Tuberculosis Mortality in the United States, 1949.

(With provisional figures for the first 11 months of 1950)

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Few diseases have shown the continuous, yearly decline in mortality exhibited by tuberculosis. Mortality from tuberculosis continued to decline in 1949, when 39,000 deaths from all forms of the disease occurred in continental United States. The death rate is estimated to be 26.2 per 100,000 population, excluding the armed forces overseas. These preliminary figures, which are based on a 10-percent sample of death certificates, are subject to a sampling error of ±1.8 percent.

In 1948, there were 43,833 deaths from tuberculosis (all forms), and the death rate was 30.0 per 100,000 population. After allowance is made for changes in procedure which affect comparability of data for the 2 years, the tuberculosis death rate decrease between 1948 and 1949 appears to be about 9 percent. Provisional figures for the first 11 months of 1950 indicate a further decrease of approximately 15 percent. The death rate for that period was 22.6 as compared with 26.7 for the corresponding period of 1949.

This report, the eighth (*1–7*) in a series of annual statistical summaries of mortality figures for tuberculosis, presents estimated data for 1949 and the first 11 months of 1950 which are based on a 10-percent sample of death certificates. The report also discusses the significant changes since 1948, as well as sampling errors, and the comparability of 1949 and 1950 data with data for previous years.

Trend of the Tuberculosis Death Rate

The death rate for tuberculosis (all forms) has decreased almost continuously since 1900, except for a slight rise in 1917 and 1918. The

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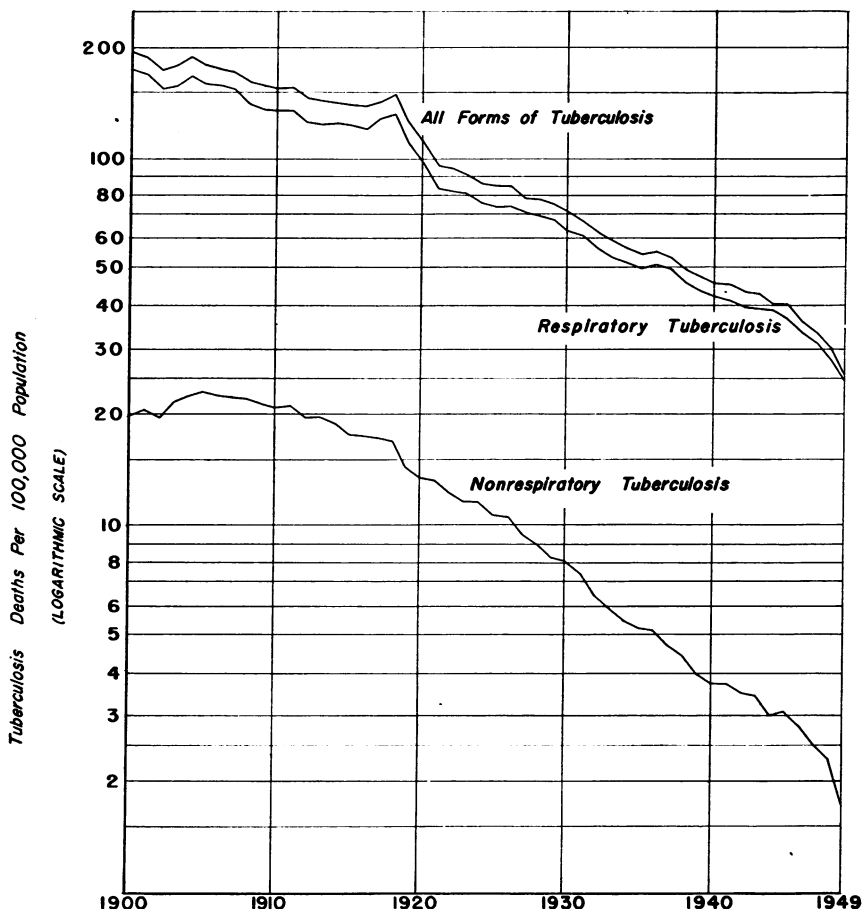
Figure 1. Death rates for tuberculosis (all forms), respiratory and nonrespiratory tuberculosis: death-registration States, 1900–1949. (The rates for 1949 are estimated from a 10-percent sample of death certificates).

interruption of the downward trend at the time of World War I was associated in part with the influenza epidemic. Death rates for respiratory tuberculosis and other forms of the disease for the period 1900–49 are shown on a semilogarithmic scale in figure 1, while data for all forms of the disease for 1940–49 appear in table 1.

