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THE CARIES-FLUORINE HYPOTHESIS AND A SUGGESTED STUDY TO TEST ITS APPLICATION[1]

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INTRODUCTION

The purpose of this thesis is to present the story of dental caries as it exists today, to present its public health significance, and to suggest a study for its control. It is proposed to introduce nontoxic doses of sodium fluoride into public drinking waters to test the fluorine-caries hypothesis, which points to an inverse ratio of caries to fluorides present in drinking water (*1*). If this hypothesis can be determined affirmatively, it will indeed revolutionize our thinking and our approach to the solution of the dental caries problem. With conclusive positive evidence, it may be possible to effect mass protection and not have to depend on the individual to do anything about it. By deliberately treating public water supplies with effective yet nontoxic doses of fluoride salts, daily protection would be afforded without the public being aware of it. The possibilities of such findings fairly stagger the imagination when the extent of dental caries today, the economic problem involved in an attempt to correct accumulated defects, and the difficulties encountered in getting persons to dentists for treatment are considered.

As far back as we can go in history, including archaeological studies dating from the Danish Stone Age, we find that human beings have been subject to the ravages of dental disease. Bremner (*2*), in his Story of Dentistry, states: "Decay of human teeth from ancient times has been found in so many places that it is legitimate to doubt whether there ever was an epoch when the human species was not cursed with toothache."

The papyrus of Ebers (*3*), the most ancient of all known works on medicine, written about 37 centuries B. C., mentions dental diseases

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and therapeutic measures for their cure. In 2700 B. C., (*4*) the Chinese Emperor, Houang-ty, founder of medicine, wrote about dental caries and indicated that worms were the causative agent. This celebrated work refers to toothache as ya-tong, and reveals nine different varieties. Yasuhori Tambu (*5*), in 892 B. C., wrote Isinho, which explained the worm theory of dental disease. It was not until about 400 B. C., when Hippocrates introduced the humeral theory to explain disease in the human body, that the worm theory was questioned. Hippocrates (*6*) included diseases of the teeth along with other human ailments. In the second century A. D., Galen (*7*) associated dental disease with nutritional deficiencies.

Following the fall of the Roman Empire through the Dark Ages superstition once more took hold and the worm theory again came into prominence and remained until the eighteenth century when men like Pierre Fauchard began to doubt it. In more recent times the writings of W. D. Miller (1885) (*8*) and G. V. Black (1886) (*9*), along with those of a number of present-day authors, give a picture of accumulated dental defects which is truly disheartening. Studies made on children of preschool age by McCall (*10*), on school children by Klein, Palmer, and Knutson (*11*), on adolescents and adults by the Economics Committee of the American Dental Association (*12*) indicate that dental disease is practically universal, affecting the entire population, starting as early as 2 years of age and continuing throughout life. Klein and Palmer (*13*) have shown the disproportionate rate of dental corrections to yearly increments which accounts for the accumulated problem today. Of even more striking significance and gaining much publicity through newspapers, magazine articles, lectures, and actual contact with the individuals involved is the report on physical defects responsible for the rejection of about 50 percent of the Nation's young manhood called up for the Selective Service in 1941. Of 2,000,000 men examined, 900,000 were rejected for physical and mental disabilities, the leading cause for rejections being dental defects (*14*).

Cause of rejection:	*Number of cases*	*Percentage*
Dental defects	188, 000	20. 9
Defective eyes	123, 000	13. 7
Cardiovascular diseases	96, 000	10. 6
Muscles—skeletal defects	61, 000	6. 8
Venereal diseases	57, 000	6. 3
Mental and nervous diseases	57, 000	6. 3
Hernia	56, 000	6. 2
Defects of ears	41, 000	4. 6
Defects of feet	36, 000	4. 0
Defective lungs, including tuberculosis	26, 000	2. 9
Miscellaneous	159, 000	17. 7
Total	900, 000	100. 0

Failure to meet the dental requirements assumes greater significance when we consider what these requirements are, as given in the United States War Department Mobilization Regulations MR1–9, issued August 31, 1940:

Paragraph 31. Classes 1–A and 1–B.

a. Class 1–A.

(1) Normal teeth and gums.

(2) A minimum of three serviceable natural masticating teeth above and three below opposing and three serviceable natural incisors above and three below opposing. (Therefore, the minimum requirements consist of a total of six masticating teeth and six incisor teeth.) All of these teeth must be so opposed as to serve the purpose of incision and mastication.

(3) *Definitions.*

(*a*) The term "masticating teeth" includes molar and bicuspid teeth, and the term "incisors" includes incisor and cuspid teeth.

(*b*) A natural tooth which is carious (one with a cavity), which can be restored by filling, is to be considered as a serviceable natural tooth.

(*c*) Teeth which have been restored by crowns or dummies attached to bridgework, if well placed, will be considered as serviceable natural teeth when the history and the appearance of these teeth are such as clearly to warrant such assumption.

(*d*) A tooth is not to be considered a serviceable natural tooth when it is involved with excessively deep pyorrhea pockets, or when its root end is involved with a known infection that has or has not an evacuating sinus discharging through the mucous membrane or skin.

b. Class 1–B.

Insufficient teeth to qualify for class 1–A, if corrected by suitable dentures.

Paragraph 32. Class 4.

a. Irremediable disease of the gums of such severity as to interfere seriously with useful vocation in civil life.

b. Serious disease of the jaw which is not easily remediable and which is likely to incapacitate the registrant for satisfactory performance of general or limited military service.

c. Extensive focal infection with multiple periapical abscess, the correction of which would require protracted hospitalization and incapacity.

d. Extensive irremediable caries.

All governmental agencies, Federal, State, and local, are becoming more interested in finding means to correct this morbid condition, which today is considered a public health problem. Sinai (*15*) defines a public health problem as:

(*a*) A condition or situation which is a widespread cause of morbidity or mortality, or both;

(*b*) concerning this situation there is a body of scientific knowledge which, if applied, would prevent, cure, or ameliorate that condition;

(*c*) that body of knowledge is not being applied.

In regard to the first part of the definition, there is ample evidence that dental disease affects large masses of the public. As to the knowledge regarding dental disease, several theories concerning the etiology and control have been advanced, such as *L. acidophilus*, diet

and nutrition, and fluorides. The most acceptable theory today is that relating to local environmental factors, especially *L. acidophilus*, but even the exponents of this theory admit that attacking the dental problem by curtailment of sugars and starches is well-nigh impossible with the American public. Today the only practical means of limiting the damage is periodic care by a dentist, starting as early as 2 years of age and continuing throughout life. However, economic factors and limitations of personnel, both in number and distribution, make this a remote possibility. Certainly the little that is known about the control of dental disease is not being applied when we learn that only about 25 percent of the people get anything like adequate dental care. It has also been estimated that a far higher percentage can afford adequate treatment but do not avail themselves of the opportunity (*16*). It seems unreasonable to argue that these people do not know the importance of good dental health, because so much has been said and written on the subject of late. It appears that inertia, together with a needless fear of dental treatment, keeps many persons from seeking care.

It is because of the widespread need, and the little likelihood of satisfying this need by present-day methods, that some other means more effective, less trying to the public, and within reasonable limitations of cost, have prompted the study proposed in this thesis.

CHAPTER I

THE FLUORINE-CARIES HYPOTHESIS

The low incidence of dental caries among persons living in areas supplied by waters containing fluorides has been reported from many sections of the world—long before it was known that fluorine in drinking water was the agent responsible for mottling of enamel. From studies in Japanese provinces, Masaki (*17*) reported that "the percentage of dental caries is comparatively small among those who suffer from this abnormality." In Argentina, Erasquin (*18*) found a lower incidence of caries among inhabitants of areas where mottled enamel is endemic. From Italy, Piperno (*19*) reported a similar condition. Ainsworth (*20*), in England, found in a group of 214 children, 5 to 15 years of age, living in areas where drinking water contained fluorides, that both the deciduous and permanent teeth showed a low incidence of caries. Whereas, only 12 percent of the deciduous and 7.9 percent of the permanent teeth of these children were carious, the averages for the rest of England were 43.3 percent in the deciduous and 13.14 percent in the permanent. Ainsworth also pointed out that, whereas the permanent teeth of the experimental group showed evidence of fluorosis, the deciduous teeth did not. Day (*21*), in India, also has reported a lower incidence of caries among those living in areas where fluorosis is endemic.

Of interest is the observation made by Chrichton-Browne (*22*) in 1892. He raised the question as to whether the marked deficiency of fluorine in the diet in England may be responsible for the high rate of caries there.

In this country, Eager (*23*), in 1901, was perhaps the first to report on this condition. While examining Italian emigrants from Naples and its environs, he noted a peculiar dental defect known as "Denti di Chiaie." According to Eager, "The etiology seems to be connected with volcanic fumes or the emanations of subterranean fires, either by fouling the atmosphere or forming a solution in the drinking water."

Black and McKay (*24*), in 1916, were the first to make a thorough study of the condition among the inhabitants of this country. They examined groups of children in the Rocky Mountain section where local dentists reported that mottled enamel was prevalent. In this first report Black said, "As to caries, the teeth of these children compare favorably with those of other communities where endemic mottled enamel is unknown. * * * But when teeth do decay, the frail condition of the enamel makes it extremely difficult to make good and effective fillings. For this reason, many individuals will lose their teeth, though the number of carious cavities is fewer than elsewhere."

McKay (*25*) in 1929, made similar observations and took issue with the accepted theory that defective enamel predisposed a tooth to caries. He pointed out that mottled enamel is perhaps "the most poorly constructed enamel of which there is any record in the literature of dentistry," and yet these mottled enamel teeth do not show any greater liability to decay than do normally calcified teeth. In fact, this writer feels that McKay was rather conservative in his analysis of his figures. The figures given by McKay may be interpreted to show that there was less caries among the poorly calcified mottled teeth than in those normally calcified. Dean (*26*), of the United States Public Health Service, has compiled certain of McKay's data in table 1.

