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ZINC IN RELATION TO GENERAL AND INDUSTRIAL HYGIENE

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Recent investigations have greatly simplified and defined the problems arising from the ingestion and inhalation of zinc and zinc compounds. It is now known that zinc is not a poison in the usual sense of the word. That is to say, pure zinc, in contrast to metals such as arsenic, lead, antimony, and cadmium, has no poisonous qualities even if taken in large amounts. Certain zinc compounds, such as zinc chloride, are highly irritating and caustic; but this action is the common property of an infinite number of chemical substances. Certain other zinc compounds, such as zinc oxide or zinc stearate, on account of their fine state of division or their tendency to flocculation, may do harm, but not on account of their zinc content. Such effects are understandable and avoidable, a far different situation from that which would exist were the zinc ion poisonous in itself, so that after absorption one might count upon a series of unfortunate effects ending with elimination, with safe storage, or with the death of the subject.

This point of view is not in accord with the reputation zinc has held, a reputation which has arisen from the fact that zinc in the majority of instances occurs in ores with such substances as lead, arsenic, antimony, and cadmium, all definitely poisonous. Thus the older literature of general and industrial hygiene contains frequent descriptions of what are listed as cases of acute or chronic zinc poisoning, in which the symptoms are attributable to contaminating metals (1).

In the light of present knowledge it is worth while to redefine the hygienic position of zinc. This may be done from two aspects—first, that of the relation of zinc and zinc compounds to the health of the general public, and, second, the relation of zinc and zinc compounds to the health of workers engaged in the manufacture of such compounds.

1. ZINC IN RELATION TO THE HEALTH OF THE PUBLIC

(*a*) *Zinc and zinc compounds in drinking water.*—The most important feature of this relation is the presence of zinc and zinc compounds in drinking water. The process of galvanizing is over a hundred years old; the making of brass pipe and brass containers is far older. In both cases solution of zinc may occur. In 1925, a

publication of the Public Health Service (Reprint no. 1029) placed a limit of 5 parts per million of zinc in drinking water. This limit is almost certain to be exceeded if water containing appreciable amounts of carbon dioxide remains in contact with brass (2) (3), if rainwater passes through galvanized pipe or is held in galvanized containers (4), and if the water is from springs in regions rich in zinc (5). In 14 groups of analyses made in various parts of the world, only 5 conform to the limit of 5 parts of zinc per million of water, and conformity is not invariable in water taken from the same systems of piping. The limit is often markedly exceeded. Thus Haines (6) found 56 parts of zinc per million of water drawn through galvanized iron pipe and Mason (5) 327 parts of zinc sulphate per million of spring water from the zinc-blende district of Missouri. Where such high figures have been obtained, the authors have frequently called attention to the possible hazard to health offered by the situation; but it is significant that during the many years in which zinc-lined pipe has been in use no definite cases of acute or chronic illness have ever been traced with certainty to zinc.

In a recent paper on "Zinc in Water Supplies", Bartow and Weigle (7) report upon 13 specimens of water from the zinc-producing area of Missouri, Kansas, and Oklahoma. They found 0.9 to 50 parts per million of zinc. Two sets of analyses were done, the first in April 1931, the second in May. The limit of 5 parts per million was reached in 3 specimens and doubled or much more than doubled in 12 specimens out of 21 examined. The authors made inquiries through the district and report: "From the best evidence obtainable, the water containing zinc was used by man and animals without harmful results." In order to gain further assurance, tests were made upon rats given water containing pure zinc sulphate. When the zinc so administered was as high as 100 parts per million, no appreciable ill effects were noted. Bartow and Weigle conclude:

> In reporting the harmful effects of zinc in water supplies in contact with galvanized pipes or containers, the possible effect of other impurities has not been considered—for example, cadmium and arsenic, which are sometimes present in zinc used for galvanizing.
>
> The indications that pure zinc salts in reasonable amounts in drinking water are not harmful, and the facts that pure zinc can now be made cheaply and that pure zinc is difficultly soluble, suggest the possibility that the solvent action of waters on galvanized iron pipes could be reduced by galvanizing them with pure zinc.

In connection with the large amounts of zinc sulphate used in these experiments and reported in drinking waters, we asked 10 women and 10 men to taste distilled water to which zinc sulphate had been added. Two detected 19 parts per million, 4 tasted 37 parts per million, 10 tasted 73 parts per million, and the remaining 4 made their first detection at 156 parts per million. All of the readily soluble

zinc salts possess an unpleasant, astringent taste. When added to water and this addition is wholly undisguised, most people are apparently able to detect the taste before there is possibility of taking in enough to produce gastric irritation. When, however, these salts contaminate food, their taste is easily disguised, and amounts irritating enough to cause vomiting can readily be ingested. So far as plain water is concerned it is certainly proper to conclude that a limit of zinc contamination of 5 parts per million is neither feasible nor necessary. Amounts greater than this are readily found and have not caused harm. If a limit is desirable, 30 parts per million is suggested for the following reason:

When the source of zinc contaminating water is galvanized pipe—and this is the most common situation with which we deal—the zinc compounds in the water will be a mixture of oxide, hydroxide, and carbonate, the latter predominating. If present to an amount of 30 mg of zinc per liter, or 30 parts per million, zinc carbonate will cause appreciable milkiness in the water, and many persons will complain of an astringent taste. While there is no reason to consider this harmful, it is doubtful whether any community would tolerate it without incessant complaint. Both taste and appearance act to prevent zinc in its most common form from being a persistent contaminant of water supplies even in amounts which are harmless.

It may be asked whether chlorinated water constitutes a special case. On the basis of extensive data upon the water supply of the Franklin Plant of the New Jersey Zinc Co., Anderson, Reinhard, and Hammel (8) conclude that chlorination reduces the attack of water upon zinc and is thus advantageous rather than the reverse. This conclusion is in accord with previous findings by Lothian and Ward (9).

(*b*) *Zinc and zinc compounds in foods.*—Zinc in varying amounts occurs universally in all types of plant and animal life. This fact has made it extremely difficult to perform experiments in which zinc has been eliminated from the diet, since foods freed from zinc are either so mutilated in the process as to be wholly unpalatable or have so far lost other necessary dietary constituents as to make feeding experiments impossible. The possibilities of zinc ingestion are well illustrated by the following high zinc meal taken for experimental purposes and reported by K. R. Drinker, Fehnel, and Marsh (10). The menu was as follows:

"Hors d'œuvres: yeast cake with anchovies, chopped olives, and mayonnaise; each subject ate approximately three-fourths of a yeast cake. Large raw oysters, twelve apiece. Oyster soup containing approximately eight oysters apiece. Duck, bread stuffing, apple sauce, wheat bran pudding, one large ear of corn on the cob each, butter (subject 1, bread). Strawberry gelatin, whipped cream. Coffee and sugar.

"The food materials in this meal especially rich in zinc were: Yeast (414.8 mg zinc per kilo), oysters (26 to 2,298 mg per kilo), bran (139.2 mg per kilo), corn (25.2 mg per kilo), gelatin (27.4 mg per kilo). These figures are taken from Lutz's (11) review of the normal occurrence of zinc in biological materials. An approximate estimate indicates that each subject ingested at this one meal between 225 and 275 mg of zinc."

On an ordinary mixed diet, Drinker, Fehnel, and Marsh (10) found an average excretion of 10.7 mg of zinc per day. One subject, following ingestion of the meal given above, excreted 200 mg of zinc on the next day. Such figures as these, coupled with animal experiments on feeding noncaustic zinc compounds, and with observations upon men working in zinc plants and showing zinc excretion averaging 48 mg a day, make it obvious that the mere fact of zinc ingestion is of no importance. Our concern is wholly with the zinc compound which reaches the stomach. It is possible that large amounts of zinc oxide would result in the formation of enough zinc chloride in the stomach to cause nausea, but the dosage must be great.

The facts are that practically all of the readily soluble salts of zinc are irritating or even caustic. Arranged in order of activity, the more common soluble compounds are about as follows: Zinc chloride, zinc sulphate, zinc acetate, zinc lactate, zinc tartrate, and zinc malate. Of this number, zinc chloride and zinc sulphate are not likely to contaminate food. The others are encountered fairly frequently and almost invariably because acid foods or beverages have been stored in galvanized containers. Many instances of nausea and of vomiting can be cited illustrating the inevitable result of disregard of this fact. The situation is so obvious as to make such quotations unnecessary. Zinc cannot be allowed to remain in contact with foods or beverages where moisture and acidity are encountered, without the possibility of results which may be decidedly unpleasant. This means that the householder should never cook or store food in galvanized utensils, and that brass utensils should not be used in cooking acid foods nor in storing foods or drinks. Zinc foil is a safe wrapper for dry and non-acid substances such as tea in the package or bar chocolate, but cannot, for example, be used on cheese because of the rapidity of corrosion.

2. ZINC IN RELATION TO INDUSTRIAL HYGIENE

In the course of the manufacture of zinc and zinc compounds, a single condition remains which calls attention to zinc, and this again is not due to a specific attribute of the metal but is apparently due to the physical state of certain zinc compounds. This is the familiar chill and fever formerly thought to be produced by zinc oxide alone but now known to occur when oxides or finely divided powders of other metals are inhaled. In typical cases following inhalation of

freshly formed zinc oxide or large amounts of old oxide, the worker after a few hours experiences malaise, then a severe shaking chill with a rise in temperature of considerable magnitude. Philip Drinker and his associates (12) (13) (14) (15) have studied these chills under circumstances permitting close clinical observation; that is, employing pure zinc oxide they have produced chills in themselves and in volunteers. Their findings were that ague occurs in from 2 to 4 hours after zinc-oxide inhalation. They found that in laboratory experiments 45 mg of zinc oxide (measured as zinc) per cubic meter of air can be inhaled for 20 minutes without causing symptoms. In a metallurgical plant 14 mg per cubic meter produced no reaction in 8 hours. Differences in susceptibility make it impossible to give absolute figures as to the concentrations certain to cause chills. Apparently no one is entirely immune, given a sufficiently large inhalation.

Philip Drinker found it easy to demonstrate the immunity which exists following a zinc chill, and this immunity is perhaps related to the leucocytosis which occurs with the chill and persists in many workmen exposed daily to zinc-oxide inhalation (1).

Batchelor, Fehnel, Thomson, and K. R. Drinker (1) made an exhaustive clinical and laboratory study of 24 workmen exposed over periods of time varying from 2 to 35½ years to the inhalation of varying concentrations of zinc in the form of zinc oxide, zinc sulphide, or fine metallic dust, and found no acute or chronic illness in any way ascribable to zinc. These observations are significant because the exposure to zinc was high and contamination by lead and cadmium was very low. Many of the men studied had often had zinc chills and, in addition, had experienced persistent dosage by inhalation.

Turner and Thompson (16) examined 212 brass founders and reported that those who experienced attacks of metal fume fever were "apparently in somewhat poorer condition than those exposed to the fumes but not affected by them."

