]	4	2	3	0	1.8	1	3	3	11	6	11	10
Mississippi [3]	0	0	0	0	10	4	8	1	21	8	4	5
WEST SOUTH CENTRAL												
Arkansas	0	0	0	0	5	2	21	0	8	3	3	3
Louisiana [4]	0	0	1	1	0	0	7	2	12	5	5	7
Oklahoma	0	0	2	0	0	0	30	0	12	6	7	7
Texas [4]	1.7	2	5	1	3	4	58	3	30	35	31	25
MOUNTAIN												
Montana [3]	10	1	0	0	10	1	2	1	77	8	7	3
Idaho [3]	0	0	1	0	21	2	0	0	32	3	6	2
Wyoming [3]	0	0	0	0	0	0	2	0	67	3	5	5
Colorado	0	0	0	0	0	0	2	1	44	9	12	12
New Mexico	0	0	0	0	0	0	0	0	49	4	0	3
Arizona	0	0	0	0	0	0	0	0	13	1	0	2
Utah [2]	0	0	0	0	0	0	0	0	60	6	4	4
PACIFIC												
Washington	0	0	0	0	0	0	1	1	57	18	19	9
Oregon	0	0	0	0	0	0	2	1	20	4	9	10
California	0.8	1	2	2	0.8	0	33	19	42	49	50	54
Total	1.3	33	66	66	2.7	66	414	250	37	927	939	937
31 weeks	2.7	2,072	4,057	4,027	1.0	794	2,485	2,315	177	135,655	163,175	163,175

Division and State	Smallpox				Typhoid and paratyphoid fever				Whooping cough	
	Aug. 6, 1938, rate	Aug. 6, 1938, cases	Aug. 7, 1937, cases	1933–37 median	Aug. 6, 1938, rate	Aug. 6, 1938, cases	Aug. 7, 1937, cases	1933–37 median	Aug. 6, 1938, rate	Aug. 6, 1938, cases
NEW ENGLAND										
Maine	0	0	0	0	12	2	0	3	79	13
New Hampshire	0	0	0	0	0	0	0	0	10	1
Vermont	0	0	0	0	0	0	0	0	477	35
Massachusetts	0	0	0	0	2	2	1	4	121	103
Rhode Island	0	0	0	0	8	1	0	0	107	14
Connecticut	0	0	0	0	9	3	2	2	174	58

See footnotes at end of table.

Cases of certain diseases reported by telegraph by State health officers for the week ended August 6, 1938, rates per 100,000 population (annual basis), and comparison with corresponding week of 1937 and 5-year median—Continued