Since 1918, the mortality rates for both respiratory and other forms of tuberculosis have decreased more rapidly than they did between 1900 and 1916. Comparison of the slopes of the curves for respiratory and nonrespiratory tuberculosis death rates, shows that the rate for the latter went down even more sharply than the rate for the former between 1921 and 1945. From 1945 to 1948, the relative decrease in the death rates for the two types of tuberculosis was about the same.

Between 1948 and 1949 the death rate for nonrespiratory tuberculosis appears to have been cut by one-fourth. This is a significant

Table 1. *Deaths and death rates for tuberculosis (all forms), by race and sex: United States, 1940–49*

Exclusive of deaths among armed forces overseas. Rates per 100,000 estimated midyear population in each specified group, excluding armed forces overseas]

Year	All races			White			Nonwhite		
	Both sexes	Male	Female	Both sexes	Male	Female	Both sexes	Male	Female
	Number								
1949 [1]	39,032	25,483	13,549	27,683	18,712	8,971	11,349	6,771	4,578
1948	43,833	28,552	15,281	31,750	21,616	10,134	12,083	6,936	5,147
1947	48,064	30,585	17,479	34,783	23,167	11,616	13,281	7,418	5,863
1946	50,911	31,886	19,025	37,340	24,283	13,057	13,571	7,603	5,968
1945	52,916	32,934	19,982	38,623	25,055	13,568	14,293	7,879	6,414
1944	54,731	33,717	21,014	39,958	25,596	14,362	14,773	8,121	6,652
1943	57,005	34,786	22,219	41,209	26,162	15,047	15,796	8,624	7,172
1942	57,690	34,801	22,889	41,306	25,899	15,407	16,384	8,902	7,482
1941	59,251	34,966	24,285	42,283	25,957	16,326	16,968	9,009	7,959
1940	60,428	35,795	24,633	43,211	26,598	16,613	17,217	9,197	8,020
	Rate								
1949 [1]	26.2	34.6	18.1	20.8	28.3	13.4	72.2	88.2	56.9
1948	30.0	39.4	20.8	24.3	33.3	15.4	78.4	92.1	65.4
1947	33.5	43.0	24.2	27.1	36.3	18.0	88.1	100.6	76.1
1946	36.4	46.2	26.9	29.8	39.2	20.6	92.3	106.2	79.2
1945	40.1	53.0	28.6	32.7	45.1	21.7	102.6	120.9	86.5
1944	41.3	53.1	30.5	33.7	45.0	23.3	106.2	122.7	91.3
1943	42.6	52.9	32.6	34.3	44.4	24.7	112.9	126.4	100.0
1942	43.1	52.3	34.0	34.4	43.3	25.6	118.4	131.4	106.0
1941	44.5	52.5	36.5	35.4	43.3	27.4	124.2	134.3	114.5
1940	45.8	54.1	37.5	36.5	44.7	28.2	127.6	138.7	116.9

[1] Estimated from a 10-percent sample of death certificates classified by the Sixth Revision of the International List.

decrease even after allowance is made for a sampling error of 6.7 percent. On the other hand, the net decrease between 1948 and 1949 in the death rate for respiratory tuberculosis amounted to only 8 percent.

Over the past 50 years, the death rate for respiratory tuberculosis has decreased 86 percent from 174.5 per 100,000 population in 1900 to 24.5 in 1949. In the same period, the rate for other forms of the disease has declined 91 percent, from 19.9 to 1.7.