One may sum up the evidence in regard to the zinc chill as follows: It is a decidedly unpleasant experience and, to a mild degree, incapacitates one the day following the attack. Where the inhaled oxide is uncontaminated by other metals, there is no definite evidence of chronic damage even from repeated chills. At the same time the condition is easy to prevent by properly arranged exhaust hoods or the wearing of masks, and conditions making chills possible should be eliminated. Mention has been made of the fact that the chill is not necessarily due to zinc. Philip Drinker (15) found it possible to produce fume fever in himself and his associate (R. M. Thomson) by inhaling magnesium oxide. The same result has been reported for other metals. The ease with which zinc assumes a physical or physico-chemical state capable of causing fume fever when inhaled

is apparently the reason why the condition has been thought to be due to zinc alone.

Finally, it is proper to mention a mechanical effect of zinc experienced in connection with zinc stearate dusting powders. Such powders have frequently caused death in infants who have inhaled them directly from the can. These instances have no bearing upon the problem of zinc toxicity. They occur when any dry, adhesive powder is inhaled in sufficient amount.

SUMMARY

A summary of the hygienic position of zinc with such restrictions as experience makes advisable is as follows:

1. In 1925, a publication of the Public Health Service (Reprint no. 1029) placed a limit of five parts per million of zinc in drinking water. This limit has been applied freely to many conditions in which zinc is ingested. Since the zinc ion is not of itself poisonous, and many times five parts per million may be taken without harmful effects, it is suggested that this limit, which gives a relatively innocuous metal an undeserved reputation for toxicity, be increased or done away with altogether.

2. Foods or beverages, with the exception of simple or chlorinated drinking water, should not be stored in zinc-lined or galvanized containers. Acid drinks and foods will invariably cause solution of zinc and the formation of simple compounds of zinc which irritate the stomach and may cause vomiting.

3. A single industrial condition arises from zinc and this condition is not produced by zinc alone. This is the "zinc chill", better known as metal fume fever. The different groups of symptoms described as chronic industrial zinc poisoning, together with other complex ills which have been ascribed to zinc, may be disregarded, as they are due to contamination by other substances.

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THE SIZE FREQUENCY OF INDUSTRIAL DUSTS

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The question as to the relative significance of various sizes of dust particles in the production of lung fibrosis has as yet not been satisfactorily answered by pathologists engaged on this problem. However, we do know that the inhalation of certain industrial dusts has been found to be associated with definite injury to the pulmonary tissues. Hence a knowledge of the size frequency of these dusts might cast some light on this problem. Of equal importance is the fact that such data will determine to a large degree the type of dust-sampling instrument and the method of dust counting to be employed in evaluating the industrial dust hazard.

Moir (1), Watkins-Pitchford (2), and Mavrogordato (3), of South Africa, have shown that the majority of the dust particles recovered by them from both human and animal silicotic lungs were between 1

and 3 microns in size. Only 13 percent of the particles were found to be less than 0.5 micron. These results have been recently checked by Scheid (4) of Germany. Drinker (5), in comparing the size frequency of the particles measured by Moir with the particles found by him in the sputum of men employed in ore mills, found a close correspondence. The results of these findings raise two pertinent questions; namely, (1) to what extent are minute particles of dust retained by the human lungs, and (2) do appreciable percentages of industrial dusts ever fragment into those minute sizes less than 0.5 micron?

The work of Drinker (6) and his associates and that of Brown (7) (8) on the retention of certain dusts and fumes by man when known amounts were breathed, seem to indicate that the coarser suspensions were retained more effectively than the more finely divided fumes. Percentage retention was found to be directly proportional to particulate size and to the density of dust suspended in the air. Owens (9), upon measuring the amount of dust in expired air of London inhabitants, found that only 25 percent of the dust was retained.[1] The average size of the dust inhaled in Owens' experiments was about 0.5 micron. This work on dust retention seems to bear out the theory that dust particles of a size less than 0.5 micron play but a small role in the problem of industrial dust inhalation and in a manner disposes of the first question raised. The answer to the second question—that is, the ability of the ordinary industrial process to fragment appreciable quantities of dust to a size less than 0.5 micron—is best answered by a particle-size study of the dusts actually suspended in industrial atmospheres.

The present contribution deals with such a study of the size frequency of certain industrial dusts encountered in the course of investigating the health of workers in dusty trades. In addition, a discussion is presented concerning the sampling and enumeration of aerial dusts in relation to the results obtained on the size range of such dusts.

INSTRUMENTS AND METHODS USED IN STUDY

In order to obtain a sample of dust from the air in more or less unaltered condition, the Owens jet dust counter (10) was used, since with this instrument the atmospheric dust is directly projected on a naked cover slip. Badham (11), in his study of the characteristics of this instrument, obtained correlations between the Owens and the impinger apparatus (12), and concluded from his study that the efficiency of the Owens counter is of the same order as the impinger,

[1] In 1927 R. R. Sayers et al. called attention to the fact that fine particulate matter such as lead dust from automobile exhaust gas was retained to an average extent of only 15 percent of the amount inhaled. (Experimental Studies on the Effect of Ethyl Gasoline and its Combustion Products, U.S. Bureau of Mines Monograph No. 2.)

for particles between 0.5 micron and 10 microns and that the results obtained by the Owens counter indicate, for all practical purposes, the dustiness of the air. Our own experience with this instrument also leads us to believe that it samples the dust in the air effectively, and especially the smaller sized particles.

After obtaining dust samples, the cover slips were mounted in the usual manner. The dust particles were measured by the use of a filar ocular micrometer (13) at a magnification of 1,000 diameters (oil immersion objective). The horizontal diameter of at least 200 dust particles in several representative fields was measured for each sample. With this magnification it was found possible to measure particles as small as 0.5 micron in size, while particles smaller than this size are easily distinguished at this magnification and their presence recorded.

Photographic methods have been suggested and used for measuring dust particles; but, in order to obtain good photomicrographs, it is essential that the dust particles be in one plane, free from Brownian movement and well dispersed. Since industrial dusts are seldom of a uniform size, it is difficult to fulfill the first requirement. At the beginning of this study a comparison was made between the results obtained with the direct filar measurements and the photographic method on a typical industrial dust sample. This comparison demonstrated that the simpler and less expensive filar method yielded practically the same results. Since the filar method fulfilled the requirements of our problem, it was selected for the present study.

RESULTS OF STUDY

Table 1 presents the results of the measurements of some 6,000 industrial dust particles and 18,000 outdoor dust particles. The latter measurements were obtained during the course of a study of the atmospheric smoke pollution problem in our large cities and are presented for the sake of comparison. The number of samples obtained for each dust, the median size,[2] and the average frequency in percent for each size group is indicated in this table. In all 26 samples of 11 different kinds of industrial dusts were examined. These dusts ranged from the dust present in sandblasting operations to that associated with the fine pulverizing operations in trap rock and talc milling plants.

[2] The median is the center item in an array and may be strictly defined as a point on the abscissal scale of a frequency distribution with 50 percent of the items on either side.

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

Kind of dust	Number of samples	Median	Average frequency in percent—Size group in microns										
			0 to 0.49	0.5 to 0.99	1.0 to 1.49	1.5 to 1.99	2.0 to 2.49	2.5 to 2.99	3.0 to 3.49	3.5 to 3.99	4.0 to 4.49	4.5 to 4.99	5.0 to 5.49	5.5 to 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	0	13.0	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	0.5	22.0	42.0	17.3	9.2	5.0	1.5	2.0	.5			
General foundry air	1	1.2	0	26.0	48.0	17.0	8.0	1.0						
Talc milling	1	1.5	0	16.0	32.0	20.0	13.0	7.0	5.0	2.0	2.0	2.0	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	0	1.0	
Marble cutting	1	1.5	0	12.0	37.0	21.0	10.0	11.0	3.0	0	1.0	2.0	2.0	1.0
Soapstone dust	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					

An examination of the data in table 1 discloses a striking difference between the size frequency of outdoor dust and indoor industrial dust. Ninety-seven percent of the outdoor dust particles were found to be of a size less than 1 micron in diameter, with a median size of 0.5 micron. Practically no dust particles larger than 1.5 microns were found to exist in outdoor air. These results on the size frequency of outdoor dust are similar to those obtained by Owens in London (9). In contrast with this result we find that only 2 percent of the industrial dust particles are less than 0.5 micron, and but 21 percent less than 1 micron. The average (median) size of these particles was found to be 1.5 microns. It is evident from the results shown in table 1 that the majority (69 percent) of the dust particles present in industrial atmospheres investigated by the writer was found to be between one and three microns in average diameter, with but 10 percent of the particles exceeding 3 microns.

One of the interesting findings of the present study is revealed by the distribution shown in table 1, which indicates that although no two industrial dusts have the same size frequency, differing for the same dust created by different operations, yet for all practical purposes the dust particles fall into very narrow limits, the majority of them being between 1 and 3 microns. From this evidence on the particle-size distribution of industrial dusts in air it is apparent that our concern should be only for those particles ranging from 0.5 micron to 5 microns, and that the lower limit of particle size may certainly be taken at 0.5 micron.

An application of the size-frequency data presented in table 1 to some of the results obtained in studies of dust concentrations in industry reveals very interesting information. Figure 1 presents a comparison between the number of dust particles of different sizes

found in the general air of granite cutting plants and that found in outdoor air in the vicinity of these plants. The average dust counts for both the granite cutting plants (20.2) and the outdoor air (4.7 millions of particles per cubic foot) are based on about 50 samples obtained with the impinger apparatus. It is obvious that if we apply the size-frequency data shown in table 1 (computed with a class interval of 0.1 micron) to the average dust counts just cited, that in

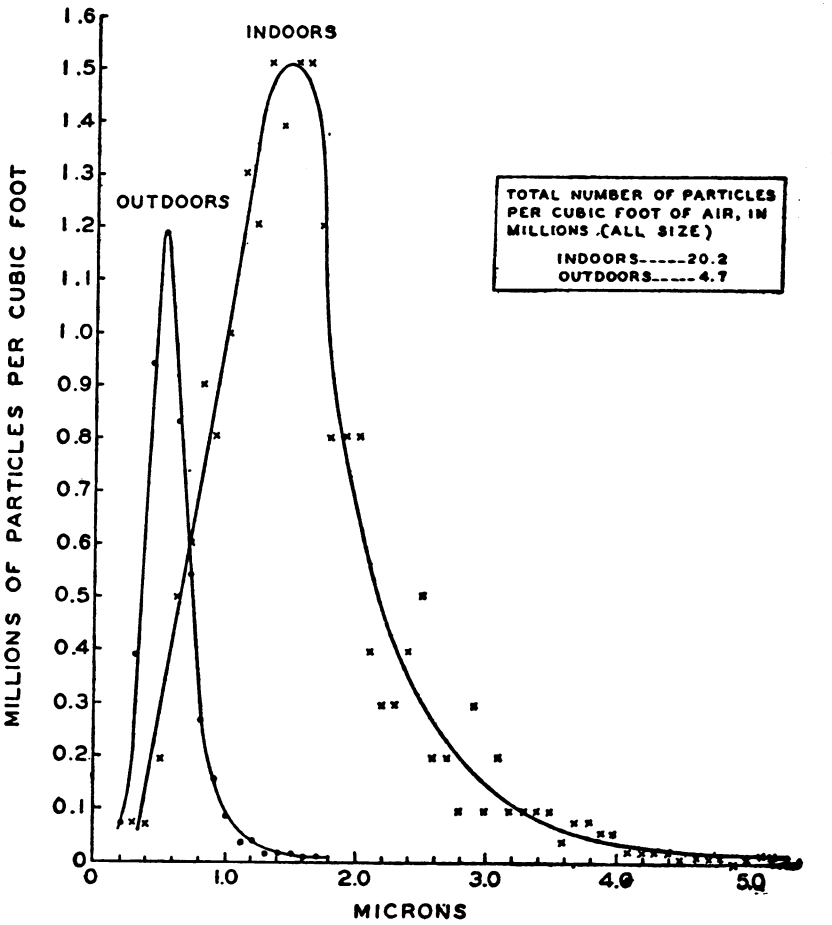

FIGURE 1.—Amount of dust of different sizes found in the general atmosphere of granite cutting plants in comparison with that outdoors in the vicinity of these plants.

the case of the indoor samples we cannot expect to obtain appreciable counts until the 0.7 micron size and larger sizes are reached, whereas apparently the opposite would hold true for the outdoor dust samples. This result is what one would expect from a consideration of the particle-size data of the two types of dusts and is merely presented to illustrate more lucidly the significance of the data. These results serve to indicate very clearly that if we are to differentiate between

dusts present in normal air (not proved to be harmful) and certain dusts found in industrial air (known to be hazardous), we should leave out of consideration those particles that are less than 0.5 micron in diameter. (The two curves actually cross at 0.6 micron.)