Division and State	Smallpox				Typhoid and paratyphoid fever				Whooping cough	
	Aug. 6, 1938, rate	Aug. 6, 1938, cases	Aug. 7, 1937, cases	1933–37 median	Aug. 6, 1938, rate	Aug. 6, 1938, cases	Aug. 7, 1937, cases	1933–37 median	Aug. 6, 1938, rate	Aug. 6, 1938, cases
MIDDLE ATLANTIC										
New York	0	0	0	0	10	24	18	18	233	579
New Jersey	0	0	0	0	8	7	7	7	383	319
Pennsylvania	0	0	0	0	8	15	25	25	223	436
EAST NORTH CENTRAL										
Ohio	2	3	0	0	23	30	28	31	344	444
Indiana	9	6	2	0	36	24	6	18	11	7
Illinois	1	2	0	0	12	18	19	28	325	491
Michigan [2]	1	1	0	0	4	4	5	11	301	279
Wisconsin	4	2	1	3	5	3	3	3	649	364
WEST NORTH CENTRAL										
Minnesota	2	1	2	1	0	0	3	2	100	51
Iowa [3]	12	6	3	2	18	9	4	4	49	24
Missouri	3	2	0	0	13	10	14	32	37	28
North Dakota	0	0	3	0	0	0	2	2	118	16
South Dakota	0	0	6	4	15	2	1	1	38	5
Nebraska	8	2	1	2	0	0	1	1	38	10
Kansas	3	1	1	0	14	5	15	15	232	83
SOUTH ATLANTIC										
Delaware	0	0	0	0	0	0	5	2	20	1
Maryland [2 3]	0	0	0	0	40	13	14	14	115	37
District of Columbia	0	0	0	0	0	0	2	2	83	10
Virginia [3]	0	0	0	0	37	19	57	38	92	48
West Virginia	0	0	0	0	14	5	15	31	53	19
North Carolina [3 4]	0	0	0	0	27	18	25	25	270	181
South Carolina [4]	0	0	0	0	56	20	12	31	211	76
Georgia [4]	0	0	0	0	73	43	36	44	44	26
Florida	0	0	0	0	16	5	4	4	53	17
EAST SOUTH CENTRAL										
Kentucky	2	1	0	0	82	46	43	55	95	53
Tennessee [3 4]	2	1	1	0	72	40	43	55	88	49
Alabama [4]	0	0	0	0	47	26	19	19	68	38
Mississippi [3]	0	0	0	0	34	13	16	19	-------	-------
WEST SOUTH CENTRAL										
Arkansas	5	2	0	0	74	29	37	25	20	8
Louisiana [4]	0	0	0	0	32	13	23	24	120	49
Oklahoma	2	1	0	0	39	19	23	23	37	18
Texas [4]	3	3	0	0	70	83	113	70	127	150
MOUNTAIN										
Montana [3]	0	0	9	2	0	0	3	3	648	67
Idaho [3]	21	2	6	1	63	6	0	1	42	4
Wyoming [3]	0	0	0	0	0	0	2	1	89	4
Colorado	24	5	0	0	10	2	0	4	219	45
New Mexico	0	0	0	0	74	6	2	7	161	13
Arizona	0	0	0	0	0	0	1	3	202	16
Utah [2]	0	0	0	0	0	0	1	1	522	52
PACIFIC										
Washington	19	6	5	5	6	2	11	4	201	64
Oregon	25	5	2	2	10	2	8	3	188	37
California	21	25	10	3	11	13	18	10	158	186
Total	3	77	52	52	23	582	687	822	190	4,628
31 weeks	16	12,603	7,847	5,257	9	6,982	6,813	7,786	178	134,900

[1] New York City only.
[2] Period ended earlier than Saturday.
[3] Rocky Mountain spotted fever, week ended Aug. 6, 1938, 22 cases, as follows: Iowa, 1; Maryland, 3; Virginia, 8; North Carolina, 4; Tennessee, 2; Montana, 1; Idaho 2; Wyoming, 1.
[4] Typhus fever, week ended Aug. 6, 1938, 76 cases, as follows: North Carolina, 2; South Carolina, 2, Georgia, 40; Tennessee, 1; Alabama, 6; Louisiana, 1; Texas, 24.

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 fever
June 1938										
Hawaii Territory	0	17	21	----	17	----	0	----	0	7
July 1938										
Connecticut	1	8	12	----	98	----	2	77	0	11
Delaware	0	2	2	----	11	----	0	6	0	4
District of Columbia	0	41	1	----	35	----	1	13	0	25
North Carolina	6	56	14	97	945	222	4	67	1	98
Pennsylvania	12	83	----	1	1,604	1	1	491	0	53

June 1938

	Cases
Hawaii Territory:	
Chickenpox	37
Dysentery (amoebic)	1
Dysentery (bacillary)	1
Encephalitis, epidemic or lethargic	1
Hookworm disease	6
Impetigo contagiosa	5
Jaundice, infectious	1
Leprosy	4
Mumps	35
Paratyphoid fever	1
Tetanus	2
Trachoma	3
Typhus fever	3
Undulant fever	1
Whooping cough	115

July 1938

	Cases
Actinomycosis:	
Pennsylvania	1
Anthrax:	
Delaware	1
Chickenpox:	
Connecticut	94
Delaware	2
Chickenpox—Con.	
District of Columbia	9
North Carolina	41
Pennsylvania	717
Dysentery:	
Connecticut (bacillary)	1
Pennsylvania (amoebic)	1
Encephalitis, epidemic or lethargic:	
Pennsylvania	2
German measles:	
Connecticut	10
North Carolina	12
Pennsylvania	53
Lead poisoning:	
Connecticut	1
Mumps:	
Connecticut	86
Delaware	15
Pennsylvania	1,187
Ophthalmia neonatorum:	
North Carolina	2
Pennsylvania	4
Paratyphoid fever:	
Connecticut	6
North Carolina	5
Rabies in animals:	
Connecticut	7
Rocky Mountain spotted fever:	
District of Columbia	6
North Carolina	13
Septic sore throat:	
Connecticut	9
North Carolina	8
Trichinosis:	
Pennsylvania	1
Typhus fever:	
North Carolina	7
Undulant fever:	
Connecticut	3
North Carolina	5
Pennsylvania	15
Vincent's infection:	
North Carolina	3
Whooping cough:	
Connecticut	339
Delaware	22
District of Columbia	36
North Carolina	1,247
Pennsylvania	1,170

PLAGUE INFECTION IN FLEAS FROM CHIPMUNKS IN FRESNO COUNTY, CALIF.