Comparability of Data

At the beginning of 1949, several changes were made in mortality reporting and classification procedures which should be considered in comparing data for 1949 and previous years. These changes were: (1) revision of the list of causes of death which is used for classification; (2) a change in the principle of selecting the cause of death for primary tabulation purposes; and (3) changes in the death certificate form on which physicians report the cause or causes of death.

The International List of Causes of Death, which is used for classifying deaths, is revised decennially. The Sixth Revision (8) of this list became effective in 1949 and deaths reported in that year were

classified according to its categories. Deaths in 1939–48, however, were classified according to the Fifth Revision. Although the Sixth Revision provides for more detailed subdivision of tuberculosis than the Fifth, when the whole categories of respiratory tuberculosis and nonrespiratory tuberculosis are considered, the two revisions correspond closely.

Changes in the method of selecting one cause of death for statistical purposes in 1949, however, make data for that and subsequent years not strictly comparable with those for earlier years. In 1949, the principle of having the physician indicate the underlying cause of death for primary tabulations when reporting more than one cause was introduced in the United States. This is in accordance with regulations of the World Health Organization (9) and is in contrast to the previous procedure, based on the use of priority tables in the Manual of Joint Causes of Death (10). Under the latter procedure for selecting one cause of death when two or more were named on the death certificate, all of the diseases or conditions mentioned were taken into consideration, regardless of the sequence in which they were reported. The new form of medical certification on the death certificate, adopted in 1949, is designed to facilitate reporting of the *underlying* cause of death when more than one condition is involved.

The most important change in the reporting and classification procedure is from the use of priority tables to selection of the underlying cause reported by the physician. In the priority tables used until 1949, tuberculosis took precedence over most other causes even when it was only contributory to another pathological condition reported with it. Under the new procedure, when tuberculosis is mentioned as a contributory cause of death, but heart disease, cancer, or some other condition is named as the underlying cause, the death is assigned to the latter. Thus, under the procedure introduced in 1949, tuberculosis loses some of its former priority and yields to other chronic diseases or conditions when they are considered more important in the physician's opinion.

An index of the effect of these changes on cause-of-death statistics has been obtained through the development of comparability ratios (table 2). Deaths in 1949 were classified separately according to the Fifth Revision (using the Joint Cause Manual) and also by the Sixth Revision, selecting the underlying cause of death. Comparability ratios were then calculated by dividing the number of deaths arrived at under the Sixth Revision by the number arrived at under the Fifth. These ratios are important in comparing mortality statistics for 1949 and earlier years, since they make it possible to determine to what extent a change in the number of deaths assigned to a cause is due to the change in the certification and classification procedures, as contrasted with a real change in the force of mortality. It should be

Table 2. *Percentage changes in death rates for tuberculosis, by age, 1948 to 1949 and comparability ratios for tuberculosis deaths classified by the Sixth and Fifth Revisions, by age: United States, 1949*

Age (years)	Tuberculosis (all forms)			Respiratory tuberculosis			Tuberculosis, other forms		
	Total percentage change 1948–49 [1]	Comparability ratio 1949 [2]	Net percentage change 1948–49 [3]	Total percentage change 1948–49 [1]	Comparability ratio 1949 [2]	Net percentage change 1948–49 [3]	Total percentage change 1948–49 [1]	Comparability ratio 1949 [2]	Net percentage change 1948–49 [3]
All ages	−12.7	0.958	−8.7	−11.6	0.956	−7.5	−26.1	0.986	−26.1
Under 1	−4.0	.971	−1.0	−11.9	.923	−5.1	0	1.000	0
1–14	−8.1	.985	−5.6	+6.3	.912	+13.3	−19.0	1.062	−22.7
15–24	−17.3	.991	−16.4	−17.1	.983	−15.5	−20.0	1.074	−25.0
25–44	−13.2	.981	−11.6	−11.5	.979	−9.7	−38.1	1.033	−40.9
45–64	−13.6	.953	−9.3	−12.5	.952	−8.1	−30.8	.984	−30.8
65–74	−12.2	.920	−4.6	−12.0	.927	−5.1	−14.6	.811	+6.1
75 and over	−2.0	.909	+7.8	−0.1	.918	+8.8	−33.3	.733	−9.7

[1] Computed from rates based on final 1948 data classified by the Fifth Revision and 1949 data estimated from a 10-percent sample classified by the Sixth Revision.
[2] Ratio of deaths in a 10-percent sample classified by the Sixth Revision of the International List to deaths classified by the Fifth Revision.
[3] Computed from rates based on final 1948 data with allowance for change in classification and 1949 data estimated from a 10-percent sample classified by the Sixth Revision.

noted, however, that the ratios are provisional since they are based on the 10-percent sample.