THE SAMPLING AND ANALYSIS OF INDUSTRIAL DUSTS

From the data presented in this paper it is apparent that in order to obtain a representative sample of industrial dust in air, one should employ an instrument capable of arresting with a high degree of efficiency all kinds of dust, of sizes ranging from 0.5 micron to 5 microns and at both low and high concentrations. In addition, the method of counting the dust in the samples should have small analytical errors and should reveal only those significant particles present in industrial atmospheres. It should not be the aim to count all the dust particles which may be present in the samples (as may be accomplished by either the use of high magnifications, dark-field illumination, or combinations of both), since it is necessary to differentiate between the dust content in normal air and industrial air. As has already been shown in this paper, this difference is sharply marked insofar as the dust particles between 0.5 micron and 5 microns are concerned; but this difference would be masked and lost should we include in our determination the particles of ultramicroscopic size which are present in vast numbers in all air.

Many methods have been devised and used for the purpose of determining the quantity of dust in air. Suffice it to say that for the purpose of dust sampling in either high or low concentrations, the Greenburg-Smith impinger apparatus (12) now finds universal favor. This instrument has been used by the United States Public Health Service in all of its dust studies for the past 10 years and is also being used by other workers in this country and abroad. Since this instrument has already been described in numerous publications, no further mention will be made at this time concerning construction details or method of operation. However, certain advantages that this instrument possesses over other dust sampling instruments should be mentioned. These are, briefly, a high dust collecting efficiency at both low and high concentrations (98 percent against finely divided silica dust), simplicity of construction, low cost, and finally it permits samples to be examined either microscopically, gravimetrically, or chemically. Recently, Hatch (14), in studying the operating characteristics of the modified impinger developed by him, investigated the effect of particle-size on the sampling efficiency of this instrument. Against a silica-dust suspension of approximately 1.5 microns average diameter, this instrument yielded an efficiency of more than 98 percent at the normal sampling rate of 1 cubic foot per minute. Even against very finely divided magnesium oxide fumes,

formed by burning magnesium ribbon in the flame of a blast lamp, this instrument showed an efficiency of 55 percent.

The method of dust counting employed by us during the past 10 years has been presented in detail elsewhere (12) (15). Recently, in order to establish the lower limit of particle size revealed by our standard microscopic technique, quartz dust particles ranging from 0.4 micron to 1.6 microns and averaging 0.9 micron were examined by this technique. This study showed that with our method of counting dust an experienced observer is capable of seeing quartz dust particles as small as 0.7 micron. Our size-frequency data shows that only 15 percent of the dust in industrial air is less than 0.7 micron. It is obvious, therefore, that our present method of counting dust is capable of disclosing about 85 percent of the dust particles collected by our instrument. The small percentage of dust our method fails to reveal is negligible, when one takes into consideration the simplicity of the method, the fact that results may be checked by trained observers, and that it is one of practical application.

The best criterion of the value of any method of measurement is its successful use in a practical application. Such a test was offered in the study of the health of workers exposed to the inhalation of granite dust (16). In this study it was definitely established that a high correlation existed between the intensity of exposure to dust and the degree of silicosis and active tuberculosis. It is obvious, therefore, that the technique of dust analysis which we have been using constitutes a valuable index of the hazardousness of dust inhalation.

SUMMARY

The results of measurements of 18,000 outdoor dust particles showed that nearly all of these are less than 1 micron in average diameter. The median size was found to be 0.5 micron. In contrast with this result it was found that only 21 percent of about 6,000 industrial dust particles were less than 1 micron in size, the majority (69 percent) being between 1 and 3 microns. The median size of the industrial dust particles was found to be 1.5 microns. These results clearly indicate that in conducting industrial dust studies our concern should be only for those particles ranging in size from 0.5 micron to 5 microns.

The instrument used in sampling industrial dust in air, the standard impinger apparatus, is shown to be capable of collecting, with a high degree of efficiency, dust particles of the sizes found in this study. The standard method used in enumerating dust particles is shown to take into account about 85 percent of the dust present in industrial atmospheres. In addition, our studies have shown that a high correlation exists between dust counts obtained with our technique and the degree of silicosis and tuberculosis found in a study of the health

of granite cutters. The present study clearly indicates that the method used in enumerating dust particles collected by the impinger apparatus constitutes a valuable and practical index of the hazardousness of dust inhalation.

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RELATION OF ARSENOXIDE CONTENT TO TOXICITY OF FRESH AND OLD SAMPLES OF ARSPHENAMINE

New Chemical Tests upon the Arsphenamines

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The arsphenamines are easily oxidized into compounds of the arsenious oxide type, of which 3-amino-4-hydroxylphenyl arsenious oxide (designated as arsenoxide) is one of the primary products. Much work has been done by Voegtlin and coworkers (1) (2) (3) to demonstrate the role of this compound in trypanocidal action and in toxicity to the host of the arsphenamines.

In a recent communication (4) we have described a method for the estimation of arsenoxide based on a color reaction with beta-naphthoquinone under such conditions that the arsphenamines and all other arsenic compounds which we tested reacted negatively. With this method the importance of arsenoxide as an oxidation product of the arsphenamines was demonstrated *in vitro* and *in vivo*. *In vitro* from 30 to 40 percent concentrations could be reached in solutions of the arsphenamines when oxygen was bubbled through them at body temperature. In the animal body it was possible to demonstrate arsenoxide in the liver after injection of arsphenamine and in the kidney after injection of neoarsphenamine.

Since arsenoxide is approximately 10 times as toxic to animals as is arsphenamine, its presence in commercial samples becomes of importance, because the biological standardization of such samples is made upon a basis of toxicity. The presence of arsenoxide is of further significance because of the probable role that it would play in some of the untoward effects encountered in man following the therapeutic administration of arsphenamine.

In the present communication the arsenoxide test was carried out on commercial samples of arsphenamine to investigate two questions: (*a*) The relation between animal toxicity and the amount of arsenoxide in the sample; (*b*) whether arsphenamine becomes more toxic after several years of storage and if so whether such toxicity is due to the development of arsenoxide in the sample.

THE TEST

This test has been previously described (4). For application to commercial samples of arsphenamine the following technique is used:

The arsenoxide is extracted from the powder by dissolving 50 mg in 9.0 cc of water in a stoppered test tube or small flask. Five tenths cc of 50 percent cadmium sulphate is now added and the solution is mixed; 0.45 cc of 0.1 normal sodium hydroxide is then added drop by

drop, mixing the solution several times during the addition by inversion of the tube. The solution is now filtered; the filtrate should be acid to litmus as this prevents precipitation of appreciable amounts of the cadmium. The colorimetric test is now run upon the filtrate without letting it stand for any length of time. Filtrates obtained according to the method of Voegtlin and Smith (1) have also been employed in several instances with satisfactory results.

The test may be made upon 2.5 or 5 cc of the filtrate. To 5 cc in a test tube are added 2 cc of 10 percent sodium cyanide and then 2 cc of a 0.25 percent solution of 1.2 naphthoquinone-4-sodium sulphonate[1] made up in 10 percent sodium thiosulphate. This should be made up 10 minutes before addition. A layer of mineral oil is now run over the top of the solution. After this has stood for 20 minutes, 0.5 cc of 20 percent sodium sulphite is added and direct comparison is made with the standards.

By employing test tubes of the same size the comparison can be made directly with the standard tubes. For the standards five tubes are used, containing from 0.2 to 1.0 mg of arsenoxide in 4.75 cc of water plus 0.25 cc of 50 percent cadmium sulphate in each tube. They are tested simultaneously with the filtrates.

The color produced by the 0.2 mg standard, corresponding to 0.8 percent of arsenoxide in arsphenamine, is too weak for accurate comparison, and this amount of arsenoxide or less has been designated in the tables as a trace.

The procedure was checked by adding up to 4 percent of arsenoxide to several samples of arsenoxide-free arsphenamine and obtaining complete recovery. The cadmium sulphate serves a double purpose, aiding in the precipitation of arsphenamine and intensifying the color reaction. However, the intensification of the color varies directly with the amount of cadmium present, so that this should be approximately the same in the unknown as in the standard solution.

In the previous work it was found that ortho-aminophenol behaved similarly to arsenoxide in giving the color reaction, and a modification of the test with stannous chloride permitted a distinction to be made between these two compounds. We have applied this modification to samples of arsphenamine giving the color reaction and in no instance was o-aminophenol found to be present.

METHOD OF STUDY

The toxicity tests were carried out by one of us (T. F. P.) employing the official procedure regularly used in this laboratory for the biological standardization of these products, as described by Roth (5).

The concentration of the solution, the process of alkalization, the rate of intravenous injection, and the weight and source of the rats

[1] Obtained from the Eastman Kodak Co., Rochester, N.Y.

employed are all accurately controlled. Five rats were employed upon each dosage, and from 3 to 5 doses were necessary for each product. Some of the tests were carried out as part of the official standardization and the upper limits of toxicity (the M.L.D.), beyond the official requirements, were not established in all cases.

In the correlation of arsenoxide content with toxicity of arsphenamine it is necessary first to consider the toxicity of arsphenamine itself. Voegtlin, Johnson, and Dyer (6) have shown a relation between such physical characteristics as viscosity and toxicity and numerous observers have emphasized the importance of the colloidal nature of arsphenamine in its behavior upon intravenous injection (for reviews, see Voegtlin (7); Roth (8)). It is to be expected therefore that the basic toxicity of arsphenamine should show variations. Our studies have served to show that the toxicity of present-day products of arsphenamine which contain no appreciable arsenoxide varies from a M.L.D. of 180 mg to 220 mg per kilogram body weight when tested upon rats in this laboratory. In order to calculate the toxicity of a product containing arsenoxide we have taken 200 mg per kilo as the toxicity of the arsphenamine, and added to this figure the toxicity of the arsenoxide present, in order to arrive at a theoretical value for comparison with the actually determined toxicity. Accepting 200 mg per kilo as the dose of arsphenamine producing death in 80 percent of rats in 3 days, and 20 mg per kilo as the analogous M.L.D. of arsenoxide,[2] the theoretical M.L.D. was calculated according to the formula

$$10\frac{x}{100}M+\frac{100-x}{100}M=200$$

where x is the percentage of arsenoxide and M is the theoretical M.L.D. of the arsphenamine.