Under date of August 3, 1938, Dr. W. M. Dickie, State Director of Health of California, reported plague infection proved in a pool of 13 fleas from 7 chipmunks collected July 14 at Cedar Crest Resort, in the Sierra National Forest, 2 miles west of Lake Shore, Huntington Lake, Fresno County, Calif.

WEEKLY REPORTS FROM CITIES

City reports for week ended July 30, 1938

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	106	35	13	750	311	329	5	377	84	1,359	----
Current week [1]	93	34	12	547	251	285	7	344	73	1,789	----
Maine:											
Portland	0	----	0	2	0	0	0	0	1	3	22
New Hampshire:											
Concord	----	----	----	----	----	----	----	----	----	----	----
Manchester	1	----	0	0	1	0	0	0	0	0	22
Nashua	0	----	0	0	0	0	0	0	0	0	4
Vermont:											
Barre	0	----	0	0	0	0	0	0	0	0	5
Burlington	0	----	0	0	0	2	0	0	0	0	4
Rutland	0	----	0	0	0	0	0	0	0	0	4
Massachusetts:											
Boston	0	----	0	41	5	25	0	13	0	26	197
Fall River	0	----	0	0	2	2	0	1	0	0	30
Springfield	0	----	0	13	0	3	0	2	0	17	21
Worcester	0	----	0	0	2	1	0	1	1	13	37
Rhode Island:											
Pawtucket	0	----	0	0	0	0	0	0	0	0	14
Providence	0	----	0	0	0	4	0	0	0	18	38
Connecticut:											
Bridgeport	0	----	0	0	1	1	0	0	1	0	19
Hartford	0	----	0	3	1	2	0	0	0	2	44
New Haven	0	1	0	0	0	0	0	1	0	5	24
New York:											
Buffalo	0	----	0	2	8	8	0	5	0	40	113
New York	14	1	1	142	48	26	0	61	11	321	1,145
Rochester	0	----	0	11	2	1	0	1	0	5	48
Syracuse	0	----	0	25	1	0	0	0	1	7	42
New Jersey:											
Camden	2	1	0	2	1	1	0	1	0	1	22
Newark	0	----	0	3	4	4	0	5	0	66	102
Trenton	0	----	0	0	0	1	0	0	0	2	25
Pennsylvania:											
Philadelphia	2	1	1	22	12	16	0	14	9	75	356
Pittsburgh	4	----	0	4	8	9	0	7	0	33	129
Reading	2	----	0	0	2	1	0	0	0	5	20
Scranton	0	----	----	1	----	1	0	----	0	5	----
Ohio:											
Cincinnati	5	----	0	1	1	8	0	8	2	14	103
Cleveland	0	2	0	11	6	10	0	11	1	72	165
Columbus	2	----	0	0	0	9	0	3	0	1	66
Toledo	0	----	0	2	2	0	0	0	0	25	57
Indiana:											
Anderson	0	----	0	0	0	0	0	1	0	2	6
Fort Wayne	0	----	0	0	1	0	0	0	0	0	24
Indianapolis	2	----	0	5	9	3	0	2	2	7	105
Muncie	0	----	0	0	1	0	0	0	0	0	15
South Bend	0	----	0	0	3	1	0	1	0	2	18
Terre Haute	0	----	0	0	0	0	0	0	0	0	11
Illinois:											
Alton	1	----	0	0	1	2	0	0	0	0	11
Chicago	11	3	2	8	13	39	0	41	2	319	575
Elgin	0	----	0	0	0	0	0	0	0	0	8
Moline	0	----	0	0	1	0	0	0	0	2	5
Springfield	0	----	0	0	0	0	0	0	0	3	19
Michigan:											
Detroit	4	----	1	2	4	12	0	19	0	247	228
Flint	0	----	0	2	4	6	0	0	0	3	19
Grand Rapids	0	----	0	11	1	5	0	0	0	1	32
Wisconsin:											
Kenosha	0	----	0	4	0	1	0	0	0	17	4
Madison	2	----	0	6	0	2	0	0	0	3	16
Milwaukee	0	1	1	5	3	3	0	1	0	168	88
Racine	0	----	0	0	0	0	0	0	0	30	9
Superior	0	----	0	0	0	0	0	0	0	3	6

[1] Figures for Concord, N. H., and Salt Lake City, Utah, estimated; reports not received.