The provisional comparability ratios (table 2) which indicate the combined effect of the changes in procedures in 1949 mentioned above show that deaths from tuberculosis (all forms) at all ages, when classified by the Sixth Revision and the new procedures, are 95.8 percent of the number assigned to tuberculosis by the Fifth Revision and the previous method of joint-cause selection. The comparability ratio of 0.958 may be used to determine what part of the difference between the crude death rate for tuberculosis in 1949 and its counterpart for 1948 is due to a difference in comparability, and what part reflects a real change in mortality. When the tuberculosis death rate of 30.0 for 1948 is multiplied by the comparability ratio of 0.958, the rate of 28.7 so obtained is comparable to the rate of 26.2 for tuberculosis in 1949, since it indicates approximately what the rate would have been in 1948 if the data had been classified by the Sixth Revision. Consequently, the net decrease from 1948 to 1949 which may be regarded as a real change in mortality is 2.5 deaths per 100,000, or 8.7 percent.

Examination of the comparability ratios in table 2 shows that there is some variation in the comparability at different ages. The ratio decreases from 0.991 for deaths of persons 15 to 24 years to 0.909 for persons 75 years and over. For deaths at ages under 45 years, disagreement between the two procedures is less than 3 percent, since the ratios range from 0.971 for infants to 0.991 for deaths in the age group 15 to 24 years. In the older age groups, from 4.7 to 9.1 percent fewer deaths were assigned to tuberculosis by the Sixth than by the Fifth Revision.

Comparability ratios computed separately for respiratory tuberculosis and other forms of the disease by age show that in the three age groups between 1 and 44 years, fewer deaths are classified as respiratory tuberculosis by the Sixth Revision than by the Fifth, but more fall into the nonrespiratory tuberculosis group. However, since the ratios for nonrespiratory forms of the disease are based on small frequencies in the sample, a small relative increase in the number of deaths assigned to nonrespiratory tuberculosis by the Sixth Revision is not significant.

Under the Sixth Revision, fewer deaths at the older ages were assigned to both respiratory and nonrespiratory tuberculosis. This would indicate that physicians regard such conditions as diseases of the heart, vascular lesions affecting the central nervous system, and cancer as the underlying cause of death, even though tuberculosis is also present.

With regard to race and sex, there is some variation in the comparability ratios for tuberculosis. Fewer deaths of white persons are assigned to tuberculosis by the new classification procedure than when the Joint Cause Manual and Fifth Revision are used. The ratios are 0.938 for respiratory, and 0.963 for other forms of the disease. On the other hand, there is close correspondence between the numbers of deaths of nonwhites assigned to respiratory tuberculosis by the two classification procedures, the ratio being 1.003. There is a small, but insignificant increase in the number of deaths of nonwhites assigned to nonrespiratory forms of the disease by the new procedures.

The difference between comparability ratios for whites and nonwhites is probably associated with the fact that more than one cause of death is reported on a larger proportion of death certificates for white persons than·for nonwhites. This is more likely to result in assignment of the death to a cause other than tuberculosis when the physician designates the underlying cause, than when the priority tables of the Joint Cause Manual are used.

For deaths of white persons, comparability ratios for males and females are different. The ratio for respiratory tuberculosis for white males is 0.926 as compared with 0.967 for white females. For deaths from nonrespiratory tuberculosis, the ratios are 0.944 for white males and 0.988 for females. This difference in comparability ratios, which is not apparent for nonwhites, may be associated with differences in the proportion of certificates on which more than one cause of death is reported. On the other hand, if confirmed by final figures, this difference in the ratios for males and females may suggest that tuberculosis ends fatally in the absence of other significant diseases relatively more often among females than among males.