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				
			Teeth	All teeth		Molar teeth	
				Number examined	Percent carious	Number examined	Percent carious
Towner, Colo. (population 154, 1930).	[1] 55	1,264	Normal	879	11	254	36
			Mottled enamel	385	9	101	33
Bruneau, Idaho (population 481, 1930).	54	1,142	Normal	356	16	126	46
			Mottled enamel	[2] 797	8	213	29
Pima Indian School, Sacaton, Ariz. (population unstated).	[1] 78	2,178	Normal	283	22	99	57
			Mottled enamel	1,895	14	529	45

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

[2] Presumably, 11 deciduous teeth were included in this total (797); all other teeth referred to are apparently permanent teeth.

Bunting, Crowley, Hard, and Keller (*27*) were perhaps the first to investigate the dental caries problem in areas where mottled enamel is endemic. This study was made in 1928 at Minonk, Ill. It was observed there that the extent and activity of dental decay was decidedly limited among the children in the mottled enamel areas. "In regard to the prevalence of dental caries among the children, the percentage of those affected was about the same as would be found in any community, but although caries occurred in the mouths of most children, the extent and activity were remarkably limited. The great majority of cavities consisted of small pit and fissure lesions in the molars, and seldom did caries extend beyond that stage. In this respect, the behavior of dental caries in the mouths of these children is distinctly different to that which usually occurs." While the causative factor of mottled enamel had not been established at this time, this group of investigators stated, "There may be some principle in the drinking water which either inhibits the activity of dental caries or protects the teeth from injury."

It was McKay (*28*) who first really associated the etiology of this condition with something in the drinking water. In 1927 he visited areas around Naples and observed a correlation existing between those manifesting signs of mottled enamel and their supply of drinking water from wells sunk in ancient lava flow. He also observed that in Pozzuoli, where dental fluorosis had previously been reported endemic, no new cases developed after a change in the water supply.

The most interesting and conclusive contribution made by McKay (*29*) was his study in Oakley, Idaho, where in 1925 he recorded an incidence of 100 percent dental fluorosis. Fully convinced that this condition was caused by something in the drinking water, he persuaded the community to change its source of water and to obtain its supply from a nearby area where no mottling had been found. After 8 years another survey was made of the permanent teeth of all children born in Oakley after the change had been effected, and this examination showed no mottling. A chemical analysis of the first and second sources of drinking water made some time later showed 6 p. p. m. of F. in the former, and 0.5 p. p. m. of F. in the latter.

While the causative agent of fluorosis was not determined until 1931, Gautier and Clausmann (*30*) demonstrated a high fluorine content in the water and gases in volcanic areas around Naples in 1913 and 1914. However, at that time no attempt was made to correlate these findings with the "Chiaie" teeth.

In 1931 three independent studies demonstrated that fluorine was the etiologic factor in dental fluorosis. Churchill (*31*) found 2 to 13.7 p. p. m. F. in water in endemic areas where mottling was observed. Smith, Lantz, and Smith (*32*) confirmed Churchill's finding and demonstrated it experimentally with white rats. In the same year Balozet and Velu (*33*) made similar observations in the rock phosphate areas of North Africa.

Dean, in a series of papers, was the first to correlate in a statistical manner the incidence of dental caries with the fluorine content of the drinking water. His epidemiological studies have been most thorough, and the evidence points strongly to the accuracy of the hypothesis that there is an inverse ratio of caries to fluorine found in drinking water. In selecting communities for comparable studies, he was careful to control, as far as possible, all variable factors with the exception of the fluorine content of the water supply. He considered such variables as climate, days of sunshine, latitude, economic status, diet, age, sex, color, and nativity, and, as far as possible, employed the same examiners to negate that variable factor.

Dean's studies (*34*) in 1938 revealed that in various areas of endemic fluorosis in the West and Middle West, there were more caries-free children than in communities where there was little or no fluorine in the drinking water. This limited immunity seemed operative with respect to the deciduous teeth as well as the permanent teeth. Studying a large number of children in South Dakota, Colorado, and Wisconsin, he found that the severity of dental caries was low in areas of mottled enamel. In this paper he suggests the control of dental caries by control of the water supply with a minimal threshold of safety of approximately 1 p. p. m. of F.

In 1939 Dean et al. (*35*) reported a study of four cities in Illinois. In Galesburg and Monmouth, where the water contained 1.8 and 1.7 p. p. m. fluorine, respectively, the number of carious permanent teeth per 100 children was 201 and 205, respectively. In Macomb and Quincy, where the water contained only 0.2 p. p. m. fluorine, the rates were 401 and 633 per 100 children, respectively. An interesting finding of this study was that in areas of fluorosis the eight incisor teeth showed little or no interproximal decay. The 2,318 surfaces examined in Galesburg and Monmouth showed only 0.59 carious lesions per 100 surfaces, whereas in Macomb and Quincy, in 2,814 surfaces examined, there were 8.9 carious lesions per 100 surfaces.

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TABLE 2.—*A summary of the incidence and amount of dental caries in selected 12- to 14-year-old white children of 4 Illinois cities*

City	Number of children examined	Children with one or more carious permanent teeth		Children with caries-free permanent teeth		Number of carious permanent teeth per 100 children			
						Age in years, last birthday			
		Number	Percent	Number	Percent	12	13	14	Total
(a) 696 CHILDREN WITH HISTORY OF CONTINUOUS USE OF PUBLIC WATER SUPPLY									
Galesburg	243	155	63.8	88	36.2	177	207	201	194
Monmouth	99	63	63.6	36	36.4	115	213	271	208
Macomb	63	54	85.7	9	14.3	315	422	367	368
Quincy	291	279	95.9	12	4.1	563	615	732	628
(b) 885 CHILDREN EXAMINED, INCLUDING THOSE IN (a) AND THOSE WHO HAVE CONTINUOUSLY USED THE CITY WATER SINCE 6 YEARS OF AGE; WATER HISTORY PRIOR TO 6, VARIABLE									
Galesburg	319	207	64.9	112	35.1	182	226	196	201
Monmouth	148	96	64.9	52	35.1	150	200	266	205
Macomb	112	96	85.7	16	14.3	346	411	453	401
Quincy	306	294	96.0	12	4.0	567	623	740	633

L. acidophilus studies also were made on the Galesburg and Quincy children, and the differences in oral *L. acidophilus* counts closely reflected the differences in dental caries. The percentage of lactobacilli counts over 30,000 was 3.4 times higher in Quincy than in Galesburg. There was no difference in the amylolytic activity of the saliva from the Quincy and Galesburg children.

Two studies were reported by Dean et al. in 1941. The first study (*36*) was concerned with the town of Bauxite, Ark., where the water supply had been obtained chiefly from deep wells found to have a fluorine content of about 14 p. p. m. This was one of the highest fluoride concentrations ever recorded in any known common water supply in this country. Twelve years previous to this study the town had changed the source of water supply from that of the deep wells to that of the nearby Saline River which was fluorine-free. Children who had used the deep-well water while their teeth were developing all showed mottled enamel. Children born subsequent to the water change were free from mottled enamel. In an examination of these children, it was found that those who had developed mottled enamel while using the high fluoride water and had used the fluorine-free water only for the past 12 years showed markedly less dental caries than a comparable group of pupils from Benton, Ark., who had used the fluorine-free water of the Saline River throughout their lifetime. Children born within a few years of the change in water supply showed practically no mottling but had a low caries experience. Children born latest, and thus having the shortest period of exposure to the risk, showed the highest caries index. *L. acidophilus* counts were consistent with the clinical findings in these groups. Teeth severely mottled showed no tendency to rampant caries, even though fluorine-free water had been used during the past 12 years.

TABLE 3.—*Dental caries findings in Bauxite (Ark.) elementary and high school pupils with and without mottled enamel and exposed to a fluoride-free water for the past 12 years, and in Benton (Ark.) high school pupils exposed to a fluoride-free water throughout life. (Survey made April 22–27, 1940)*

Age in years, last birthday	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	
A. 75 BAUXITE (ARK.) PUPILS WITH HISTORY OF CONTINUOUS USE OF COMMON WATER SUPPLY																
	26 pupils using fluoride-free water throughout life A-1			23 pupils of transitional group [1] A-2			26 pupils using fluoride-free water past 12 years only; high fluoride water previously A-3									Total
Number of pupils examined	3	15	8	11	7	5	3	3	6	6	7	1	0	0	0	75
First permanent molars:																
Number (missing included)	12	60	32	44	28	20	12	12	24	24	28	4	----	----	----	300
Number showing dental caries experience	6	47	15	21	9	1	3	3	15	7	10	3	----	----	----	140
Percent showing dental caries experience		65			34			25		46		39		41		
Number of pupils, caries free	1	1	2	2	4	4	1	2	0	3	1	0	----	----	----	------
Percent of pupils, caries free		15			43			50		25		27		13		
Approximate number of years of risk of exposure to caries		3.2			5.7						10.5					
Mottled enamel:																
Number of pupils with	0	1	0	0	1	0	3	3	6	6	7	1	----	----	----	------
Degree of clinical affection (weighted average) [2]	0	0.1	0	0	0.2	0	2.7	3.7	3.8	3.2	3.7	4.0	----	----	----	------

B. 50 BAUXITE (ARK.) PUPILS SHOWING A RELATIVELY MODERATE TO SEVERE TYPE OF MOTTLED ENAMEL. GROUP CONSISTS OF 26 OF "A-3" ABOVE AND 24 OTHERS WITH MINOR VARIATIONS IN CONTINUITY OF USE OF PUBLIC WATER SUPPLY

Number of pupils examined	4	8	12	9	9	4	3	0	1	50
First permanent molars:										
Number (missing included)	16	32	48	36	36	16	12	0	4	200
Number showing dental caries experience	3	12	32	11	17	7	0	----	1	83
Percent showing dental caries experience	31		51		42		37			
Number of pupils, caries free	2	3	0	4	1	2	3	----	0	15
Percent of pupils, caries free					30					
Approximate number of years of risk of exposure to dental caries					10.8					
Mottled enamel:										
Number of pupils with	4	8	12	9	9	4	3	----	1	------
Degree of clinical affection (weighted average) [2]	3.0	3.0	3.8	3.1	3.8	3.5	3.7	----	4.0	------

C. 45 BENTON (ARK.) HIGH SCHOOL PUPILS WITH A HISTORY OF CONTINUOUS USE OF THE COMMON WATER SUPPLY. (NO CHANGE OCCURRED IN BENTON WATER SUPPLY DURING LIFETIME OF THIS GROUP)

Number of pupils examined	11	9	10	9	4	0	1	1	0	45
First permanent molars:										
Number (missing included)	44	36	40	36	16	----	4	4	----	180
Number showing dental caries experience	30	27	20	33	13	----	0	1	----	124
Percent showing dental caries experience	71		70		69		58			
Number of pupils, caries free	1	1	3	0	0	----	1	0	----	6
Percent of pupils, caries free					13					
Approximate number of years of risk of exposure to dental caries					9.9					
Mottled enamel.										
Number of pupils with	0	0	0	0	0	----	0	0	----	-----
Degree of clinical affection (weighted average) [2]	0	0	0	0	0	----	0	0	----	-----

[1] The public water supply at Bauxite was changed from deep well to filtered river water in May 1928; the "transitional group" includes those pupils born within 1½ years of the change in water supply (a 3-year period covering 1½ years prior to the change and 1½ years subsequent to the change).