City reports for week ended July 30, 1938—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								
Minnesota:											
Duluth	0	------	0	14	1	5	0	1	0	13	22
Minneapolis	0	------	1	12	0	6	0	1	0	6	76
St. Paul	0	------	0	7	1	1	0	4	0	3	60
Iowa:											
Cedar Rapids	1	------	--------	1	--------	0	0	--------	0	4	--------
Davenport	0	------	--------	0	--------	0	0	--------	0	0	--------
Des Moines	1	------	0	0	0	4	1	0	0	0	22
Sioux City	0	------	--------	8	--------	2	0	--------	0	10	--------
Waterloo	0	------	--------	2	--------	1	0	--------	0	0	--------
Missouri:											
Kansas City	0	------	1	1	10	5	0	6	0	2	88
St. Joseph	0	------	0	0	3	0	0	0	1	1	24
St. Louis	2	------	0	2	0	2	0	8	0	4	212
North Dakota:											
Fargo	0	------	0	1	0	3	0	0	0	1	9
Grand Forks	0	------	--------	0	--------	1	0	--------	0	0	--------
Minot	0	------	0	3	0	0	0	0	0	1	7
South Dakota:											
Aberdeen	0	------	--------	0	--------	0	0	--------	0	0	--------
Sioux Falls	0	------	0	0	0	0	0	0	0	0	12
Nebraska:											
Lincoln	0	------	--------	2	--------	0	0	--------	0	5	--------
Omaha	0	------	0	4	3	0	0	1	0	0	52
Kansas:											
Lawrence	0	------	0	0	0	0	0	0	0	3	4
Topeka	1	------	0	0	3	0	0	0	0	10	15
Wichita	0	------	0	1	1	3	0	1	0	0	29
Delaware:											
Wilmington	0	------	0	0	2	0	0	0	0	3	18
Maryland:											
Baltimore	2	2	1	2	4	7	0	11	1	21	184
Cumberland	0	------	0	0	0	0	0	0	0	0	6
Frederick	0	------	0	0	0	0	0	0	0	0	7
Dist. of Col.:											
Washington	4	1	1	3	1	3	0	12	1	4	124
Virginia:											
Lynchburg	1	------	0	0	2	0	0	1	0	2	16
Norfolk	0	------	0	0	2	0	0	1	0	1	22
Richmond	0	------	1	8	4	0	0	2	1	0	53
Roanoke	0	------	0	0	0	0	0	1	0	0	16
West Virginia:											
Charleston	1	------	0	0	0	1	0	0	0	0	8
Wheeling	0	------	0	0	0	0	0	0	0	2	15
North Carolina:											
Gastonia	1	------	--------	0	--------	0	0	--------	0	0	--------
Raleigh	0	------	0	0	0	0	0	1	5	2	9
Wilmington	0	------	0	0	0	0	0	0	0	5	13
Winston-Salem	0	------	0	20	0	0	0	0	0	1	14
South Carolina:											
Charleston	1	------	0	1	1	1	0	1	4	0	14
Florence	0	------	0	0	0	0	0	0	0	0	15
Greenville	0	------	0	2	0	1	0	0	1	3	12
Georgia:											
Atlanta	0	9	1	0	5	5	0	4	0	7	86
Brunswick	0	------	--------	0	--------	0	0	--------	0	2	--------
Savannah	0	------	0	0	0	0	0	1	0	0	28
Florida:											
Miami	0	------	0	0	3	0	0	0	0	1	21
Tampa	1	1	0	4	1	0	0	3	0	0	24
Kentucky:											
Ashland	0	------	--------	0	--------	0	0	--------	0	1	--------
Covington	0	------	0	0	0	0	0	0	0	0	13
Lexington	2	------	0	0	0	0	0	0	0	0	21
Louisville	1	------	0	7	1	0	0	3	2	10	70
Tennessee:											
Knoxville	0	------	0	0	3	2	0	0	2	13	35
Memphis	0	------	0	0	1	2	0	11	1	2	83
Nashville	0	------	0	0	1	0	0	5	4	4	50
Alabama:											
Birmingham	2	4	0	1	2	1	0	3	4	0	64
Mobile	0	------	0	0	1	0	0	1	0	0	15
Montgomery	1	------	--------	0	--------	0	0	--------	0	3	--------
Arkansas:											
Fort Smith	1	1	--------	0	--------	1	0	--------	1	1	--------
Little Rock	0	------	--------	0	4	0	0	2	0	0	--------