It should be noted that comparability ratios have been computed

by age alone, irrespective of race and sex. Therefore, when comparing the data for 1949 with those of 1948 (tables 4 and 6), allowance can be made for changes in comparability by age only by applying the comparability ratios shown in table 2 to figures for all races and both sexes.

Deaths and Death Rates

In 1949, the total estimated number of tuberculosis deaths were distributed as follows: 36,400, or 93 percent, were attributed to respiratory tuberculosis and the remaining 2,600, or 7 percent, to the nonrespiratory forms of the disease. It is therefore evident that respiratory tuberculosis deaths continue to constitute an increasingly greater proportion of all deaths from the disease.

An examination of tuberculosis mortality data by race and sex (table 3) shows higher death rates among males than females and among nonwhites than whites. Data for respiratory tuberculosis, which constitutes over 90 percent of all deaths from the disease, show a similar distribution pattern. The respiratory tuberculosis mortality rate of 32.5 for males is almost twice that of 16.6 per 100,000 for females, and the rate of 66.3 for nonwhites is more than 3 times the rate of 19.6 for whites. For the two sexes within each racial group, the relative difference appears to be greater for whites than for nonwhites. The rate of 26.9 for white males is more than double the rate of 12.3 for white females, whereas the rate of 80.8 for nonwhite males is 54 percent higher than that of 52.5 for nonwhite females.

Table 3. *Estimated deaths and death rates for tuberculosis of the respiratory system and for other forms, by race and sex: United States, 1949*

[Estimated from a 10-percent sample of death certificates classified by the Sixth Revision of the International List. Exclusive of deaths among armed forces overseas. Rates per 100,000 estimated midyear population in each specified group, excluding armed forces overseas].

Race and sex	Respiratory tuberculosis			Other forms of tuberculosis		
	Number	Rate	Percent error	Number	Rate	Percent error
All races	36,444	24.5	1.8	2,588	1.7	6.7
Male	23,988	32.5	2.0	1,495	2.0	8.2
Female	12,456	16.6	2.8	1,093	1.5	9.6
White	26,023	19.6	1.9	1,660	1.2	7.8
Male	17,790	26.9	2.4	922	1.4	10.4
Female	8,233	12.3	3.5	738	1.1	11.6
Nonwhite	10,421	66.3	3.1	928	5.9	10.4
Male	6,198	80.8	4.0	573	7.5	13.2
Female	4,223	52.5	4.9	355	4.4	16.8

Mortality from nonrespiratory tuberculosis evidences race-sex differentials similar to those for mortality from respiratory tuberculosis. The rate for males is 33 percent higher than the rate for females and the rate for nonwhites is nearly five times that of whites.

The ratio of respiratory tuberculosis to other forms of the disease is 50 percent greater for white males than for any other population group.

Death Rates by Age and Race

Generally, the tuberculosis mortality patterns for age and race, and for age and sex observed in 1948 also hold true for the estimates for 1949, exhibiting the usual peaks in infancy and old age.

Variations in the rates with age show the same general tendencies for both races (table 4 and fig. 2), but the points of greatest mortality tend to occur at a slightly younger age for nonwhites than for whites. For both races, the lowest rates are in the age group 1–14 years. The rate rises continuously and reaches a peak for whites in the age group 75 years and over, but this maximum for nonwhites is attained earlier, in the 65–74 age group.