[2] The following weights were given to the diagnosis recorded for each individual: Normal, 0; questionable, 0.5; very mild, 1; mild, 2; moderate, 3; severe, 4.

The second study by Dean et al. (*37*) was made to determine the lowest concentration at which fluorine was effective in the reduction of the incidence of dental caries, and the extent of the magnitude of this inhibition. Eight suburban cities around Chicago were selected. Elmhurst, Maywood, Aurora, and Joliet had, respectively, 1.8, 1.2, 1.2, and 1.3 p. p. m. fluorine in their drinking water. Elgin was intermediate with 0.5 p. p. m., and Evanston, Oak Park, and Waukegan used fluorine-free water.

It is interesting to observe that in white children 12 to 14 years of age the total caries experience in Elmhurst, Maywood, Aurora, and Joliet does not quite equal the number of already filled teeth in the fluorine-free communities. The drinking water in Elgin, with 0.5 p. p. m. of fluorine, seems to have offered some freedom from caries to the school population, which is intermediate in nature. Bacteriological studies were carried out on 1,761 children in these cities. There were fewer negative salivas and more *L. acidophilus* counts of 30,000 and over in the fluorine-free cities than in the cities supplied with water containing fluorine. This ratio held true with more or less regularity throughout the series. The bacterial counts followed the difference in dental caries experience.

TABLE 4.—*Summary of the percentage incidence and dental caries experience, permanent teeth, in the 1,761 children for whom a single* L. acidophilus *count was made*

City	Number of children examined	Children showing dental caries experience	Children showing no dental caries experience	Dental caries experience, permanent teeth				
				Filled teeth (past dental caries) (a)	Teeth with untreated dental caries (b)	Extraction indicated (c)	Missing (d)	Total (a+b+c+d)
				(A) Number				
Elmhurst	154	112	42	220	147	7	7	381
Maywood	139	100	39	174	164	1	13	352
Aurora	340	255	85	360	556	7	34	957
Joliet	233	191	42	265	469	10	41	785
Elgin	250	223	27	529	536	7	41	1,113
Evanston	208	200	8	802	485	23	89	1,399
Oak Park	208	202	6	1,010	424	13	61	1,508
Waukegan	229	223	6	884	816	31	160	1,891
	Percent of total examined clinically			(B) Number per 100 children				
Elmhurst	90.6	72.7	27.3	143	95	4.5	4.5	247
Maywood	81.3	71.9	28.1	125	118	.7	9.4	253
Aurora	53.7	75.0	25.0	106	164	2.1	10.0	281
Joliet	52.1	82.0	18.0	114	201	4.3	17.6	337
Elgin	62.0	89.2	10.8	212	214	2.8	16.4	445
Evanston	81.3	96.2	3.8	386	233	11.1	42.8	673
Oak Park	63.2	97.1	2.9	486	204	6.3	29.3	725
Waukegan	54.1	97.4	2.6	386	356	13.5	69.9	826

TABLE 5.—*Summary of the percentage distribution of oral* L. acidophilus *in salivas from 1,761 school children in 8 suburban Chicago communities*

City	Distribution of children according to the number of *L. acidophilus* per cc. of saliva								
	Negative	Less than 100	100 to 1,000	1,000 to 3,000	3,000 to 12,000	12,000 to 21,000	21,000 to 30,000	30,000 and over	Total
	(A) Number								
Elmhurst	39	16	13	9	16	13	10	38	154
Maywood	35	20	14	10	17	8	6	29	139
Aurora	86	44	24	10	43	34	12	87	340
Joliet	61	22	20	6	24	26	12	62	233
Elgin	40	13	24	14	40	22	14	83	250
Evanston	19	13	19	2	28	29	11	87	208
Oak Park	24	14	13	14	27	19	6	91	208
Waukegan	21	13	13	6	27	15	9	125	229
	(B) Percent								
Elmhurst	25.3	10.4	8.4	5.9	10.4	8.4	6.5	24.7	100
Maywood	25.2	14.4	10.1	7.2	12.2	5.7	4.3	20.9	100
Aurora	25.3	12.9	7.1	2.9	12.7	10.0	3.5	25.6	100
Joliet	26.2	9.4	8.6	2.6	10.3	11.2	5.1	26.6	100
Elgin	16.0	5.2	9.6	5.6	16.0	8.8	5.6	33.2	100
Evanston	9.1	6.3	9.1	1.0	13.5	13.9	5.3	41.8	100
Oak Park	11.5	6.7	6.3	6.7	13.0	9.1	2.9	43.8	100
Waukegan	9.2	5.7	5.7	2.6	11.8	6.5	3.9	54.6	100

In 1942, Arnold, Dean, and Elvove (*38*) reported a study in which they examined the caries experience of 109 children, averaging 13 years in age, in the town of Garrettsville, Ohio. In 1939 a new well which contained 1.7 p.p.m. of fluorine was dug in the community. By mixing this water with wells which contained no fluorine, the tap water had an average concentration of 0.7 to 0.8 p.p.m. of F. The children showed an average caries experience which would be expected from a community using fluorine-free water. *L. acidophilus* counts were made during a 1-year period, eight separate saliva samples being collected from each child. The results showed that there was, if anything, an increase in high counts at the end of the year, suggesting no arrest of dental caries. However, the authors caution that the relatively unchanged counts may be the result of dental caries activity and lesions which had started prior to the change of water and which were still active. The experiment might indicate that exposure to low concentrations of fluorine over a number of years is required in order to produce beneficial results.

Studies on rats have been made by numerous investigators in an attempt to determine the effect of fluorine on caries. Miller (*39*), working on the hypothesis that the enzyme systems, capable of transferring the phosphate radical, are the important factors in the localized decalcification of the enamel, investigated the effects of small amounts of fluorides and iodoacetic acid added to food and water of rats placed on a caries-producing diet. He observed that both sodium fluoride

and iodoacetic acid added to the caries-producing diets for rats resulted in a very low incidence of dental caries compared with a group of rats on a diet similar in type but without sodium fluoride or iodoacetic acid.

Hodge and Finn (*40*) demonstrated that rats fed casein plus fluorine had from 60 to 70 percent less caries than groups fed powdered milk and plain casein. This observation somewhat negates the findings of Lilly (*41*) who attributed a decrease in rat caries to commercial casein in the diet. It is of interest to note that commercial casein was found to contain 0.2 percent of fluorine (*42*).

Cox et al. (*43*) observed that diets containing up to 40 p. p. m. of sodium fluoride fed to pregnant rats significantly reduced the incidence of dental caries in their offspring, when compared with control litters. An important point brought out by this investigation is that the only fluorine contained in the teeth of young rats is that which is derived from the mother. After the young rats were weaned, all rats used in the study were placed on the same low-fluoride, caries-producing diet. These data show that resistance to caries is inherent in the rat, presumably in the enamel.

McClure (*44*), in 1941, observed that the caries resistance of rats is correlated with the amount of fluorine in the diet; also that the fluorine content of the molar teeth of the experimental rats is changed in direct proportion to the amount of fluorine in the diet. However, it could not be determined from these experiments whether the action of fluorine in dental caries is local, systemic, or associated with tooth structure.

To eliminate the question of local action of fluorine, Arnold and McClure (*45*) administered subcutaneous injections of sodium fluoride to rats, and the dental caries experiences of the injected rats were compared with those of a control group and a group receiving 10 p. p. m. F. in drinking water. The results of this investigation indicated that:

1. Fluorine, when injected subcutaneously, did not reduce the susceptibility of rats to induced dental caries. This group had as much dental caries as either the control group or the group receiving 10 p. p. m. F. in the water. The investigators were led to assume that the action of fluorine in reducing dental caries in rats is possibly more closely related to local or oral factors than to systemic influence.

2. The fluorine content of the enamel and dentin of the incisor and molar teeth of the rats was increased by both the subcutaneous injections of sodium fluoride and by feeding water containing 10 p. p. m. F. This led to the assumption that the composition of enamel and dentin of molar teeth of rats may be altered by way of the blood stream from within the tooth, even after the crowns of these teeth have erupted in place.

Chapter II

TOXICOLOGY OF FLUORINE

Since fluorine is the twentieth most common element making up the composition of the earth's surface, it is not surprising to find endemic fluorosis so prevalent throughout the world. Over 75 reports in the literature tell of its widespread distribution in varying degrees on every continent of the world.

The existence of the element fluorine as a constituent of the teeth was first observed by Morichini (*46*) in 1802 while analyzing fossil teeth. Gay-Lussac and Berthollet (*47*) in 1805, however, were the first to show that fluorine was found as an element in normal human enamel.

It has been only within a comparatively few years that analytical methods for fluorine determination have been perfected. Among the most reliable figures are those given by Armstrong and Brekhus (*48*), who found that the fluorine content of the enamel from sound teeth was .0111 percent, and the fluorine content of dentin from sound teeth .0169 percent.