City reports for week ended July 30, 1938—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								
Louisiana:											
Lake Charles	0		0	1	0	0	0	0	0	0	4
New Orleans	8	2	0	3	8	1	0	14	5	40	133
Shreveport	0		0	0	2	0	0	1	0	0	40
Oklahoma:											
Oklahoma City	0		0	0	1	2	0	1	3	1	46
Tulsa	0			8		0	0		0	12	
Texas:											
Dallas	2		0	2	1	2	0	4	5	4	41
Fort Worth	0		0	0	2	2	0	2	0	6	42
Galveston	1		0	0	2	0	0	1	0	0	9
Houston	5		0	0	3	3	0	3	4	2	73
San Antonio	0		0	0	9	0	0	9		4	58
Montana:											
Billings	0		0	0	0	0	0	0	0	9	6
Great Falls	0		0	0	0	1	0	1	0	1	7
Helena	0		0	1	0	0	0	0	0	0	4
Missoula	0		0	0	0	0	2	0	0	0	2
Idaho:											
Boise	0		0	0	1	0	0	0	0	0	10
Colorado:											
Colorado Springs	0		0	0	0	1	0	0	0	5	10
Denver	4		0	6	2	3	0	0	1	19	76
Pueblo	0		0	8	1	0	0	0	0	1	8
New Mexico:											
Albuquerque	0		0	1	0	0	0	5	0	5	14
Utah:											
Salt Lake City											
Washington:											
Seattle	0		0	5	7	1	0	3	0	7	93
Spokane	0		0	3	1	0	4	0	1	1	24
Tacoma	0		0	0	0	3	1	0	0	1	33
Oregon:											
Portland	0	1	0	9	1	1	1	0	0	3	57
Salem	0			0		0	0		0	3	
California:											
Los Angeles	10	4	0	24	12	17	0	27	1	46	305
Sacramento	0		0	10	3	0	0	1	0	6	29
San Francisco	0	1	0	3	5	4	0	4	0	16	133

State and city	Meningitis, meningococcus		Poliomyelitis cases	State and city	Meningitis, meningococcus		Poliomyelitis cases
	Cases	Deaths			Cases	Deaths	
Rhode Island:				Maryland:			
Pawtucket	0	0	1	Frederick	0	0	1
Providence	0	0	1	Florida:			
Connecticut:				Tampa	0	0	1
Bridgeport	0	0	1	Kentucky:			
New Haven	0	0	1	Louisville	0	0	1
New York:				Tennessee:			
Buffalo	0	2	0	Knoxville	0	0	1
New York	2	0	4	Memphis	0	0	1
Pennsylvania:				Alabama:			
Philadelphia	0	0	1	Birmingham	0	0	3
Ohio:				Louisiana:			
Columbus	0	0	1	New Orleans	0	0	1
Illinois:				California:			
Chicago	3	0	1	Los Angeles	0	0	1

Encephalitis, epidemic or lethargic.—Cases: New York, 2; Denver, 1.
Pellagra.—Cases: Charleston, S. C., 1; Atlanta, 1; Louisville, 1; Nashville, 1; Birmingham, 5; New Orleans, 2.
Rabies in man.—Deaths: St. Louis, 1.
Typhus fever.—Cases: New York, 1; Wilmington, N. C., 1; Charleston, S. C., 2; Atlanta, 1; Savannah, 2; Tampa, 1; Mobile, 1; Houston, 2. Deaths: Savannah, 1.