RESULTS

In table 1 are shown the results of tests on 35 samples of arsphenamine less than 1½ years old. A sufficiently close agreement was obtained between the theoretical toxicity based upon the arsenoxide content and the actual toxicity upon rats to conclude that the principal factor in causing increased toxicity is arsenoxide. Some variations occur, but they are no greater than would be expected in view of the errors inherent in the method of calculation, in the biological standardization, and in the test itself.

[2] In the previous papers the M.L.D. of arsenoxide was stated to be 26 mg per kilo. This was on a basis of 100 percent mortality. The above value is upon a basis comparable with that of arsphenamine.

TABLE 1.—*The arsenoxide content and toxicity of recent samples of arsphenamine. The theoretical toxicity is computed on a basis of 200 mg per kilo; M.L.D. as the toxicity of the arsphenamine itself plus the toxicity of the arsenoxide present. Under "found toxicity", + indicates that the M.L.D. was not determined, but was above the given value.*

Arsphenamine	Arsenoxide content	Theoretical toxicity	Found toxicity	Arsphenamine	Arsenoxide content	Theoretical toxicity	Found toxicity
Brand A				*Brand E*			
Lot No.—	*Percent*	*mg/kilo, M.L.D.*	*mg/kilo, M.L.D.*	Lot No.—	*Percent*	*mg/kilo, M.L.D.*	*mg/kilo, M.L.D.*
1	(1)	200	220	1	3.5	152	180
2	(1)	200	220	2	3.2	155	
3	(1)	200	180+	3	2.8	159	
4	(1)	200	160+	4	3.3	154	150
5	1.5	176	170–190	5	3.5	152	130–150
6	(1)	200	200	6	2.4	164	130
7	(1)	200	200	7	4.2	145	130
Brand B				8	2.3	165	130–150
Lot No.—				*Brand F*			
1	(1)	200	200+	Lot No.—			
2	(1)	200	160+	1	1.6	174	160+
3	(1)	200	160+	2	1.5	176	180+
Brand C				3	2.0	170	180+
Lot No.—				4	(1)	200	180+
1	3.5	150	140	5	(1)	200	200+
2	2.0	170	140–170	6	(1)	200	200+
3	3.5	150	150	7	1.5	176	180
4	4.0	147	180	*Brand G*			
5	(1)	200	160+	Lot No. 1	3.5	152	160
Brand D				*Brand H*			
Lot No.—				Lot No. 1	1.5	176	150+
1	1.7	173	180				
2	(1)	200	160+				
3	1.6	174	160				

[1] Trace (< 1 percent).

In order to investigate the problem of deterioration of arsphenamine with age, a group of samples was chosen that had been stored in a basement, protected from light, for 3 to 10 years. Eleven products were selected upon which satisfactory toxicity tests had been originally run, and whose original M.L.D. upon rats had been 200 mg or more per kilogram; the presence of arsenoxide in the sample when first tested was therefore unlikely.

Redetermination of toxicity tests upon rats revealed an increased toxicity that was roughly proportionate to the age of the drug. Estimation of the arsenoxide content revealed its presence in every case in sufficient quantity to account for the increase in toxicity (table 2). The effect of age on arsenoxide content is particularly brought out when one compares these samples with fresh samples of similar brands (table 1). One exception to these results was found. The two products of brand D when tested 9 years previously had shown the extremely low toxicities of 250 and 300 mg M.L.D. per kilo. The toxicity of these two samples showed no appreciable change after 9 years and no measurable amounts of arsenoxide could be detected in them. These products also furnish an example of how factors, other than arsenoxide, perhaps physicochemical, can influence the basic toxicity of arsphenamine, in this instance to bring about a product of unusually low toxicity.

TABLE 2.—*The increase in toxicity and presence of arsenoxide in old samples of arsphenamine. In computation of theoretical toxicity, the original M.L.D. was taken as the basic toxicity of the arsphenamine*

	Age of sample	Arsenoxide content	Original toxicity	Actual present toxicity	Theoretical present toxicity
Lot no.— *Brand A*	*Years*	*Percent*			
1	3	1.6			
2	5	1.6	200+	160	174
3	7	1.9	240	210	205
4	9	3.0	240	180	188
5	9	4.5	210	180	150
Lot no.— *Brand B*					
1	6	3.2	200	160	155
2	9	4.0	220	150	161
Brand D					
Lot no. 1	9	4.5	160–180	120	
Lot no.— *Brand F*					
1	9	(1)	250	250	250
2	9	(1)	300	260	300

INFLUENCE OF ALKALIZATION AND OF STANDING

Several experiments were conducted to study the influence of alkalization upon the arsenoxide content of arsphenamine. The standard method of alkalization was carried out, and the naphthoquinone test was performed upon the solution after reacidification with an equivalent quantity of hydrochloric acid.

It is seen from the results (table 3) that some increases in arsenoxide were present as a result of alkalization. When the alkaline solutions were permitted to stand in stoppered glass cylinders for 2 hours at room temperature, still further increases up to 2 percent were observed. The greatest increases (2 percent) occurred in two samples that were 3 and 5 years old; and this suggests that with some products old samples may be more unstable than fresh ones. Fresh samples that contained up to 4.2 percent of arsenoxide did not show any significant increases after the alkaline solutions had stood for 2 hours. However, more extensive tests must be made to establish these findings.

TABLE 3.—*The effect of alkalization and of 2 hours standing in stoppered cylinders of alkaline solutions of arsphenamine on the arsenoxide content*

Brand	Age	Arsenoxide content		
		Acid solution	Freshly alkaline solution	2 hours' standing
A. No.—		*Percent*	*Percent*	*Percent*
1	2 months	(1)	1.5	1.6
2	3 years	1.6	2.0	3.5
3	5 years	1.6	1.6	3.6
C. No.—				
1	1 year	3.5	4.2	4.2
2	2 months	1.2	1.5	2.6
E. No.—				
1	4 years	4.0	4.0	4.2
2	1 year	4.0	4.0	4.0
3	9 months	2.8	2.8	2.8
4	2 months	4.2	4.2	
5	6 months	2.4	2.2	2.8
F. No. 1	2 months	(1)	1.3	1.6

[1] Trace (<1%).

TESTS UPON NEOARSPHENAMINE

An attempt was made to apply the test for arsenoxide to neoarsphenamine. It was found, however, that in all samples there were present larger quantities of byproducts of a strongly reducing nature which inhibited the naphthoquinone color reaction. Up to the present time, various types of precipitation, including acids, alcohols, and heavy metals, singly or combined, have not proved satisfactory, as shown by incomplete recovery of added arsenoxide. From a considerable amount of work with these imperfect methods, the indications are that insufficient arsenoxide is present in commercial samples fully to account for variations in toxicity. This is in accord with the previous experiments on oxidized solutions of neoarsphenamine (4).

During this work a color reaction was observed which seemed worthy of further study. Certain samples of neoarsphenamine give a color from brown to deep red with a saturated solution of lead acetate. The reaction is observed chiefly in those samples of neoarsphenamine with sulpharsphenamine-like properties and also in sulpharsphenamine. The degree of color depends on the concentration of both constituents; we have adjusted the procedure so that in one concentration sulpharsphenamine reacts negatively and in another positively. Most fresh products of neoarsphenamine react negatively in all proportions.

(1) To 50 mg of neoarsphenamine or sulpharsphenamine in a small dry test tube add 1 cc of a saturated aqueous solution of lead acetate (sugar of lead) and stir immediately with a stirring rod. Sulpharsphenamine and typical neoarsphenamine remain light in color and do not dissolve.

(2) To 100 mg of powder add 0.5 cc of lead acetate and stir similarly. Sulpharsphenamine goes into solution with a deep red color. Typical neoarsphenamine remains light in color. Other samples vary from a brown coloration of the powder to a behavior similar to sulpharsphenamine.

That this procedure detects oxidation products in neoarsphenamine is indicated by the fact that certain products develop the color if the ampoules are left at 56° C. for 1 or more days. Analysis of the powder before and after heating, by Elvove's method (9), indicated that an oxidation had resulted from the heating.

Various chemicals were tried with the lead acetate solution to determine whether the color reaction resulted from byproducts present. All reacted negatively except sodium hydrosulphite ($Na_2S_2O_4$), which gave a red precipitate, changing to black lead sulphide within 10 minutes. The behavior was different from that of neoarsphenamine and sulpharsphenamine.

A SIMPLE PROCEDURE FOR DIFFERENTIATING THE ARSPHENAMINES

Occasionally it becomes necessary to identify a type of arsphenamine. The following procedure has satisfactorily fulfilled this requirement in a large number of samples which we have tested. It is carried out with the reagents ordinarily used for precipitation of blood by the Folin-Wu method of analysis.

To a small quantity of the powder, approximately 50 mg, in 5 cc of water, add 1 cc of 10 percent sodium tungstate; mix, and add 1 cc of 2/3 normal sulphuric acid.

Arsphenamine gives a bulky yellow precipitate. Sulpharsphenamine stays in solution for several hours without change in color, while neoarsphenamine develops a persistent deep blue-green color within a few minutes.

COMMENT

The toxicity of arsphenamine has been the subject of considerable investigation, and no uniformity of opinion exists as to the factors involved. In this laboratory the investigations of Voegtlin and coworkers have served to emphasize the importance, on the one hand, of physical characteristics (viscosity) and, on the other hand, of chemical changes with oxidation to arsenoxide.

In our previous work with the naphthoquinone test for arsenoxide the enhanced toxicity to rats of oxidized solutions of arsphenamine could be fully explained by the formation of arsenoxide, and that of sulpharsphenamine and neoarsphenamine could be partially so explained. The results upon commercial samples of arsphenamine and neoarsphenamine are in accord with these findings.

From a point of view of usefulness in testing arsphenamine for clinical use, the estimation of arsenoxide should be of value either as a supplement to animal toxicity or for use where the latter is not feasible. Concentrations of arsenoxide above 2.5 percent would be undesirable.

The demonstration that arsphenamine can deteriorate after several years of storage would emphasize the value of placing the date of release upon the ampoules. The process of deterioration is sufficiently slow as to involve no hardship on the manufacturer. Roth (10) has produced evidence of similar deterioration with neoarsphenamine.

The tungstic-acid color test is described as a simple means of differentiating between arsphenamine, neoarsphenamine, and sulpharsphenamine, with reagents available in most clinical laboratories.