Gautier and Clausmann (*49*) observed that teeth had a higher content of fluorine than any other body tissue. This observation led them to assume that fluorine was a necessary element in the body. By dividing the organs of the body into three groups according to their metabolic activity, they found that those organs most active had the least amount of fluorine, and those least active had the most fluorine. The fluorine, they claimed, became fixed in the tissue and acted as the stabilizer and hardener of that tissue. Although the physiological value of this element was purely speculative from their viewpoint, the concept has, nevertheless, received some verification in the light of present findings relative to the role of fluorine in the inhibition of dental caries and the retardation of the production of rickets in rats fed a rachitogenic diet. Fluorine is as yet the only variable constitu ent in sound and carious teeth. It has been suggested by Trebitsch (*50*) that the hardest apatite, fluorapatite, occurs in dental enamel, and that the hardness of teeth depends upon the presence of apatite crystals. Thus there is some support for the assumption that fluorine is an essential element to the human body.

Sharpless and McCollum (*51*), in 1933, feeding rats a diet free of fluorine, could see no harmful effects from the omission of this element from the diet. However, there is some doubt as to whether this element was totally absent, and since the percentage of fluorine necessary for the body is extremely minute, this experiment is of questionable value.

Although mottled enamel produced by ingestion of more than 1.0 p. p. m. of fluorine during the development of the dentition is the

first objective indication of chronic fluorine poisoning, other effects of fluorine intoxication have been reported in the literature. These reports deal mainly with the intake of large doses and are noted chiefly in animal experimentation and among workers in cryolite mines and phosphate plants. The quantities reported are very much higher than those that are suggested in this paper for study purposes to test the caries-fluorine hypothesis.

That fluorine is acutely toxic was discovered fortuitously by the chemists Thenard and Davy (*52*) who became seriously ill when they breathed hydrofluoric acid vapors, and by Louyet and Nickels (*53*) who met death from the same cause. Today, it is well known how acutely toxic fluorides may be. Fatal doses have been reported as low as 0.2 gm. of sodium fluosilicate, but the usual lethal dosage runs from 5 to 15 gm. of sodium fluoride (*54*).

The consideration of chronic fluorosis has been directed for the most part to the skeletal structures, endocrine glands, and enzymatic processes of the body. Certainly the literature to date justifies the statement that reports are quite confusing regarding some aspects of chronic fluorine toxicosis, and further work in this field is warranted and should be encouraged.

The source of chronic fluorine toxicosis for human beings has been considered as existing in the United States principally in the drinking water. It has been stated (*55*) that the fluorine content of foods appears to play only a minor role in the production of mottled enamel, and although foods may possess a large quantity of fluorine, they have not been associated with mottled enamel unless there is an accompanying source of drinking water containing fluorine. Recent evidence offered by Sognnaes (*56*) is not in agreement with this statement. Evidence of mottled enamel was found on the island of Tristan di Cunha, where the drinking water contained only 0.2 p. p. m. of fluorine. The inhabitants, however, subsisted mainly on fish, which are known to have a high fluorine content. It may be necessary to change our concept of the importance of flourine in foods, especially in nontoxic doses, as related to the inhibition of dental caries.

Ingested fluorine is excreted in the urine, and there appears to be, according to Machle (*57*), a normal urinary fluorine secretion of approximately 1 mg. per liter of urine. Where the fluorine content of the drinking water is very high, there is a direct correlation between the fluorine ingested and the amount excreted in the urine. Fluorine is also stored in the body, in the bones, and in the teeth. Sonntag (*58*) found an increase of fluorine in the bones and teeth when animals were fed sodium fluoride.

In a consideration of the chronic toxicity of fluorine, particular attention should be given to the hard tissues and supporting structures

of the body, since it is in these tissues and structures that the first and probably the most severe indications of fluorosis are seen. Moller and Gudjonsson (*59*), Roholm (*60*), and others have reported that cryolite workers, after long periods of continued exposure to fluorine, developed a disease called osteosclerosis. Their diagnoses were made clinically, radiographically, and from autopsy material. They characterized the disease as a calcification of the ligaments and osteophytic outgrowths on various bones, and almost complete synostosis of the various joints which sometimes produced complete ankylosis of the vertebral column. According to Shortt et al. (*61*), the kidney function also was impaired in the majority of cases examined in an area in India where mottled enamel was prevalent.

Roholm (*62*) describes osteosclerosis as a disease which attacks bone and is characterized pathologically as a diffuse type in which the pathological formation starts both in the periosteum and endosteum. The capita densifies and thickens; the spongiosa trabeculae thicken and fuse together. The medullary cavity decreases in diameter and new bone forms from the periosteum. There is also calcification of the ligaments. All signs of bone destruction are absent from the syndrome. Cases of osteosclerosis have been reported by Speder (*63*) and by Gaud (*64*) among men in phosphate areas of North Africa, and by Bauer, Bishop and Wolff (*65*) and by Wolff and Kerr (*66*) among phosphate fertilizer workers in the United States. These workers have received considerable doses of fluorine, and they present a serious problem. However, no clinical reports from areas supplied by water containing 1 p. p. m. or slightly more of fluorine offer any evidence that these pathological changes exist. (Bauxite, Ark., reported 14 p. p. m.)

Studies within areas in the United States where the fluorine content of the water supply is comparable with concentrations which the study in this paper suggests, offer very little conclusive evidence of any changes other than mottled enamel.

Blue (*67*) made a clinical study of the general physical development of children in Oklahoma living in normal areas and in areas where mottled enamel is endemic. The finding of a greater number of fractures, rickets, and dental deformities in the mottled enamel areas indicated to him a retarded physical development. This was purely a clinical study and lacks confirmation, since no calcium or phosphorous studies were undertaken on these children. Calcium and phosphorous balance studies conducted by Lantz, Smith, and Leverton (*68*), and by Smith, Lantz, and Smith (*69*) on girls who lived in endemic areas of Arizona revealed a normal assimilation of these elements.

Capizzano et al. (*70*) observed definite bone changes in endemic areas of Argentina. However, their number of cases was small, and other factors may have played a secondary part. Their observations warrant further investigation to determine whether these alterations hold true generally.

Many investigators have pointed out that fluorine acts upon various endocrine glands of the body to alter their metabolism. More emphasis has been placed, with some justification, upon the thyroid gland. Although the evidence for alteration in thyroid structure in experimentally produced fluorosis in animals is conflicting, and there is at present no general agreement, it is highly possible that some correlation, as yet unproved, will come to light.

Goldemberg (*71*) reported beneficial results from the use of sodium fluoride in the treatment of hyperthyroidism. It is his contention that excess fluorine may cause endemic goiter. Phillips (*72*) was unable to confirm Goldemberg's findings in experimental animals. May (*73*), in 1935, reported that the administration of fluorine produced epithelial changes in the thyroid gland and altered staining reaction of the colloid. Having experimented with over 800 patients he advocated internal fluorine therapy in the treatment of toxic goiter.

In 1941 Wilson (*74*) reported a study on the incidence of dental fluorosis in the goitrous and nongoitrous areas of rural sections of England. It was found that 103 children from the nongoitrous area showed no mottled enamel, while of 378 children examined in the goitrous area, 55 showed mottled enamel. This observation is offered as evidence that goiter is associated with fluorine in the water supply and lends weight to Goldemberg's conclusion. However, the association of the two factors may be coincidental and not related. Wilson and DeEds (*75*), in 1940, offered evidence that thyroid extract has a synergistic action in the production of fluorine intoxication. Certainly further studies are indicated on the effect of fluorine on thyroid glands.

McCollum et al. (*76*), in 1925, were the first to observe changes in incisors of rats from the incorporation of fluorine in the diet. By the addition of 226 p. p. m. of sodium fluoride to the stock diet, rats developed bleaching, mottling, friability, and overgrowth of the incisors. During the same year, Schultz and Lamb (*77*) reported changes among rats similar to those seen by McCollum and his coworkers. Many other investigators followed with like reports.

Chaneles (*78*), in 1929, made a microscopic study of the incisors of rats fed fluorine and noticed histologic changes which were more completely described in 1935 by Schour and Smith (*79*), who stated that the action of fluorine is directly upon the enamel forming cells and not through changes in blood calcium and phosphorous.

Although the condition now known as mottled enamel was first described in 1901 by Eager (*80*), who reported the dental abnormality among Italian emigrants, it was not until Black and McKay (*81*), in 1916, studied the abnormality thoroughly from both the gross and microscopic aspects that a complete picture of the appearance and pathology of mottled enamel was obtained. The etiology in 1916 was still unknown. Black (*82*) describes the macroscopic appearance of mottled teeth as showing opaque white or yellow through brown to black spots over the surface of the teeth. Some had dark bands running horizontally across the teeth, fading off to yellow or opaque white. However, the surface of the enamel appeared glazed and not penetrable to the tine of an explorer. The pigmentation appeared only on the outer one-third or one-fourth of the enamel. The dentin was described as normal. Histologically there appears to be a lack of inner cementing substance between the enamel rods. The discoloration is described as due to the deposition of a pigment, which Black called "brownin," in the interstices between the rods. The teeth were of normal shape. Mottled enamel was found to be endemic in certain regions, affecting 87.5 percent of all children reared in that area.

Churchill (*83*), in 1931, in an analysis of the water from these endemic areas found a high fluorine content of over 1 p. p. m. in the drinking water.

However, it remained for Smith et al. (*84*), in 1931, to prove definitely that the condition of fluorosis produced in rats was the same as mottled enamel in human beings. An investigation conducted in St. Davids, Ariz., disclosed that all persons reared in the area, and who imbibed the water during the time of tooth formation, showed mottled enamel. These investigators produced fluorosis in rats also by feeding them St. David's water. It is now known that 14 p. p. m. of fluorine in the drinking water is toxic to rats; as low as 1 p. p. m. is toxic to man. This is the first evidence of fluorine toxicity.

CHAPTER III

PROPOSED PLAN FOR A STUDY

The objective of this proposed plan for a study is to test the accuracy of the caries-fluorine hypothesis by deliberately placing nontoxic doses of sodium fluoride in the public drinking water of one community, and using a comparable community with fluorine-free water as a control. The practicability as well as the efficacy of such a plan, studied over a period of from 10 to 12 years, may thus be determined.