FOREIGN AND INSULAR

AUSTRALIA

South Australia—Infectious diseases—1935-37.—For the years 1935, 1936, and 1937, certain infectious diseases have been reported in South Australia as follows:

Disease	1935		1936		1937	
	Cases	Deaths	Cases	Deaths	Cases	Deaths
Cerebrospinal meningitis	3	1	2	1	2	
Chickenpox	1,192		1,423		1,223	
Diphtheria	526	19	1,279	30	774	27
Dysentery (amoebic)	1		4	3	4	1
Dysentery (bacillary)		5	19	1	3	2
Erysipelas	95	6	121	6	110	3
Favus	3					
Influenza	705	25	135	14	143	10
Lethargic encephalitis	2	2	2	3	4	5
Malaria	2					
Measles	454		88		72	
Mumps	84		198		107	
Paratyphoid fever	2		1			
Poliomyelitis	18	2	5		85	2
Puerperal fever	46	8	89	9	58	6
Scarlet fever	461		397	1	214	2
Tuberculosis (pulmonary)	318	233	266	199	331	225
Typhoid fever	29	5	51	3	27	4
Typhus fever	14	1	13	3	6	
Whooping cough	3,616	25	751	9	684	5

Deaths by causes—1935-37.—The following table shows the deaths by important causes in South Australia with rates per 10,000 population for the years 1935, 1936, and 1937:

Cause	1935		1936		1937	
	Deaths	Rate	Deaths	Rate	Deaths	Rate
Diseases of the heart	1,029	17.59	1,101	18.74	1,041	17.67
Cancer and other malignant tumors	610	10.43	710	12.60	725	12.31
Tuberculosis (all forms)	260	4.44	235	4.00	256	4.35
Cerebral hemorrhage and softening	488	8.34	527	8.97	492	8.35
Pneumonia (all forms)	390	6.67	343	5.84	275	4.67
Bronchitis	59	1.01	74	1.26	57	.97
Nephritis (acute and chronic)	274	4.68	252	4.29	276	4.69
Diabetes mellitus	88	1.50	110	1.87	116	1.97
Puerperal causes	49	.84	53	.89	46	.78
Congenital debility and malformations	199	3.40	216	3.67	223	3.79
Senility	322	5.50	318	5.41	285	4.84
Suicides	59	1.01	69	1.17	56	.95
Violent deaths exclusive of suicides	242	4.14	270	4.59	318	5.40
Diarrhea and enteritis	27	.46	40	.68	48	.81
Whooping cough	23	.39	9	.15	5	.08
Diphtheria and croup	20	.34	30	.51	27	.46
Influenza	26	.45	17	.29	9	.15
Typhoid fever	5	.09	4	.07	4	.07
Appendicitis	40	.68	35	.60	52	.88
Hernia, intestinal obstruction	53	.91	52	.89	61	1.03
Cirrhosis of the liver	23	.39	15	.25	22	.37
Tetanus	14	.24	11	.19	7	.12

The population for 1937: 591,201.

Births and deaths—1935–37.—The following table shows the number of births and deaths with the rates per 1,000 population, and the number of deaths of infants under 1 year of age with the rate per 1,000 live births. The infant death rate for 1936 of 31.09 was stated to be just a little higher than the world low record of 30.96 for New Zealand. The 1937 rate increased to 33.05, which is the lowest South Australian rate except for 1936 and 1933. The infant death rate in this State is less than half the rate 20 years ago, and only about one-third of the rate about 1900.

Births and deaths in South Australia

	1935	1936	1937
Number of births	8,270	8,911	8,985
Births per 1,000 population	14.14	15.17	15.25
Total deaths	5,163	5,464	5,247
Deaths per 1,000 population	8.83	9.30	8.91
Deaths under 1 year of age	289	277	297
Deaths under 1 year of age per 1,000 live births	34.95	31.09	33.05

CHINA

Typhoid fever.—Under date of August 3, 1938, the American Consul General at Shanghai, China, reported 103 new cases of typhoid fever with 55 deaths for the week ended July 30, 1938, in Shanghai and outlying areas.

CUBA

Habana—Communicable diseases—4 weeks ended July 30, 1938.—During the 4 weeks ended July 30, 1938, certain communicable diseases were reported in Habana, Cuba, as follows:

Disease	Cases	Deaths	Disease	Cases	Deaths
Diphtheria	13		Tuberculosis	13	3
Dysentery (bacillary)	1		Typhoid fever	[1] 40	9
Malaria	[1] 23				

[1] Includes imported cases.