While the lead acetate test upon neoarsphenamine could not be correlated with toxicity tests, it indicates chemical differences that cannot always be detected by other means. The methods of analysis of neoarsphenamine and sulpharsphenamine developed by Elvove (9) have demonstrated the wide variability among products, and it is

hoped that procedures which detect chemical differences may lead to a greater uniformity of manufacture. This is highly desirable for a drug of the importance of neoarsphenamine, particularly in view of the fact that it is administered by intravenous injection.

SUMMARY

The arsenoxide content and toxicity of 35 recent samples of arsphenamine were determined. A correlation sufficiently close was obtained to attribute the enhanced toxicity of certain products chiefly to arsenoxide.

Eleven samples of arsphenamine from 3 to 10 years of age, originally of low toxicity, were studied. With the exception of one product, an increase of toxicity roughly proportionate to the age was found. Sufficient arsenoxide was present to account for the increases in toxicity.

A color reaction between neoarsphenamine and lead acetate is described which detects chemical differences between various products.

A simple test to distinguish between arsphenamine, neoarsphenamine, and sulpharsphenamine, is described.

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STUDIES OF THE POLLUTION AND NATURAL PURIFICATION OF STREAMS

A Resurvey of the Ohio River Between Cincinnati, Ohio, and Louisville, Ky., with a Discussion of the Changes Since 1914–16 and the Effects of Canalization

During the period November 1929 to May 1931 the United States Public Health Service, in cooperation with the Kentucky State Board of Health and the Sewerage Commission of the City of Louisville, made a sanitary survey of the Ohio River between Cincinnati and Louisville in order to determine the changes in sanitary conditions since an investigation made some 15 years previously and to study

the effects of canalization of the river on the phenomena of natural stream purification. The results of this investigation have recently been made available in Public Health Bulletin No. 204.

The study included the collection of nearly 2,000 water samples for chemical and bacteriological examination; the tabulation of stream discharge, with estimates of times of flow; a study of existing sources of pollution, including the distribution of total and sewered population, and the waste-producing industries on the watershed.

Changes affecting the sanitary status of the river between Cincinnati and Louisville since the 1914–16 survey include the construction of 2 additional dams, making 4 dams between the 2 cities; an increase of 8 feet in the elevation of the dam at Louisville, lengthening the time of flow through the pool above that city; an increase in total population on the watershed above Cincinnati of 1,227,000 and between Cincinnati and Louisville of 433,000; an increase in sewered population within the zone of investigation of 212,000, making a total of 890,000 persons contributing untreated sewage to this section of the stream; and a possible decrease in industrial waste pollution because of the elimination of brewery and distillery waste and curtailed activity in other industrial establishments since the previous investigation.

The field and laboratory studies were conducted along lines similar to those of the original survey, and all procedures were closely coordinated and standardized, making the results of both investigations entirely comparable.

A study of the data collected has indicated that during winter periods of unobstructed flow, when stream discharge and channel conditions were essentially the same during both investigations, the amount of dissolved oxygen at the present time was approximately the same, the oxygen demand about one half, and the bacterial pollution somewhat higher, as compared with the winter periods of 1914–16.

In the present summer periods of pool stage, the average dissolved oxygen and the oxygen demand were both found to be less than in 1914–16, but the ratio of available to required oxygen was practically the same in both periods. However, during certain days in August 1930, when stream flows decreased to about 2,000 second-feet, the dissolved oxygen below Cincinnati was completely exhausted, indicating the marked effect of sewage and probably sludge deposits in the pool at Cincinnati.

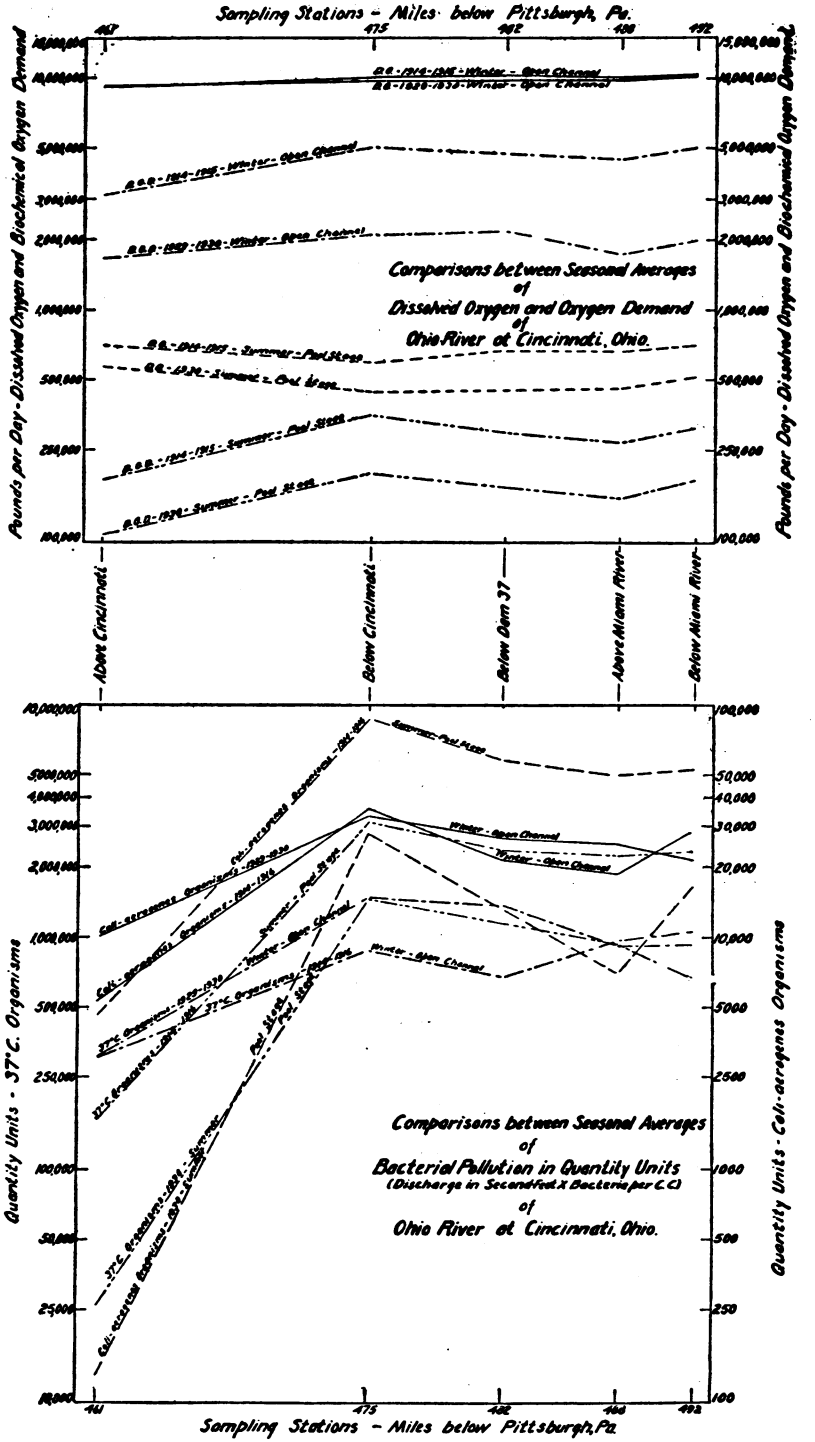
At the Cincinnati and Louisville waterworks intakes the bacterial concentration in the raw water was such that these plants, with highly elaborated processes of treatment, combined with efficient operation and supervision, were able to produce, without difficulty during a large part of the time, an effluent conforming to the Treasury Department standard for drinking water. The removal of turbidity in the pools, resulting in increased numbers of microscopic plants, during

pool stages, resulted in shortened filter runs and taste and odor troubles in the filter effluents.

The summer and winter rates of bacterial decrease below Cincinnati in 1929–30 were quite similar, as were also the 1929–30 and 1914–16 winter rates under comparable stream flow and channel conditions. During 1930, with much longer times of flow, due to complete canalization, rates of bacterial decrease were much less rapid than those of the summer months of 1914–16, with partial canalization. Decreasing velocities apparently lowered the bacterial concentration immediately below the zone of pollution, and the lower initial concentrations seemed to result in decreased rates of change further downstream. Initial concentration, however, does not appear to account entirely for differences in bacterial changes, and mechanical removal by sedimentation, differences in the condition or types of organisms present after long periods of flow, or the presence of larger microscopic organisms may be factors which influence rates of change under these conditions.

At times of unobstructed flow in the river a secondary increase in bacteria was indicated below the mouth of the Great Miami River, a tributary entering the main stream about 16 miles below the lower Cincinnati sewers, suggesting that rates of bacterial change may be affected by dilution, which disturbs, for a time at least, the biological balance of the stream.

Canalization, resulting in decreased velocities through the several pools, permits the sedimentation of sewage solids, which materially decreases the dissolved oxygen, with the possible creation of nuisances in the vicinity of the sewer outlets rather than at points downstream. These same conditions reduce considerably the bacterial load below the sources of pollution. While decreased bacterial loads at the waterworks intakes make the production of a satisfactory effluent less difficult, the reduced turbidity and increased numbers of microscopic organisms, resulting in shortened filter runs and possible taste and odor troubles in the treated water, more than offset the good effects of decreased bacterial loads. The concentration of decomposition products due to the fermentation of organic deposits within the pools may have public-health aspects not heretofore appreciated. Reference is made to the suspected water-borne outbreak of gastroenteritis along the Ohio River at the time of and following the low flows in the fall and winter of 1930–31. Canalization, at least between Cincinnati and Louisville, appears to have had the tendency to complicate, rather than simplify, the problems connected with sewage disposal, nuisance production, the operation of water-treatment devices, and the preservation of public health.



Comparison of the data secured by the two surveys

COURT DECISION RELATING TO PUBLIC HEALTH

State law and county ordinance regulating establishment of cemeteries held valid.—(Maryland Court of Appeals; *Gordon* v. *Commissioners of Montgomery County*, 164 A. 676; decided Feb. 15, 1933.) A legislative act of 1927 authorized the commissioners of Montgomery County to license and regulate graveyards within the county, and, in order to safeguard the public health, safety, and welfare, to pass ordinances for the purpose of executing the powers granted by the act. Proper standards for the exercise of the discretion conferred were to be contained in such ordinances, and there was to be uniform operation. The act declared unlawful the establishment or conduct of a cemetery without a license from the county commissioners, if required by any authorized ordinance. In pursuance of such law, the county commissioners passed an ordinance requiring a permit from them before any cemetery should be established or operated, and providing that no cemetery should be so located as to permit drainage of water into any well, spring, etc., used for drinking purposes by human beings, or as to endanger the safety and health of residents in the community. The ordinance also provided that no cemetery should be established within 500 yards of any school, hospital, sanitarium, or orphan asylum, or in any part of the county where there were then residing more than 100 persons within a radius of 500 yards of the outside limits of the burial reservation. Whenever, in the commissioners' opinion, the location of a cemetery would endanger the health, welfare, or safety of the public in its vicinity, a permit for its establishment there was to be refused.

The plaintiff brought an injunction suit to prevent the county commissioners from interfering with his effort to open and operate a cemetery in Montgomery County without applying for and obtaining a permit. It was his theory that the statute and ordinance were invalid and that he was therefore justified in disregarding their requirements. The contention was made that the county commissioners could not be constitutionally invested by the legislature with the power which the ordinance proposed to exercise.