Much care must be exercised in the selection of study areas which should be comparable in as many essential factors as possible. Dean

and Arnold (*85*) spent over a month studying approximately 20 cities in the northern Illinois area before deciding on the 8 cities which were included in their studies in that State. This care is necessary in order to rule out variables which may possibly affect the end result. Such variables are (1) chemical composition of past and present water supply; (2) composition of population in regard to size, age, sex, color, nativity, economic status; (3) geographic and climatic considerations, including latitude, days of sunshine; (4) diet; (5) same person conducting the examinations; (6) past dental caries experience.

The term "dental caries experience" was introduced by Klein and Palmer of the United States Public Health Service who state that a "reconstitution of the caries experience in the permanent teeth of children may be accomplished with a fair degree of precision by totaling the mutually exclusive numbers of carious teeth (irrespective of the number of defects per tooth), the number of filled teeth, and the number of extracted teeth plus those indicated for extraction. The summation of these values gives a count of the number of permanent teeth showing evidence of having been attacked by caries." When it is desired to express the dental caries experience as a rate per 100 children, the sum of the four aggregates referred to (number of teeth with untreated dental caries, filled teeth, extracted teeth, and those indicated for extraction) is divided by the number of children examined and the quotient multiplied by 100.

Study areas should have populations of over 25,000 in order to have a sufficient number of children at the end of the 10- to 12-year period who have lived continuously for this period in the areas under investigation. Dean (*86*) has estimated that by the end of the study approximately 65 percent of the original group will probably either have moved away or will present discontinuities in exposure, warranting their elimination from the study. In Dean's investigations, records were eliminated of all cases with an absence from their cities of 30 days or more in any one year.

The period of investigation should extend over 10 to 12 years because the current theory is that the effective action of the fluorine takes place during the years of tooth development. Although calcification of the crowns of the permanent teeth (with the exception of the third molars) is completed when a child is between 7 and 8 years of age, eruption is not completed until he is 12 or 13 years old (*87*).

The two areas to be selected should have 0.1 p. p. m. fluorine or less in the public drinking water. One of these areas is to have the fluorine

content of its water raised to 0.8 p. p. m., and the other is to be used as a control.

After careful selection of the areas, dental examination with mouth mirror and sharp explorer should be made of all children, preschool and school age (through age 14), in both the control and study areas. These examinations should be made annually throughout the decade or more of investigation. Also, the examinations throughout the study should be made by the same dentist because of the marked variation in diagnosis of small carious lesions, pits, and fissures by different dentists.

Bacteriologic examinations for *L. acidophilus* in the saliva of the children under study should be made periodically. These findings may serve as a useful index of caries activity. Jay (*88*) has demonstrated a correlation of high counts of *L. acidophilus* and active caries. Other investigators (*89*, *90*, *91*) have made similar observations.

A record card suitable for punch card machine analysis is suggested, similar to the one used by the United States Public Health Service in its Illinois study in 1941. This form allows for recording, in addition to the general history of the patient, the past and current caries experience of the primary as well as the permanent teeth, special references to the first permanent molar and the incisors, *L. acidophilus* counts, and sufficient history to determine continuity of exposure to the public water supply.

ADDITION OF SODIUM FLUORIDE TO THE PUBLIC WATER SUPPLIES

A dose of 0.8 p. p. m. of fluorine represents a dose of 1.77 p. p. m. in terms of sodium fluoride. This dosage would necessitate the addition of 14.7 pounds of sodium fluoride per million gallons of water. This small dose of sodium fluoride may be added to public water supplies through the use of one of several available types of equipment. The soluble chemical may be dissolved in a solution tank, and then applied in measured volumes through the use of a constant level box and calibrated orifice, identical to equipment used for applying alum solutions. In other instances, water under pressure may be treated through the use of small chemical pumps which are capable of being adjusted to apply known volumes of sodium fluoride solution. Possibly the simplest equipment of this nature would be one of the several makes of chemical feeders designed for applying sodium hypochlorite solution for disinfection purposes.

There will be no interference between the two procedures of chlorination and the addition of sodium fluoride to the water supply. The

sodium fluoride should, however, be added to the alum-treated water after filtration, because the coagulant is likely to remove more or less of the added fluoride, and it is desired that the filtered water, ready for consumption, contain 0.8 p. p. m. fluorine.

There should be no difficulty in the application of sodium fluoride to public water supplies in view of the fact that small, constant doses would be adequate. Laboratory control would therefore be restricted to the periodic examination of samples of raw water, and of the treated water for fluoride content, so that the actual dose applied could be subject to appropriate checking.

CONCLUSION

All of the early studies on fluorine were concerned with toxic doses and the resultant mottling of the enamel. The chief problem to be solved, following these findings, was that of eliminating fluorine from the public drinking water.

As these studies progressed, the relationship of a low caries rate to the fluorine content of water became evident, and this discovery opened up a field of tremendous importance to those in public health who are trying to solve the problem of dental caries.

With investigations to date indicating the apparent safety of small doses such as 1.0 p. p. m. fluorine in water, further experimentation with this threshold dose or less is warranted in order to test the efficacy and practicability of using this element universally, under strict control, to reduce the incidence and ravages of dental caries.

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INCIDENCE OF HOSPITALIZATION, MARCH AND APRIL 1943

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

Item	March	
	1942	1943
1. Number of plans supplying data	62	64
2. Number of persons eligible for hospital care	8,265,931	9,281,942
3. Number of persons admitted for hospital care	73,069	79,699
4. Incidence per 1,000 persons, annual rate, during current month (daily rate × 365)	104.0	101.0
5. Incidence per 1,000 persons, annual rate for the 12 months ending March 31	107.0	108.1
	April	
	1942	1943
1. Number of plans supplying data	60	65
2. Number of persons eligible for hospital care	7,932,108	10,003,704
3. Number of persons admitted for hospital care	70,444	85,482
4. Incidence per 1,000 persons, annual rate, during current month (daily rate × 365)	107.9	103.9
5. Incidence per 1,000 persons, annual rate for the 12 months ending April 30	107.1	106.7

DEATHS DURING WEEK ENDED MAY 22, 1943

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

	Week ended May 22, 1943	Corresponding week, 1942
Data for 88 large cities of the United States:		
Total deaths	8,847	8,158
Average for 3 prior years	8,215	
Total deaths, first 20 weeks of year	194,888	177,243
Deaths under 1 year of age	594	544
Average for 3 prior years	517	
Deaths under 1 year of age, first 20 weeks of year	13,463	11,262
Data from industrial insurance companies:		
Policies in force	65,524,713	64,976,942
Number of death claims	12,886	11,468
Death claims per 1,000 policies in force, annual rate	10.3	9.2
Death claims per 1,000 policies, first 20 weeks of year, annual rate	10.6	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

REPORTS FROM STATES FOR WEEK ENDED MAY 29, 1943

Summary

As compared with figures for the preceding week, reports for the current week show decreased incidence for all of the first nine diseases included in the following table with the exception of smallpox; but the current incidence of influenza, measles, meningococcus meningitis, poliomyelitis, and whooping cough continued above the corresponding 5-year (1938–42) median figures. Of these nine diseases, however, the cumulative totals for the first 21 weeks of the year for only measles, meningococcus meningitis, poliomyelitis, and whooping cough are above the corresponding median figures of the past 5 years.

The number of reported cases of meningococcus meningitis declined from 544 for the preceding week to 423 for the current week. Decreases were shown in all of the nine geographic areas except the Mountain States. States reporting more than 20 cases for the week (last week's figures in parentheses) are as follows: New York, 92 (89); New Jersey, 35 (41); Pennsylvania, 30 (39); Michigan, 27 (18); California, 22 (31).

Cumulative figures for the first 21 weeks of the year for other diseases reported currently (figures for the corresponding period of last year in parentheses) are as follows: Anthrax, 28 (33); dysentery, all forms, 6,008 (2,929); infectious encephalitis, 230 (175); leprosy, 10 (24); Rocky Mountain spotted fever, 70 (101); tularemia, 364 (395); endemic typhus fever, 967 (748).

Deaths registered in 89 large cities of the United States for the current week totaled 8,946, as compared with 8,856 for the preceding week, and a 3-year (1940–42) average of 7,741. The cumulative total for the first 21 weeks of the year is 204,172, as compared with 185,391 for the corresponding period of last year.

Telegraphic morbidity reports from State health officers for the week ended May 29, 1943, and comparison with corresponding week of 1942 and 5-year median

In these tables a zero indicates a definite report, while leaders imply that, although none were reported, cases may have occurred.