Provinces—Notifiable diseases—4 weeks ended July 23, 1938.—During the 4 weeks ended July 23, 1938, cases of certain notifiable diseases were reported in the Provinces of Cuba as follows:

Disease	Pinar del Rio	Habana	Matanzas	Santa Clara	Camaguey	Oriente	Total
Cancer	2	3	1	3	2	3	14
Chickenpox		1	2	1		1	5
Diphtheria	1	10	3	2		2	18
Dysentery (bacillary)				8			8
Leprosy	1						1
Malaria	43	19	8	57	21	54	202
Measles	1	4	2	3			10
Tuberculosis	48	98	34	36	19	25	260
Typhoid fever	13	114	37	85	28	113	390
Whooping cough				2			2

EGYPT

Infectious diseases—Third quarter 1937.—During the third quarter of 1937, certain infectious diseases were reported in Egypt as follows:

Disease	Cases	Deaths	Disease	Cases	Deaths
Anthrax		2	Mumps	214	6
Cerebrospinal meningitis	29	26	Poliomyelitis	1	
Chickenpox	53	2	Puerperal septicemia	129	112
Diphtheria	511	227	Rabies	8	8
Dysentery	781	144	Scarlet fever	12	1
Erysipelas	1,125	256	Tetanus	107	70
Influenza	2,635	47	Tuberculosis (pulmonary)	1,333	548
Jaundice, epidemic	3	2	Typhoid fever	1,946	414
Leprosy	49	12	Typhus fever	165	15
Lethargic encephalitis	2	1	Undulant fever	4	2
Malaria	10,574	43	Whooping cough	383	26
Measles	2,601	653			

Vital statistics—Third quarter 1937.—Following are vital statistics for the third quarter of 1937 for all places in Egypt having a health bureau:

Number of live births	50,757
Live births per 1,000 inhabitants	43.2
Number of stillbirths	1,036
Number of deaths	40,195
Deaths per 1,000 inhabitants	34.2
Deaths under 1 year of age	14,249
Deaths under 1 year of age per 1,000 live births	281

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 29, 1938, pages 1322-1335. A similar cumulative table will appear in future issues of the PUBLIC HEALTH REPORTS for the last Friday of each month.

Cholera

China.—Cholera has been reported in China as follows: Week ended July 2, 1938, Hankow, 35 cases; Kwangtung Province, 6,267 cases. Week ended July 30, 1938, Canton, 5 cases; Hong Kong, 25 cases; Macao, 24 cases; Shanghai, 121 cases; Swatow, 4 cases. (The American Consul General at Shanghai reported under date of August 3, 1938, that for the week ended July 30, there had been reported 1,233 new cases of cholera, with 233 deaths in Shanghai and outlying areas, 49 percent of which cases were said to be true cholera.)

French India.—For the week ended June 25, 1938, 2 cases of cholera were reported in Pondichery Territory, and 6 cases in Chandernagor. During the week ended July 2, 1938, 1 case of cholera was reported in Yanaon, French India.

French Indochina.—During the week ended July 30, 1938, cholera was reported in French Indochina as follows: Annam Province, 290 cases; Tonkin Province, 42 cases, Hanoi, 2 cases.

Japan.—Cholera has been reported in Japan as follows: During the week ended July 23, 1938, 5 cases with 4 deaths in Okayama Prefecture, and on July 27, 1938, 2 cases were reported in Wakamatsu, Fukuoka Prefecture.

On vessel—S. S. Kikukawa Maru.—Information dated August 1, 1938, states that 57 cases of Asiatic cholera with 16 deaths occurred on the *S. S. Kikukawa Maru* which arrived from Shanghai, at Fukuoka, Japan, on July 28, 1938. The vessel was being held in the quarantine station at Moji.

Plague

Bolivia—Tarija Department.—During the period June 13–26, 1938, 44 cases of plague were reported in Tarija Department, Bolivia.

United States—California.—A report of plague-infected fleas in Fresno County, Calif., appears on page 1491 of this issue of PUBLIC HEALTH REPORTS.

Yellow Fever

Gold Coast.—During the week ended July 30, 1938, 1 case of yellow fever was reported in Tjalele, Gold Coast, and on July 25, 1938, 1 case of yellow fever was reported in Akateng, Gold Coast.