The court of appeals held that the regulation of cemeteries in the interest of the public health was within the police power of the State and that the exercise of that power could be validly delegated by the legislature to a municipal corporation or other qualified agency of local government. "It is clearly within the powers and duties", said the court, "which the constitution declares may be conferred upon county commissioners."

The act under consideration was stated by the court not to attempt to confer upon the county an indefinite and wholly discretionary authority to grant or refuse permission to locate cemeteries, but required that the regulations on the subject should contain proper

standards to safeguard the public health and welfare and to govern uniformly the use of the delegated power. The standards prescribed by the commissioners were held to be clearly appropriate and adequate to fulfill the conditions under which the police power, when delegated by the State, could be validly applied by such a governmental agency as a county. Said the court:

* * * Consideration is required to be given to the question of drainage, the number of adjacent residents, and the proximity of public institutions, within a specified distance, all with the distinct view to the protection of the public health. The only indicated ground for refusing a permit is a conclusion that the projected cemetery, in the location proposed, would endanger the health, welfare, or safety of the public in its vicinity. Proper compliance with the standards defined by the ordinance is an assurance against arbitrary discriminations or abuses of discretion. It is to be presumed that the action of the county commissioners on applications for cemetery permits will not disregard but will duly observe the limitations which the ordinance has imposed. * * *

As to the argument that the act was an invalid extension of the commissioners' authority into the field of legislative action, contrary to the historical limitation of their powers and in disregard of the home rule amendment to the State constitution, the court said that the fact that a county may formerly have been granted more limited legislative functions was far from being a conclusive reason why their powers could not be enlarged, and that the home rule amendment had no effect upon the question then being determined, as the privilege afforded by the amendment had not been exercised in Montgomery County.

The court closed its opinion as follows:

The plaintiff was obligated by the terms of a valid statute and ordinance to obtain a permit from the County Commissioners of Montgomery County before proceeding to locate the cemetery which he has planned, and his suit for an injunction to aid him in that purpose, without any application having been made for such a permit, is not maintainable. The demurrer to the bill of complaint was correctly sustained for that reason. Leave to amend not having been utilized, the bill was properly dismissed.

DEATHS DURING WEEK ENDED JULY 22, 1933

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

	Week ended July 22, 1933	Corresponding week 1932
Data from 85 large cities of the United States:		
Total deaths	6,772	7,549
Deaths per 1,000 population, annual basis	9.5	10.8
Deaths under 1 year of age	504	593
Deaths under 1 year of age per 1,000 estimated live births (81 cities)	42	49
Deaths per 1,000 population, annual basis, first 29 weeks of year	11.3	11.8
Data from industrial insurance companies:		
Policies in force	67,722,700	71,774,641
Number of death claims	11,151	11,998
Death claims per 1,000 policies in force, annual rate	8.6	8.7
Death claims per 1,000 policies, first 29 weeks of year, annual rate	10.3	10.0

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

Reports for Weeks Ended July 29, 1933, and July 30, 1932

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended July 29, 1933, and July 30, 1932

Division and State	Diphtheria		Influenza		Measles		Meningococcus meningitis	
	Week ended July 29, 1933	Week ended July 30, 1932	Week ended July 29, 1933	Week ended July 30, 1932	Week ended July 29, 1933	Week ended July 30, 1932	Week ended July 29, 1933	Week ended July 30, 1932
New England States:								
Maine	-	1	4	-	2	22	0	0
New Hampshire	-	-	-	-	23	3	0	0
Vermont	-	2	-	-	8	7	0	0
Massachusetts	19	37	-	-	120	147	2	1
Rhode Island	-	1	-	2	-	7	0	0
Connecticut	4	3	2	1	22	34	0	0
Middle Atlantic States:								
New York	39	30	[1] 1	[1] 4	261	445	7	3
New Jersey	12	15	3	1	77	141	0	0
Pennsylvania	26	31	-	-	228	184	5	5
East North Central States:								
Ohio	11	24	30	6	10	87	1	1
Indiana	10	26	19	19	10	7	1	8
Illinois	11	26	19	16	50	73	3	2
Michigan	20	16	3	-	87	293	0	1
Wisconsin	3	4	10	12	38	101	0	2
West North Central States:								
Minnesota	7	3	1	4	24	17	0	2
Iowa [2]	2	6	-	-	4	3	2	0
Missouri	19	12	-	2	21	13	1	1
North Dakota	2	6	-	-	20	5	3	1
South Dakota	-	-	-	-	-	-	1	0
Nebraska	3	3	-	-	-	4	0	0
Kansas	7	6	-	-	12	16	0	0
South Atlantic States:								
Delaware	-	-	-	-	-	-	1	0
Maryland [2 3]	3	6	9	-	11	7	0	0
District of Columbia [3]	2	9	1	2	4	2	0	0
Virginia [3 4]	7	9	-	-	18	37	0	0
West Virginia	10	7	1	-	3	51	0	2
North Carolina [3 4]	20	22	3	26	51	79	0	0
South Carolina [4]	8	8	72	74	33	24	0	2
Georgia [4]	19	8	-	10	21	-	0	0
Florida [4]	1	5	1	-	15	2	0	0
East South Central States:								
Kentucky	6	8	-	-	-	-	3	0
Tennessee	8	3	4	2	25	2	0	1
Alabama [4]	11	19	4	4	10	1	1	1
Mississippi [3]	10	7	-	-	-	-	0	0

See footnotes at end of table.

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended July 29, 1933, and July 30, 1932—Continued

Division and State	Diphtheria		Influenza		Measles		Meningococcus meningitis	
	Week ended July 29, 1933	Week ended July 30, 1932	Week ended July 29, 1933	Week ended July 30, 1932	Week ended July 29, 1933	Week ended July 30, 1932	Week ended July 29, 1933	Week ended July 30, 1932
West South Central States:								
Arkansas	4	5	1	2	27		0	0
Louisiana	8	13	9	1	20	31	1	0
Oklahoma [5]	9	17	8	7	18	5	1	0
Texas [4]	57	36	63	38	69	5	3	1
Mountain States:								
Montana [3]	1	1			8	56	0	0
Idaho [3]		3	3		1		1	0
Wyoming [3]	1				9	3	0	0
Colorado [3]	4	6			5	2	0	0
New Mexico	1	9			3	1	1	1
Arizona	1	2		2	2		1	0
Utah [3]	2				23	2	0	0
Pacific States:								
Washington		1			25	8	0	0
Oregon	1		9	7	28	14	0	0
California	37	26	18	32	169	54	1	2
Total	426	491	298	274	1,624	1,995	40	37

Division and State	Poliomyelitis		Scarlet fever		Smallpox		Typhoid fever	
	Week ended July 29, 1933	Week ended July 30, 1932	Week ended July 29, 1933	Week ended July 30, 1932	Week ended July 29, 1933	Week ended July 30, 1932	Week ended July 29, 1933	Week ended July 30, 1932
New England States:								
Maine	0	1	6	5	0	0	2	3
New Hampshire	0	0	10	6	0	0	0	0
Vermont	0	0	3	2	0	0	1	0
Massachusetts	24	1	85	105	0	0	4	5
Rhode Island	0	1	6	9	0	0	0	1
Connecticut	2	0	14	19	0	0	2	1
Middle Atlantic States:								
New York	45	6	90	122	0	4	22	31
New Jersey	4	2	34	43	0	0	11	7
Pennsylvania	8	19	122	116	0	0	29	30
East North Central States:								
Ohio	3	5	124	96	0	0	31	56
Indiana	0	0	10	20	0	3	19	28
Illinois	7	10	89	73	2	14	25	36
Michigan	3	2	76	75	1	1	7	11
Wisconsin	0	4	20	12	2	0	2	9
West North Central States:								
Minnesota	10	3	9	22	2	0	0	1
Iowa [2]	0	3	10	10	4	4	1	4
Missouri	1	0	15	29	0	2	23	40
North Dakota	5	1		2	1	9	0	5
South Dakota	0	0	2	1	0	2	2	3
Nebraska	0	0	2	1	1	3	0	1
Kansas	1	1	12	13	0	2	6	19
South Atlantic States:								
Delaware	0	0			0	0	1	3
Maryland [2 3]	0	0	29	16	0	0	15	23
District of Columbia [3]	0	0	3	7	0	0	4	4
Virginia [3 4]	1	2	20	11	0	0	55	55
West Virginia	2	1	7	4	0	0	25	50
North Carolina [3 4]	0	2	24	35	0	2	33	59
South Carolina [4]	0	0	3	1	0	0	36	56
Georgia [4]	0	0	3	5	0	0	50	77
Florida [4]	2	0	1	2	0	0	7	5
East South Central States:								
Kentucky	1	2	6	20	0	6	116	108
Tennessee	7	1	12	7	0	3	86	141
Alabama [4]	0	0	12	7	0	0	43	29
Mississippi [2]	0	1	5	5	0	2	8	39

See footnotes at end of table.

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended July 29, 1933, and July 30, 1932—Continued

Division and State	Poliomyelitis		Scarlet fever		Smallpox		Typhoid fever	
	Week ended July 29, 1933	Week ended July 30, 1932	Week ended July 29, 1933	Week ended July 30, 1932	Week ended July 29, 1933	Week ended July 30, 1932	Week ended July 29, 1933	Week ended July 30, 1932
West South Central States:								
Arkansas	0	2	6	----	0	4	23	29
Louisiana	1	0	6	4	0	0	28	71
Oklahoma [5]	0	0	8	8	0	1	39	48
Texas [4]	1	5	21	23	18	8	103	40
Mountain States:								
Montana [3]	0	0	1	2	0	4	5	4
Idaho [3]	0	0	2	1	1	0	4	6
Wyoming [3]	1	0	4	2	0	0	1	0
Colorado [3]	0	0	6	8	0	0	1	5
New Mexico	0	0	----	3	0	0	1	16
Arizona	0	0	2	1	0	2	3	2
Utah [2]	0	0	1	----	0	0	0	1
Pacific States:								
Washington	0	1	8	14	1	5	3	4
Oregon	0	1	4	6	3	4	7	3
California	4	6	61	39	5	9	9	10
Total	133	83	994	1,012	41	94	893	1,179

[1] New York City only.
[2] Week ended earlier than Saturday.
[3] Rocky Mountain spotted fever, week ended July 29, 1933, 14 cases as follows: Maryland, 3; District of Columbia, 1; Virginia, 1; North Carolina, 3; Montana, 2; Idaho, 1; Wyoming, 2; Colorado, 1.
[4] Typhus fever, week ended July 29, 1933, 80 cases as follows: North Carolina, 2; Virginia, 3; South Carolina, 5; Georgia, 16; Florida, 4; Alabama, 27; Texas, 23.
[5] Exclusive of Oklahoma City and Tulsa.