Division and State	Diphtheria			Influenza			Measles			Meningitis, meningococcus		
	Week ended		Median 1938–42	Week ended		Median 1938–42	Week ended		Median 1938–29,	Week ended		Median 1938–42
	May 29, 1943	May 30, 1942		May 29, 1943	May 30, 1942		May 29, 1943	May 30, 1942		May 29, 1943	May 30, 1942	
NEW ENGLAND												
Maine	1	0	0	------	1	------	81	62	106	3	3	0
New Hampshire	0	0	0	------	------	------	27	40	28	0	0	0
Vermont [2]	0	0	0	------	------	------	224	290	140	0	0	0
Massachusetts	0	4	3	------	------	------	1,715	968	943	13	3	1
Rhode Island	0	0	0	------	------	------	60	200	133	9	0	0
Connecticut	0	0	1	4	2	1	473	400	397	12	4	1
MIDDLE ATLANTIC												
New York	13	13	20	[1] 10	[1] 6	[1] 6	4,081	776	2,181	92	9	5
New Jersey	3	1	7	10	1	4	2,543	661	708	35	2	1
Pennsylvania	8	9	17	1	1	------	1,658	1,143	1,143	30	7	7
EAST NORTH CENTRAL												
Ohio	17	6	9	17	9	7	792	309	309	8	0	1
Indiana	7	3	5	11	4	3	162	234	159	1	0	0
Illinois	27	23	23	4	15	15	1,706	287	287	16	0	0
Michigan [2]	4	1	3	2	------	2	4,315	450	667	27	0	1
Wisconsin	0	0	1	37	48	35	2,374	1,274	1,274	1	1	1
WEST NORTH CENTRAL												
Minnesota	3	2	1	------	------	1	647	514	254	4	1	0
Iowa	5	2	2	------	------	------	397	268	268	1	0	0
Missouri	1	10	7	------	1	4	208	189	189	13	3	0
North Dakota	0	0	0	40	4	2	11	18	76	1	2	0
South Dakota	0	1	1	------	------	------	64	53	37	0	0	0
Nebraska	0	1	1	6	1	1	63	258	258	0	0	0
Kansas	4	1	3	1	4	4	377	287	392	0	0	1
SOUTH ATLANTIC												
Delaware	0	0	0	------	------	------	59	20	13	2	0	0
Maryland [2]	6	5	4	5	1	1	221	300	165	14	7	1
District of Columbia	0	0	2	1	------	------	92	50	50	7	0	0
Virginia	2	2	6	81	75	57	186	167	325	[3] 11	9	2
West Virginia	2	3	3	9	4	9	51	22	32	4	0	1
North Carolina	8	3	6	4	2	3	310	557	715	18	0	1
South Carolina	2	3	5	221	146	146	94	141	118	4	0	1
Georgia	7	3	3	8	8	14	294	142	142	1	1	0
Florida	1	1	1	6	1	3	48	246	152	5	0	0
EAST SOUTH CENTRAL												
Kentucky	2	1	5	4	1	4	143	63	113	7	0	0
Tennessee	0	2	5	4	10	16	234	33	87	5	2	0
Alabama	3	7	7	13	17	28	67	105	165	7	1	2
Mississippi [2]	2	5	4	------	------	------	------	------	------	5	3	1
WEST SOUTH CENTRAL												
Arkansas	3	4	4	7	27	27	75	108	108	3	1	0
Louisiana	2	1	1	1	9	7	19	79	27	0	1	1
Oklahoma	1	3	3	10	29	19	35	98	98	0	1	0
Texas	12	16	17	398	182	179	293	641	641	10	5	2
MOUNTAIN												
Montana	0	1	1	------	4	4	258	145	84	1	2	0
Idaho	0	0	0	------	------	------	50	63	40	10	0	0
Wyoming	0	0	0	18	36	------	110	55	55	0	0	0
Colorado	7	5	8	58	32	12	430	203	231	2	1	1
New Mexico	0	1	2	1	2	2	22	23	76	0	0	0
Arizona	2	0	1	33	59	55	67	48	48	2	0	0
Utah [2]	0	0	0	7	5	3	134	978	389	2	1	0
Nevada	0	0	------	------	------	------	0	55	------	0	0	------
PACIFIC												
Washington	4	2	1	3	1	------	532	646	502	11	3	0
Oregon	3	0	2	14	6	10	137	135	135	4	0	0
California	15	6	11	33	55	55	679	5,312	734	22	8	1
Total	177	151	203	1,082	809	876	26,618	19,116	19,116	[3] 423	81	47
21 weeks	5,297	5,590	6,849	73,723	75,305	164,052	422,983	391,848	391,848	10,276	1,648	1,029

See footnotes at end of table.

Telegraphic morbidity reports from State health officers for the week ended May 29, 1943, and comparison with corresponding week of 1942 and 5-year median—Con.

Division and State	Poliomyelitis			Scarlet fever			Smallpox			Typhoid and paratyphoid fever		
	Week ended		Median 1938–42	Week ended		Median 1938–42	Week ended		Median 1938–42	Week ended		Median 1938–42
	May 29, 1943	May 30, 1942		May 29, 1943	May 30, 1942		May 29, 1943	May 30, 1942		May 29, 1943	May 30, 1942	
NEW ENGLAND												
Maine	0	2	0	17	5	13	0	0	0	0	0	0
New Hampshire	0	0	0	11	7	4	0	0	0	0	0	0
Vermont	0	0	0	14	5	5	0	0	0	0	0	0
Massachusetts	0	0	0	465	181	181	0	0	0	3	2	1
Rhode Island	0	0	0	23	4	6	0	0	0	0	0	0
Connecticut	0	0	0	107	21	58	0	0	0	1	0	2
MIDDLE ATLANTIC												
New York	0	3	1	429	247	546	0	0	0	1	5	10
New Jersey	0	0	0	101	80	221	0	0	0	1	2	1
Pennsylvania	2	0	0	239	307	384	0	0	0	5	8	8
EAST NORTH CENTRAL												
Ohio	0	1	1	201	195	241	2	0	1	8	2	6
Indiana	0	0	0	19	39	87	2	0	1	1	2	2
Illinois	0	1	1	160	194	331	0	11	7	2	4	4
Michigan [2]	0	0	0	130	192	268	0	0	6	0	4	3
Wisconsin	1	1	0	317	135	149	0	3	3	1	0	0
WEST NORTH CENTRAL												
Minnesota	0	0	0	45	52	73	0	0	4	0	2	0
Iowa	0	0	0	42	30	61	1	0	15	3	1	2
Missouri	0	0	0	58	40	40	0	1	4	0	5	2
North Dakota	0	0	0	5	5	6	1	0	1	0	0	0
South Dakota	0	0	0	3	10	7	0	0	4	0	0	0
Nebraska	0	0	0	8	13	13	0	2	2	0	0	0
Kansas	1	1	0	28	54	54	0	0	0	0	0	1
SOUTH ATLANTIC												
Delaware	0	0	0	4	17	6	0	0	0	0	0	0
Maryland [2]	0	0	0	49	53	39	0	0	0	3	9	3
District of Columbia	0	0	0	12	5	14	0	0	0	3	0	0
Virginia	1	1	0	25	7	15	0	0	0	1	4	4
West Virginia	0	1	0	13	24	25	0	0	0	1	0	4
North Carolina	0	0	0	15	14	12	1	0	0	1	1	3
South Carolina	0	1	1	5	2	5	0	0	0	0	0	3
Georgia	1	0	0	7	8	10	0	2	0	6	9	13
Florida	0	1	1	5	2	2	1	0	0	3	4	4
EAST SOUTH CENTRAL												
Kentucky	0	0	1	25	36	30	2	0	0	0	2	5
Tennessee	0	0	0	20	11	25	0	0	3	0	1	7
Alabama	0	1	1	9	5	6	0	0	0	1	4	1
Mississippi [2]	0	0	0	1	2	2	1	1	1	4	0	2
WEST SOUTH CENTRAL												
Arkansas	1	0	0	1	7	4	1	7	7	1	3	3
Louisiana	0	1	1	0	4	6	0	0	0	3	7	7
Oklahoma	0	0	0	4	12	13	0	0	3	1	4	6
Texas	6	0	0	43	36	24	6	1	4	6	8	8
MOUNTAIN												
Montana	0	1	0	14	10	14	0	0	0	0	0	0
Idaho	0	0	0	92	2	4	0	0	0	1	0	0
Wyoming	0	0	0	24	15	4	0	0	0	0	0	0
Colorado	0	0	0	56	10	20	0	2	2	1	0	2
New Mexico	1	0	0	7	0	7	0	0	0	0	1	1
Arizona	3	0	0	15	3	6	0	1	1	0	0	0
Utah [2]	0	0	0	32	20	15	0	0	0	1	0	0
Nevada	0	0	-----	0	0	-----	1	0	-----	0	0	-----
PACIFIC												
Washington	1	0	0	43	26	26	0	2	2	0	0	0
Oregon	1	0	0	16	2	9	0	1	1	1	0	2
California	9	3	3	129	83	117	0	0	1	4	6	6
Total	28	19	27	3,088	2,232	3,354	19	34	57	68	100	141
21 weeks	547	436	442	82,498	76,813	100,689	536	471	1,498	1,236	1,700	1,811

See footnotes at end of table.

Telegraphic morbidity reports from State health officers for the week ended May 29, 1943, and comparison with corresponding week of 1942 and 5-year median—Con.

Division and State	Whooping cough			Week ended May 29, 1943								
	Week ended		Median 1938–42	Anthrax	Dysentery			Encephalitis, infectious	Leprosy	Rocky Mt. spotted fever	Tularemia	Typhus fever
	May 29, 1943	May 30, 1942			Amebic	Bacillary	Unspecified					
NEW ENGLAND												
Maine	80	24	34	0	0	0	0	0	0	0	0	0
New Hampshire	5	3	3	0	0	0	0	0	0	0	0	0
Vermont	12	30	36	0	0	0	0	0	0	0	0	0
Massachusetts	118	196	161	1	0	1	0	2	0	0	0	0
Rhode Island	18	28	23	0	0	0	0	0	0	0	0	0
Connecticut	39	105	81	0	0	8	0	0	0	0	0	0
MIDDLE ATLANTIC												
New York	254	323	323	0	3	8	0	5	0	1	0	0
New Jersey	172	308	194	0	0	1	0	1	0	0	0	0
Pennsylvania	184	238	238	0	0	0	0	0	0	0	0	0
EAST NORTH CENTRAL												
Ohio	94	145	200	0	0	0	0	0	0	0	0	0
Indiana	44	68	32	0	0	0	0	0	0	0	0	0
Illinois	119	286	169	0	0	0	0	0	0	0	1	0
Michigan [2]	304	279	279	0	0	2	0	1	0	0	0	0
Wisconsin	212	184	125	0	0	0	0	0	0	0	0	0
WEST NORTH CENTRAL												
Minnesota	86	20	40	0	2	1	0	0	0	0	1	0
Iowa	65	18	28	0	0	0	0	0	0	0	0	0
Missouri	4	8	21	0	0	0	0	0	0	0	0	0
North Dakota	1	9	9	0	0	0	0	1	0	0	0	0
South Dakota	6	0	4	0	0	0	0	0	0	0	0	0
Nebraska	20	0	7	0	0	0	0	0	0	0	0	0
Kansas	101	34	63	0	0	0	0	0	0	0	0	0
SOUTH ATLANTIC												
Delaware	2	1	9	0	0	0	0	0	0	0	0	0
Maryland [2]	129	38	57	0	0	0	0	0	0	0	0	0
Dist. of Columbia	39	17	10	0	0	0	0	0	0	0	0	0
Virginia	116	89	66	0	0	0	20	0	0	0	0	0
West Virginia	26	8	29	0	0	0	0	0	0	0	0	0
North Carolina	238	165	250	0	1	0	0	0	0	0	1	2
South Carolina	97	74	62	0	0	0	0	0	0	0	0	2
Georgia	63	35	35	0	1	6	2	0	0	0	2	15
Florida	34	38	29	0	0	0	0	1	0	0	0	6
EAST SOUTH CENTRAL												
Kentucky	41	124	87	0	0	0	0	0	0	0	0	0
Tennessee	67	94	64	0	0	0	2	0	0	0	1	0
Alabama	51	35	44	0	0	0	0	0	0	0	0	8
Mississippi [2]	------	------	-----	0	0	0	0	0	0	0	0	0
WEST SOUTH CENTRAL												
Arkansas	51	32	33	0	0	1	0	0	0	0	2	0
Louisiana	14	2	5	0	0	2	0	0	1	0	0	0
Oklahoma	22	13	23	0	0	0	0	0	0	0	0	0
Texas	548	160	297	1	17	193	0	0	0	0	0	15
MOUNTAIN												
Montana	22	32	30	0	0	0	0	0	0	1	3	0
Idaho	3	3	6	0	0	0	0	0	0	0	0	0
Wyoming	1	4	3	0	0	0	0	0	0	3	2	0
Colorado	17	19	28	0	0	0	0	0	0	0	1	0
New Mexico	10	22	40	0	0	0	0	0	0	0	0	0
Arizona	23	18	35	0	0	0	30	0	0	0	0	0
Utah [2]	66	34	52	0	1	0	1	0	0	0	3	0
Nevada	0	5	------	0	0	0	0	0	0	0	0	0
PACIFIC												
Washington	57	37	83	0	0	0	0	0	0	0	0	0
Oregon	28	14	20	0	0	0	0	0	0	0	3	0
California	378	333	356	0	4	8	0	0	0	0	0	0
Total	4,081	3,752	3,806	2	29	231	55	11	1	5	20	48
21 weeks	85,198	80,538	83,808	28	626	4,342	1,042	230	10	70	364	967
21 weeks, 1942	------	------	------	33	370	1,621	938	175	24	101	395	748

[1] New York City only.
[2] Period ended earlier than Saturday.
[3] Exclusive of delayed report of 4 cases in Virginia.

WEEKLY REPORTS FROM CITIES

City reports for week ended May 15, 1943

This table lists the reports from 86 cities of more than 10,000 population distributed throughout the United States, and represents a cross section of the current urban incidence of the diseases included in the table.

	Diphtheria cases	Encephalitis, infectious, cases	Influenza		Measles cases	Meningitis, meningococcus, cases	Pneumonia deaths	Poliomyelitis cases	Scarlet fever cases	Smallpox cases	Typhoid and paratyphoid fever cases	Whooping cough cases
			Cases	Deaths								
NEW ENGLAND												
Maine:												
Portland	0	0	------	0	15	0	2	0	0	0	0	15
New Hampshire:												
Concord	0	0	------	1	1	0	3	0	2	0	0	0
Vermont:												
Barre	0	0	------	0	0	0	0	0	0	0	0	0
Massachusetts:												
Boston	1	0	------	0	258	13	18	1	208	0	0	40
Fall River	0	0	------	0	145	0	0	0	4	0	0	6
Springfield	0	0	------	0	12	0	1	0	46	0	0	1
Worcester	0	0	------	0	145	0	2	0	13	0	0	6
Rhode Island:												
Providence	0	0	1	0	17	7	0	0	20	0	0	14
Connecticut:												
Bridgeport	0	0	1	1	0	4	3	0	1	0	0	1
Hartford	0	0	------	0	56	0	2	0	4	0	0	1
New Haven	0	0	1	0	11	1	1	0	1	0	0	10
MIDDLE ATLANTIC												
New York:												
Buffalo	0	1	------	0	66	1	8	0	13	0	0	3
New York	14	1	10	1	1,233	49	84	0	424	0	5	74
Rochester	0	1	------	0	174	7	7	0	8	0	1	11
Syracuse	0	0	------	0	91	2	2	0	2	0	0	16
New Jersey:												
Camden	1	0	------	0	11	1	0	0	3	0	0	1
Newark	0		------	2	298	10	7	0	15	0	0	21
Trenton	0		------	0	21	0	6	0	4	0	0	0
Pennsylvania:												
Philadelphia	1	0	3	2	194	14	26	0	139	0	0	61
Pittsburgh	0	0	3	2	43	3	16	0	14	0	0	60
Reading	0	0	------	0	64	0	1	0	3	0	0	5
EAST NORTH CENTRAL												
Ohio:												
Cincinnati	0	0	------	0	64	2	3	0	28	0	0	3
Cleveland	3	0	5	2	32	4	14	0	52	1	0	36
Columbus	1	0	------	0	56	0	2	0	14	0	0	4
Indiana:												
Fort Wayne	1	0	------	0	14	1	2	0	0	0	0	0
Indianapolis	4	0	------	2	197	1	5	0	24	0	0	24
South Bend	0	0	------	0	3	0	0	0	0	0	0	0
Terre Haute	0	0	------	0	15	0	1	0	1	0	0	0
Illinois:												
Chicago	13	0	4	2	715	15	30	0	94	0	0	66
Michigan:												
Detroit	2	0	------	0	1,390	11	21	0	41	0	1	89
Flint	0	0	------	0	233	0	0	0	2	0	0	5
Grand Rapids	1	0	------	2	38	0	2	0	3	0	0	17
Wisconsin:												
Kenosha	0	0	------	0	3	0	0	0	8	0	0	4
Milwaukee	0	0	------	0	668	0	8	1	181	0	0	65
Racine	0	0	------	0	5	0	0	0	22	0	0	1
Superior	0	0	------	0	14	0	0	0	1	0	0	0

City reports for week ended May 15, 1943—Continued

	Diphtheria cases	Encephalitis, infectious, cases	Influenza		Measles cases	Meningitis, meningococcus, cases	Pneumonia deaths	Poliomyelitis cases	Scarlet fever cases	Smallpox cases	Typhoid and paratyphoid fever cases	Whooping cough cases
			Cases	Deaths								
WEST NORTH CENTRAL												
Minnesota:												
Duluth	0	0		0	15	0	1	0	1	0	0	4
Minneapolis	0	0		0	252	1	5	0	22	0	0	15
St. Paul	0	0		1	20	1	7	0	5	0	0	42
Missouri:												
Kansas City	0	0		0	75	0	1	0	47	0	0	14
St. Joseph	0	0		0	17	0	0	0	1	0	0	0
St. Louis	0	0	2	1	62	14	16	0	11	0	0	20
North Dakota:												
Fargo	0	0		0	5	0	1	0	1	0	0	1
Nebraska:												
Omaha	0	0		0	5	0	1	0	5	0	0	0
Kansas:												
Topeka	0	0		0	107	0	0	0	1	0	0	22
Wichita	0	0		0	2	2	4	0	3	0	0	11
SOUTH ATLANTIC												
Delaware:												
Wilmington	0	0		0	10	2	3	0	1	0	0	1
Maryland:												
Baltimore	1	0	6	1	162	7	21	0	74	0	0	76
Cumberland	0	0		0	0	0	1	0	1	0	0	0
Frederick	0	0		0	5	0	0	0	0	0	0	0
Dist. of Col.:												
Washington	0	0		1	123	2	5	0	18	0	0	38
Virginia:												
Lynchburg	0	0		0	5	0	0	0	0	0	0	7
Richmond	0	0		1	10	3	4	0	3	0	0	2
Roanoke	0	0		0	0	0	0	0	0	0	0	3
West Virginia:												
Wheeling	0	0		0	0	0	2	0	1	0	0	2
North Carolina:												
Winston-Salem	0	0		0	13	0	3	0	0	0	0	23
South Carolina:												
Charleston	0	0	3	0	4	0	0	0	2	0	0	2
Georgia:												
Atlanta	0	0	8	0	6	0	3	0	6	0	2	2
Brunswick	0	0		0	2	0	0	0	0	0	0	0
Savannah	0	0	5	0	2	1	0	0	0	0	0	1
Florida:												
Tampa	0	0		0	5	0	0	0	1	0	0	1
EAST SOUTH CENTRAL												
Tennessee:												
Memphis	0	0	2	0	113	1	5	0	5	0	0	15
Nashville	0	0		0	10	0	2	0	0	0	0	1
Alabama:												
Birmingham	0	0	3	1	15	2	6	0	0	0	0	2
Mobile	0	0	1	0	4	0	1	0	0	0	1	0
WEST SOUTH CENTRAL												
Arkansas:												
Little Rock	0	0		0	5	0	4	0	0	0	0	0
Louisiana:												
New Orleans	2	0		0	14	1	8	0	0	0	1	2
Shreveport	0	0		0	0	0	4	0	0	0	0	0
Texas:												
Dallas	2	0		0	2	1	5	0	1	0	0	7
Galveston	0	0		0	0	0	0	0	1	0	0	3
Houston	0	0		0	0	0	3	0	0	0	0	6
San Antonio	1	0	2	2	2	0	4	0	1	0	0	1

City reports for week ended May 15, 1943—Continued

	Diphtheria cases	Encephalitis, infectious, cases	Influenza		Measles cases	Meningitis, meningococcus, cases	Pneumonia deaths	Poliomyelitis cases	Scarlet fever cases	Smallpox cases	Typhoid and paratyphoid fever cases	Whooping cough cases
			Cases	Deaths								
MOUNTAIN												
Montana:												
Billings	0	0	------	0	2	0	0	0	0	0	0	2
Great Falls	1	0	------	0	30	0	2	0	2	0	0	2
Helena	0	0	------	0	29	0	1	0	0	0	0	6
Missoula	0	0	------	0	18	0	0	0	0	0	0	0
Idaho:												
Boise	0	0	------	0	2	0	0	0	0	0	0	0
Colorado:												
Denver	2	0	9	0	206	2	7	0	8	0	0	12
Pueblo	0	0	------	0	7	1	0	0	2	0	0	5
Utah:												
Salt Lake City	0	0	------	0	65	1	3	0	8	0	0	43
PACIFIC												
Washington:												
Seattle	0	0	------	1	167	0	5	0	2	0	0	13
Spokane	1	0	1	1	33	0	0	0	2	0	0	7
Tacoma	0	0	------	0	2	0	0	0	3	0	0	0
California:												
Los Angeles	3	0	8	0	181	0	3	1	29	0	0	56
Sacramento	0	0	------	0	5	2	2	0	5	0	0	15
San Francisco	0	0	3	1	87	1	19	1	25	0	0	41
Total	55	3	81	28	8,208	191	440	4	1,693	1	11	1,187
Corresponding week-1942	62	4	63	25	5,980	46	310	3	1,150	0	17	1,278
Average, 1938–42	77	------	90	[1] 20	[2] 5,931	------	[1] 341	------	1,496	10	22	1,240

[1] 3-year average, 1940–42.
[2] 5-year median.