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	Meningococcus meningitis	Diphtheria	Influenza	Malaria	Measles	Pellagra	Poliomyelitis	Scarlet fever	Smallpox	Typhoid fever
June 1933										
Alabama	5	37	23	184	137	97	2	37	4	65
California	13	133	69	7	3,375	9	12	486	63	38
Idaho	----	3	1	----	22	----	0	4	8	8
Kansas	2	17	1	2	492	----	1	72	5	32
Montana	----	----	8	----	118	----	0	47	1	21
Nevada	----	----	----	----	4	----	----	----	4	----
North Carolina	4	36	27	----	1,497	213	1	99	1	112
Oklahoma [1]	2	19	41	110	337	12	1	30	7	70
Oregon	1	7	62	1	138	----	0	67	72	10
Puerto Rico	----	44	54	2,498	177	3	0	----	0	57
Texas	8	188	316	728	----	81	3	134	----	156
Virginia	2	29	60	13	710	45	1	110	----	71
Washington	2	13	25	----	272	----	1	82	31	13
Wisconsin	3	20	54	----	834	----	1	317	26	8

[1] Exclusive of Oklahoma City and Tulsa.

June 1933

	Cases
Actinomycosis:	
California	1
Botulism:	
Washington	3
Chicken pox:	
Alabama	18
California	1,555
Idaho	13
Kansas	91
Montana	70

June 1933—Continued

	Cases
Chicken pox—Contd.	
Nevada	13
North Carolina	116
Oklahoma [1]	17
Oregon	75
Puerto Rico	39
Virginia	195
Washington	606
Wisconsin	1,924
Conjunctivitis:	
Oklahoma [1]	1

June 1933—Continued

	Cases
Diarrhea and dysentery:	
Virginia	1,384
Dysentery:	
California, amebic	3
California, bacillary	9
Oklahoma [1]	36
Puerto Rico	252
Filariasis:	
Puerto Rico	5
Food poisoning:	
California	32

[1] Exclusive of Oklahoma City and Tulsa.

June 1933—Continued

	Cases
German measles:	
California	52
Kansas	85
North Carolina	4
Washington	15
Granuloma, coccidioidal:	
California	1
Impetigo contagiosa:	
Montana	7
Oklahoma [1]	1
Oregon	7
Washington	4
Leprosy:	
Puerto Rico	1
Lethargic encephalitis:	
Alabama	4
California	1
Texas	3
Virginia	2
Washington	3
Wisconsin	1
Mumps:	
Alabama	38
California	877
Idaho	4
Kansas	107
Montana	4
Nevada	1
Oklahoma [1]	5
Oregon	5
Puerto Rico	19
Virginia	71
Washington	118
Wisconsin	254
Ophthalmia neonatorum:	
Alabama	2
California	4
North Carolina	2
Oklahoma [1]	1
Oregon	1
Puerto Rico	7
Virginia	3
Paratyphoid fever:	
California	2
Idaho	1
Kansas	3
Montana	1
North Carolina	2

June 1933—Continued

	Cases
Paratyphoid fever—Con.	
Puerto Rico	7
Texas	13
Virginia	12
Psittacosis:	
California	1
Puerperal septicemia:	
Puerto Rico	7
Rabies in animals:	
California	49
Washington	19
Rabies in man:	
Oklahoma [1]	2
Rocky Mountain spotted fever:	
California	7
Idaho	10
Montana	19
Nevada	3
Oregon	16
Virginia	4
Washington	1
Scabies:	
Oklahoma [1]	1
Oregon	11
Septic sore throat:	
California	7
Kansas	2
Montana	7
North Carolina	8
Oklahoma [1]	14
Oregon	3
Virginia	6
Tetanus:	
Alabama	8
California	8
Kansas	1
Oklahoma [1]	1
Puerto Rico	7
Virginia	4
Tetanus, infantile:	
Puerto Rico	17
Tick paralysis:	
Montana	1
Trachoma:	
California	4
Montana	1
Oklahoma [1]	5

June 1933—Continued

	Cases
Trachoma—Continued.	
Puerto Rico	2
Virginia	1
Washington	1
Trichinosis:	
California	5
Tularaemia:	
California	2
Idaho	2
Kansas	2
Montana	14
Nevada	1
Virginia	3
Washington	1
Typhus fever:	
Alabama	56
North Carolina	4
Undulant fever:	
Alabama	5
California	7
Idaho	1
Kansas	12
Montana	3
Nevada	1
North Carolina	1
Oregon	1
Virginia	1
Washington	2
Wisconsin	7
Vincent's angina:	
Kansas	6
Montana	2
Oklahoma [1]	1
Oregon	8
Washington	2
Whooping cough:	
Alabama	195
California	1,471
Idaho	8
Kansas	365
Montana	25
Nevada	5
North Carolina	972
Oklahoma [1]	43
Oregon	19
Puerto Rico	120
Virginia	368
Washington	57
Wisconsin	1,322

[1] Exclusive of Oklahoma City and Tulsa.

WEEKLY REPORTS FROM CITIES

City reports for week ended July 22, 1933

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								
Maine:											
Portland	1		0	0	2	0	0	0	0	4	14
New Hampshire:											
Concord	0		0	0	0	0	0	0	0	0	8
Nashua	0		0	0	0	0	0	0	0	0	0
Vermont:											
Barre	0		0	6	0	0	0	3	0	4	4
Burlington	0		0	0	0	0	0	0	0	0	10
Massachusetts:											
Boston	4		0	79	12	35	0	12	0	40	158
Fall River	0		0	3	1	6	0	1	0	20	32
Springfield	0		0	0	0	6	0	2	1	5	28
Worcester	3		0	20	0	3	0	4	2	5	45
Rhode Island:											
Pawtucket	0		0	0	0	0	0	0	0	0	12
Providence	8		0	0	2	2	0	3	1	35	49
Connecticut:											
Bridgeport	0		0	3	1	5	0	1	0	5	29
Hartford	2		0	3	0	6	0	0	0	0	29
New Haven	0		0	1	1	0	0	1	0	4	34

City reports for week ended July 22, 1933—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								
New York:											
Buffalo	2		1	24	8	5	0	6	1	20	139
New York	22	1	2	103	59	35	0	83	31	116	1,225
Rochester	0		0	0	0	1	0	0	1	22	58
Syracuse	0		0	0	2	2	0	0	0	4	31
New Jersey:											
Camden	0		0	0	0	3	0	1	1	0	17
Newark	0	1	0	6	4	3	0	1	3	37	64
Trenton	0		0	13	1	3	0	0	3	7	19
Pennsylvania:											
Philadelphia	2		1	101	16	38	0	23	4	11	330
Pittsburgh	1		1	1	2	15	0	7	2	88	133
Reading	1		0	0	1	0	0	3	0	1	24
Ohio:											
Cincinnati	0	1	0	5	0	9	0	10	4	13	96
Cleveland	1	12	0	2	7	17	0	13	0	40	144
Columbus	3		0	0	2	12	0	3	0	0	72
Toledo	2		0	8	2	17	0	3	0	14	61
Indiana:											
Fort Wayne	3		0	0	4	0	0	1	1	0	16
Indianapolis	1		0	12	5	2	0	3	0	5	
South Bend	0		0	1	1	0	0	0	0	1	16
Terre Haute	0		0	0	0	1	0	0	1	0	26
Illinois:											
Chicago	1	1	0	55	14	60	0	36	2	107	558
Springfield	0		0	0	0	0	0	0	0	0	18
Michigan:											
Detroit	4	3	0	5	4	17	0	24	3	124	246
Flint	3		0	1	1	2	0	0	1	7	18
Grand Rapids	0		0	1	0	1	0	0	0	5	36
Wisconsin:											
Kenosha	0		0	1	0	0	0	0	0	20	6
Madison	0		0	0		0	0		0	7	
Milwaukee	0		0	0	2	10	0	5	0	136	81
Racine	0		0	0	1	0	0	0	0	40	10
Superior	0		0	0	0	0	0	0	0	7	4
Minnesota:											
Duluth	0		0	10	0	0	0	0	0	26	14
Minneapolis	2		0	4	2	7	0	2	0	12	83
St. Paul	0		0	5	1	4	0	1	0	55	53
Iowa:											
Des Moines	0			0		1	0		0	0	27
Sioux City	0			1		0	0		0	3	
Waterloo	0			0		0	0		0	3	
Missouri:											
Kansas City	1		0	0	5	3	0	6	1	13	74
St. Joseph	0		0	0	2	1	0	1	0	0	20
St. Louis	11			8	3	5	0	4	4	29	190
North Dakota:											
Fargo	0		0	0	0	1	0	0	0	0	1
South Dakota:											
Aberdeen	0		0	0	0	0	0	0	0	0	
Nebraska:											
Omaha	0		0	6	0	3	1	2	0	9	47
Kansas:											
Topeka	0		0	1	1	0	0	0	0	1	16
Wichita	0		0	0	1	0	0	0	0	14	27
Delaware:											
Wilmington	0		0	1	1	0	0	0	0	11	27
Maryland:											
Baltimore	0	1	1	0	10	16	0	17	3	76	175
Cumberland	0		0	0	0	0	0	1	0	0	14
Frederick	0		0	0	0	0	0	0	0	0	2
District of Col.:											
Washington	0	1	0	12	4	2	0	8	0	6	134
Virginia:											
Lynchburg	1		0	8	0	2	0	0	0	32	5
Norfolk	0		0	2	2	0	0	1	6	0	36
Richmond	0		0	2	1	0	0	3	2	10	39
Roanoke	0		0	0	0	1	0	0	1	2	19
West Virginia:											
Charleston	1		0	0	2	0	0	0	1	1	22
Wheeling	0		0	0	1	1	0	0	0	6	13
North Carolina:											
Raleigh	0		0	0	0	0	0	0	0	1	10
Wilmington	0		0	0	0	0	0	0	0	0	9
Winston-Salem	0		0	0	0	2	0	0	0	2	11