Dysentery, amebic.—Cases: Boston, 1; New York, 9; Washington, 1; Los Angeles, 1.
Dysentery, bacillary.—Cases: Buffalo, 4; New York, 4; Philadelphia, 1; Detroit, 1; St. Louis, 6; Charleston, S. C., 6; Los Angeles, 7.
Dysentery, unspecified.—Cases: San Antonio, 17.
Leprosy.—Cases: Philadelphia, 1.
Rocky Mountain spotted fever.—Cases: Boise, 3.
Typhus fever.—Cases: Mobile, 2; Galveston, 1; Houston, 2.

Rates (annual basis) per 100,000 population, by geographic groups, for the 86 cities in the preceding table (estimated population, 1942, 34,540,300)

	Diphtheria case rates	Encephalitis, infectious, case rates	Influenza		Measles case rates	Meningitis, meningococcus, case rates	Pneumonia death rates	Poliomyelitis case rates	Scarlet fever case rates	Smallpox case rates	Typhoid and paratyphoid fever case rates	Whooping cough case rates
			Case rates	Death rates								
New England	2.5	0.0	7.5	5.0	1640	62.1	79.5	2.5	743	0.0	0.0	234
Middle Atlantic	7.1	1.3	7.1	3.1	979	38.8	70.5	0.0	279	0.0	2.7	112
East North Central	14.7	0.0	5.3	4.7	2030	20.0	51.8	0.6	277	0.6	0.6	185
West North Central	0.0	0.0	3.9	3.9	1094	35.2	70.4	0.0	190	0.0	0.0	254
South Atlantic	1.8	0.0	39.0	5.3	616	26.6	74.5	0.0	190	0.0	3.5	280
East South Central	0.0	0.0	35.6	5.9	843	17.8	83.2	0.0	30	0.0	5.9	107
West South Central	14.7	0.0	5.9	5.9	67	5.9	82.1	0.0	9	0.0	2.9	56
Mountain	24.1	0.0	72.4	0.0	2886	32.2	104.5	0.0	161	0.0	0.0	563
Pacific	7.0	0.0	21.0	5.2	830	5.2	50.7	3.5	115	0.0	0.0	231
Total	8.3	0.5	12.2	4.2	1239	28.8	66.4	0.6	256	0.2	1.7	179

PLAGUE INFECTION IN CALIFORNIA, NEW MEXICO, AND WASHINGTON

Plague infection has been reported proved on May 17 in a pool of 45 fleas from 4 ground squirrels, *C. beecheyi*, taken on a ranch 4 miles east of Crows Landing, Stanislaus County, Calif.; in a pool of 15 fleas from 9 prairie dogs, *Cynomys ludoviciamus arizonensis*, taken on April 22 from a location 1½ miles south of Corona, Lincoln County, N. Mex.; and in tissue from 1 rat taken on May 4 from a frame building in an industrial and commercial district of Tacoma, Pierce County, Wash.

FOREIGN REPORTS

CANADA

Provinces—Communicable diseases—Week ended May 1, 1943.—During the week ended May 1, 1943, cases of certain communicable diseases were reported by the Dominion Bureau of Statistics of Canada as follows:

Disease	Prince Edward Island	Nova Scotia	New Brunswick	Quebec	Ontario	Manitoba	Saskatchewan	Alberta	British Columbia	Total
Chickenpox	1	13	1	185	187	14	33	26	40	500
Diphtheria		16		19	2	6			1	44
Dysentery (bacillary)				23						23
German measles		4		22	110	2	1	25	7	171
Influenza		3	4		211	1	3		34	256
Measles		110	3	424	1,840	84	211	239	484	3,395
Meningitis, meningococcus		1		8	4			1		14
Mumps	2	70	7	70	904	66	76	62	104	1,361
Scarlet fever		46	39	42	421	26	36	43	29	682
Tuberculosis (all forms)	1	3	5	167	44	14		3	58	295
Typhoid and paratyphoid fever			1	28		2				31
Undulant fever				1						1
Whooping cough			1	75	134	39	22	29	39	339

CUBA

Habana—Communicable diseases—4 weeks ended May 1, 1943.—During the 4 weeks ended May 1, 1943, certain communicable diseases were reported in Habana, Cuba, as follows:

Disease	Cases	Deaths	Disease	Cases	Deaths
Diphtheria	28		Scarlet fever	2	
Leprosy	1		Tuberculosis	14	4
Malaria	10		Typhoid fever	30	6
Measles	21				

Provinces—Notifiable diseases—4 weeks ended April 24, 1943.—During the 4 weeks ended April 24, 1943, cases of certain notifiable diseases were reported in the Provinces of Cuba, as follows:

Disease	Pinar del Rio	Habana[1]	Matanzas	Santa Clara	Camaguey	Oriente	Total
Cancer	1	1	5	15		11	33
Chickenpox	1		1	1	8	20	31
Diphtheria	1	31	4	1		2	39
Hookworm disease				1			1
Leprosy		1				1	2
Malaria	38	2		2		101	143
Measles		10		1		1	12
Poliomyelitis				1		4	5
Rabies		1					1
Tuberculosis	23	17	23	32	17	64	176
Typhoid fever	9	48	8	27	19	14	125
Whooping cough				1			1

[1] Includes the city of Habana.

IRAQ

Cerebrospinal meningitis.—The following table shows the numbers of cases of cerebrospinal meningitis and deaths from the same cause in Basra Liwa, Iraq, for the period January 24 to April 17, 1943:

Week ended—	Cases	Deaths	Week ended—	Cases	Deaths
Jan. 30, 1943	10	0	Mar. 20, 1943	9	5
Feb. 6, 1943	4	2	Mar. 27, 1943	13	3
Feb. 13, 1943	2	0	Apr. 3, 1943	7	2
Feb. 20, 1943	9	1	Apr. 10, 1943	4	1
Feb. 27, 1943	7	3	Apr. 17, 1943	5	1
Mar. 6, 1943	9	3			
Mar. 13, 1943	10	1	Total	89	22

JAMAICA

Notifiable diseases—4 weeks ended May 8, 1943.—During the 4 weeks ended May 8, 1943, cases of certain notifiable diseases were reported in Kingston, Jamaica, and in the island outside of Kingston, as follows:

Disease	Kingston	Other localities	Disease	Kingston	Other localities
Chickenpox	10	37	Puerperal sepsis		2
Diphtheria	4	1	Tuberculosis	42	62
Dysentery	1	2	Typhoid fever	4	31
Leprosy		5	Typhus fever	2	1

REPORTS OF CHOLERA, PLAGUE, SMALLPOX, TYPHUS FEVER, AND YELLOW FEVER RECEIVED DURING THE CURRENT WEEK

NOTE.—Except in cases of unusual prevalence, only those places are included which had not previously reported any of the above-mentioned diseases, except yellow fever, during the current year. All reports of yellow fever are published currently.

A cumulative table showing the reported prevalence of these diseases for the year to date is published in the PUBLIC HEALTH REPORTS for the last Friday in each month.

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

Plague

Morocco—Casablanca region.—During the month of March 1943, 24 cases of plague were reported in the region of Casablanca, Morocco.

Smallpox

Algeria.—For the period April 11–20, 1943, 35 cases of smallpox were reported in Algeria, including 2 cases in Algiers and 12 cases in Cheliff.

Belgium.—For the week ended April 17, 1943, 1 case of smallpox was reported in the Province of Namur, Belgium.

Iran.—For the period December 6, 1942, to January 29, 1943, 203 cases of smallpox were reported in Iran.

Morocco.—For the month of March 1943, 253 cases of smallpox were reported in Morocco, including 1 case in Casablanca, 6 cases in Port Lyautey, 5 cases in Safi, and 1 case in Rabat.

Typhus Fever

Algeria.—For the period April 11–20, 1943, 464 cases of typhus fever were reported in Algeria, including 27 cases in Algiers, 38 cases in Bone, 13 cases in Philippeville, 56 cases in Oran, and 10 cases in Mostaganem.

Hungary.—During the week ended May 8, 1943, 34 cases of typhus fever were reported in Hungary.

Iraq.—For the month of April 1943, typhus fever (endemic and epidemic) was reported in Iraq as follows: Amara, 58 cases, 5 deaths; Baghdad, 71 cases; Basra, 111 cases, 29 deaths; Diwaniyah, 20 cases, 2 deaths; Diyala, 16 cases, 3 deaths; Dulaim, 3 cases; Erbil, 6 cases; Hilla, 6 cases; Kirkuk, 30 cases; Mosul, 109 cases, 11 deaths; Sulaimaniya, 57 cases, 1 death.

Mexico—Mexico, D. F.—Typhus fever has been reported in Mexico, D. F., Mexico, as follows: Weeks ended—March 6, 32 cases, 5 deaths; March 13, 25 cases, 3 deaths; March 20, 37 cases, 10 deaths.

Morocco.—For the month of March 1943, 3,759 cases of typhus fever were reported in Morocco, including 12 cases in Casablanca, 80 cases in Sale, 12 cases in Port Lyautey, and 21 cases in Safi.

Rumania.—For the week ended May 8, 1943, 305 cases of typhus fever were reported in Rumania, including 19 cases in Bucharest.

Slovakia.—For the week ended May 1, 1943, 6 cases of typhus fever were reported in Slovakia.

Spain.—For the week ended April 3, 1943, 9 cases of typhus fever were reported in Spain.

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