City reports for week ended July 22, 1933—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								
South Carolina:											
Charleston	0	5	0	0	2	0	0	4	3	6	19
Columbia	0	-------	0	0	2	0	0	0	0	0	17
Greenville	0	-------	0	0	0	0	0	0	1	0	9
Georgia:											
Atlanta	4	10	0	4	4	1	0	2	6	10	70
Brunswick	0	-------	0	0	0	0	0	0	0	0	2
Savannah	-------	-------	-------	-------	-------	-------	-------	-------	-------	-------	-------
Florida:											
Miami	2	-------	0	0	2	0	0	1	0	4	27
Tampa	0	-------	0	0	1	0	0	1	0	0	24
Kentucky:											
Ashland	0	-------	0	0	0	0	0	0	1	10	1
Lexington	0	-------	0	1	1	0	0	0	0	1	12
Louisville	1	-------	0	0	3	1	0	5	3	3	74
Tennessee:											
Memphis	0	-------	3	15	7	1	0	5	1	14	81
Nashville	0	-------	0	1	3	2	0	1	0	13	43
Alabama:											
Birmingham	0	-------	0	1	1	4	0	4	1	2	44
Mobile	3	-------	0	2	0	0	0	0	1	0	24
Montgomery	0	-------	-------	0	-------	0	0	-------	1	3	-------
Arkansas:											
Fort Smith	0	-------	-------	0	-------	0	0	-------	0	1	-------
Little Rock	0	-------	0	0	4	0	0	3	0	0	7
Louisiana:											
New Orleans	6	1	1	2	8	2	0	13	7	0	146
Shreveport	0	-------	0	0	3	1	0	0	0	0	25
Oklahoma:											
Oklahoma City	3	-------	-------	4	1	1	0	2	2	-------	35
Tulsa	0	-------	0	0	0	1	0	0	1	7	-------
Texas:											
Dallas	1	-------	0	0	3	5	0	2	4	2	66
Fort Worth	0	-------	0	0	4	0	0	2	2	0	30
Galveston	0	-------	0	0	0	0	0	1	0	0	12
Houston	5	-------	0	2	3	3	0	2	5	0	54
San Antonio	0	-------	0	0	5	0	0	2	0	1	58
Montana:											
Billings	0	-------	0	0	0	0	0	0	0	0	3
Great Falls	0	-------	0	0	2	0	0	0	0	4	6
Helena	0	-------	0	0	0	0	0	0	0	0	5
Missoula	0	-------	0	1	2	0	0	1	0	0	7
Idaho:											
Boise	0	-------	0	0	0	0	0	0	0	1	5
Colorado:											
Denver	0	-------	1	3	5	6	0	2	0	9	76
Pueblo	0	-------	0	0	1	0	0	0	0	2	11
New Mexico:											
Albuquerque	0	-------	0	0	0	0	0	2	0	8	8
Utah:											
Salt Lake City	0	-------	0	17	0	2	0	0	0	9	33
Nevada:											
Reno	0	-------	0	0	1	0	0	0	0	0	2
Washington:											
Seattle	1	-------	-------	0	-------	1	0	-------	0	21	-------
Spokane	0	-------	-------	14	-------	2	0	-------	0	0	-------
Tacoma	0	-------	0	2	2	3	0	0	0	1	18
Oregon:											
Portland	0	-------	0	1	1	4	6	0	0	4	40
Salem	0	3	0	1	0	0	0	0	0	0	-------
California:											
Los Angeles	9	8	2	29	7	11	1	23	0	58	236
Sacramento	0	-------	0	1	1	1	0	3	0	11	28
San Francisco	0	2	0	1	2	3	0	10	1	10	157

City reports for week ended July 22, 1933—Continued

State and city	Meningococcus meningitis		Poliomyelitis cases
	Cases	Deaths	
Massachusetts:			
Boston	0	0	15
Worcester	0	0	1
New York:			
New York	1	1	18
New Jersey:			
Newark	0	0	1
Pennsylvania:			
Philadelphia	1	0	0
Pittsburgh	0	0	2
Ohio:			
Cleveland	1	0	0
Indiana:			
Indianapolis	1	0	0
Illinois:			
Chicago	0	1	5
Michigan:			
Detroit	0	0	2
Flint	0	1	0
Minnesota:			
Minneapolis	0	0	1
St. Paul	0	0	2
Missouri:			
St. Joseph	1	0	0
North Dakota			
Fargo	0	0	1
Nebraska:			
Omaha	0	1	0
South Dakota:			
Aberdeen	0	0	1
Maryland:			
Baltimore	0	1	0
Cumberland	1	1	0
District of Columbia:			
Washington	1	0	0
West Virginia:			
Wheeling	0	0	1
North Carolina:			
Raleigh	0	1	0
Georgia:			
Atlanta	1	0	0
Tennessee:			
Nashville	0	0	2
Alabama:			
Birmingham	1	1	0
Arkansas:			
Little Rock	0	0	1
Louisiana:			
New Orleans	1	0	0
Texas:			
Dallas	0	0	1
Houston	0	0	2
California:			
Los Angeles	0	0	1
San Francisco	1	0	0

Lethargic encephalitis.—Cases: New York, 4; Pittsburgh, 1.
Pellagra.—Cases: Baltimore, 2; Charleston, S.C., 3; Miami, 2; Memphis, 1; Mobile, 1; New Orleans, 2; Dallas, 1.
Typhus fever.—Cases: New York, 2; Atlanta, 1; Tampa, 1.

FOREIGN AND INSULAR

CANADA

Provinces—Communicable diseases—Two weeks ended July 15, 1933.—The Department of Pensions and National Health of Canada reports cases of certain communicable diseases for the 2 weeks ended July 15, 1933, as follows:

Disease	Prince Edward Island	Nova Scotia	New Brunswick	Quebec	Ontario	Manitoba	Saskatchewan	Alberta	British Columbia	Total
Cerebrospinal meningitis			1	1	4					6
Chicken pox		3		73	313	24	60	6	102	581
Diphtheria		1	3	23	18	10	5	1		61
Erysipelas				13		1		2		16
Influenza		8				10			19	37
Measles			1	138	67		2		16	224
Mumps					79	9	7	1	18	114
Pneumonia					11				3	14
Poliomyelitis		1		4			1		1	7
Scarlet fever		5	6	54	73	24	1	4	19	186
Trachoma									2	2
Tuberculosis	1	5	24	134	102	11	51	5	34	367
Typhoid fever			7	27	11	6		3	1	55
Undulant fever					9		1			10
Whooping cough			6	134	250	154	15	27	42	628

Quebec Province—Communicable diseases—Ten weeks ended July 15, 1933.—The Bureau of Health of the Province of Quebec, Canada, reports cases of certain communicable diseases for the 10 weeks ended July 15, 1933, as follows:

Disease	Two weeks ended—				
	May 20	June 3	June 17	July 1	July 15
Botulism					1
Cerebrospinal meningitis	1		3		1
Chicken pox	329	293	272	134	73
Diphtheria	46	28	26	15	23
Erysipelas	7	12	7	9	13
German measles	21	3	6	6	5
Influenza	5	1	5		
Measles	478	642	388	177	133
Ophthalmia neonatorum			1	2	
Poliomyelitis	1		5	1	4
Puerperal septicemia	2	3	2	1	
Scarlet fever	103	80	78	64	54
Tuberculosis	164	127	106	150	134
Typhoid fever	60	25	36	37	27
Undulant fever		2			
Whooping cough	112	133	167	93	134
Other communicable diseases	132	104	119	64	30

CHILE

Santiago—Typhus fever.—According to information dated July 19, 1933, the epidemic of typhus fever in Santiago, Chile, had reached major proportions. On July 13, 325 cases were said to exist in the city, 294 of which were in hospitals. This represented a great increase over the weekly average of 45.1 cases from January 1 to June 10, 1933.

The present epidemic of typhus was said to have occurred first in the south-central section of the country during June and July, 1932, and later to have become prevalent in Santiago where it found fertile field for propagation in concentration camps of unemployed workers and in the poorer sections of the city.

EGYPT

Notifiable diseases—Year 1932.—The following table gives the number of cases of certain diseases reported in Egypt during the year 1932. The figures are provisional.

Disease	Cases	Disease	Cases
Anthrax	23	Poliomyelitis and polioencephalitis	11
Cerebrospinal fever	4,508	Puerperal septicemia	604
Chicken pox	740	Rabies	2,337
Diphtheria	1,990	Relapsing fever	1
Dysentery	2,117	Scarlet fever	102
Erysipelas	2,996	Smallpox	600
Influenza	5,731	Tetanus	532
Leprosy	119	Tuberculosis, pulmonary	3,580
Lethargic encephalitis	13	Typhoid fever	3,653
Malaria	1,343	Typhus fever	2,298
Measles	19,649	Undulant fever	10
Mumps	796	Whooping cough	3,305
Plague	134		

NICARAGUA

Influenza.—Information has been received, dated July 19, 1933, of an epidemic of influenza at Puerto Cabezas, Nicaragua, and in the surrounding territory. During the 2 weeks ended July 15, 304 cases of influenza, with 6 deaths, were reported.

PUERTO RICO

Notifiable diseases—4 weeks ended July 15, 1933.—During the 4 weeks ended July 15, 1933, cases of certain notifiable diseases were reported in the municipalities of Puerto Rico as follows:

Disease	Cases	Disease	Cases
Chicken pox	32	Ophthalmia neonatorum	9
Colibacillosis	1	Paratyphoid fever	1
Diphtheria	37	Pellagra	3
Dysentery	238	Puerperal fever	3
Erysipelas	3	Ringworm	3
Filariasis	3	Syphilis	22
Framboesia	2	Tetanus	4
Influenza	42	Tetanus, infantile	2
Leprosy	2	Trachoma	1
Malaria	2,543	Tuberculosis	415
Measles	128	Typhoid fever	35
Mumps	16	Whooping cough	152

YUGOSLAVIA

Communicable diseases—June 1933.—During the month of June 1933 certain communicable diseases were reported in Yugoslavia as follows:

Disease	Cases	Deaths	Disease	Cases	Deaths
Anthrax	41	3	Paratyphoid fever	12	----
Cerebrospinal meningitis	11	7	Scarlet fever	212	6
Diphtheria and croup	424	43	Sepsis	12	6
Dysentery	26	2	Tetanus	56	23
Erysipelas	171	9	Typhoid fever	216	20
Measles	595	8	Typhus fever	136	8

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

(NOTE.—A table giving current information of the world prevalence of quarantinable diseases appeared in the PUBLIC HEALTH REPORTS for July 28, 1933, pp. 896-906. A similar cumulative table will appear in the PUBLIC HEALTH REPORTS to be issued August 25, 1933, and thereafter, at least for the time being, in the issue published on the last Friday of each month.)

Cholera

China—Tientsin.—During the week ended July 1, 1933, two cases of cholera were reported in Tientsin, China.

India.—During the week ended July 8, 1933, cholera was reported in certain cities of India as follows: Bombay, 1 case, 2 deaths; Calcutta, 24 cases, 2 deaths.

Indo-China, French—Saigon and Cholon.—During the week ended July 15, 1933, Saigon and Cholon reported 1 case of cholera with 1 death.

Philippine Islands—Opon.—During the week ended July 29, 1933, 8 cases of cholera with 6 deaths were reported in Opon, Cebu Province, Philippine Islands.

Plague

Egypt—Alexandria.—During the week ended July 22, 1933, Alexandria, Egypt, reported two cases of plague.

Indo-China, French—Saigon and Cholon.—During the week ended July 8, 1933, Saigon and Cholon reported 1 case of plague with 1 death.

Iraq—Baghdad.—During the week ended July 22, 1933, Baghdad reported 1 case of plague with 1 death.

India—Bombay.—During the week ended July 22, 1933, Bombay reported 1 case of plague with 1 death.

Smallpox

China.—Certain cities in China have reported smallpox as follows: During the week ended July 1, 1933, Nanking reported 1 case of smallpox; Shanghai reported 3 cases with 1 death. During the week ended July 15, 1933, Hong Kong reported 1 case with 1 death.

Syria—Beirut.—During the week ended July 22, 1933, Beirut, Syria, reported three cases of smallpox.

Typhus Fever

Algeria.—During the week ended July 8, 1933, typhus fever has been reported in certain places in Algeria as follows: Constantine Department, 20 cases; Oran, 1 case.

Egypt.—Certain cities of Egypt have reported typhus fever as follows: During the week ended July 22, 1933, Alexandria reported 1 case with 1 death. During the week ended July 15, 1933, Cairo reported three cases of typhus fever.