As in 1948, variations in mortality for whites and nonwhites are particularly striking. The mortality peak among nonwhites is 82.1 percent higher than the peak for whites. The rate of 72.2 for nonwhites of all ages is over three times that of 20.8 for whites. For whites, the estimated age-specific rates for tuberculosis (all forms) showed significant decreases from 1948 to 1949 for persons in the age groups from 15–64 years. Taking into account the comparability ratio, it appears that the greatest percentage decrease, amounting to 20 percent, occurred in age group 15–24. There was no significant
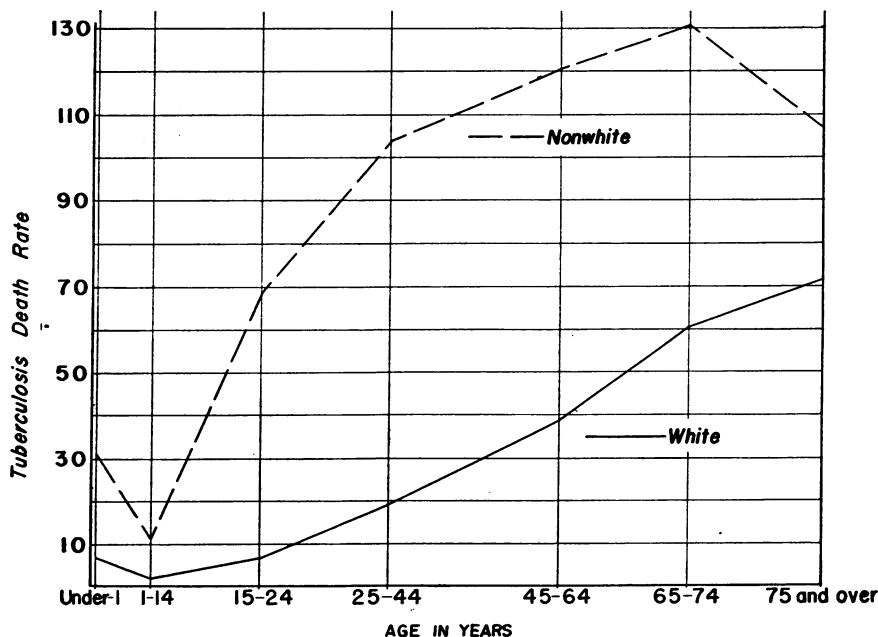
Figure 2. Estimated death rates for tuberculosis (all forms) by age and race: United States, 1949.

Table 4. *Deaths and death rates for tuberculosis (all forms), by age and race: United States, 1948 and 1949*

[Deaths for 1949 estimated from a 10-percent sample of death certificates classified by the Sixth Revision of the International List. Deaths for 1948 classified by the Fifth Revision. Exclusive of deaths among armed forces overseas. Rates per 100,000 estimated midyear population in each specified group, excluding armed forces overseas]

Race and year	Total	Under 1 year	1–14 years	15–24 years	25–44 years	45–64 years	65–74 years	75 years and over
					Number			
All races:								
1949	39,032	315	1,248	3,198	12,489	14,008	4,993	2,675
1948	43,833	325	1,311	3,933	14,164	15,902	5,545	2,623
White:								
1949	27,683	200	680	1,347	7,762	10,902	4,340	2,386
1948	31,750	190	803	1,811	8,962	12,663	4,893	2,410
Nonwhite:								
1949	11,349	115	568	1,851	4,727	3,106	653	289
1948	12,083	135	508	2,122	5,202	3,239	652	213
					Rate			
All races:								
1949	26.2	9.6	3.4	14.3	28.3	45.7	65.0	74.6
1948	30.0	10.0	3.7	17.3	32.6	52.9	74.0	76.1
White:								
1949	20.8	6.9	2.1	6.9	19.6	38.9	60.4	71.9
1948	24.3	6.6	2.6	9.0	23.0	45.9	69.9	75.5
Nonwhite:								
1949	72.2	31.0	11.8	69.3	104.4	120.2	130.9	107.8
1948	78.4	36.1	11.0	78.9	116.2	128.7	133.6	83.5

change in the rates for nonwhites from 1948 to 1949, according to estimates based on the 10-percent sample. The percentage errors of the estimated numbers of deaths and death rates for 1949 by age, race, and sex appear in table 5.

Death Rates by Age and Sex

Death rates specific for sex as well as age are shown in table 6. The mortality pattern for males is similar for both 1948 and 1949, with the low and high points of the curve occurring at the same age-groups—low at 1–14 years and rising to a peak at 65–74 years. For females, the death rate decreases after infancy, and is lowest in the 1–14-year age group. The curve, which is somewhat W-shaped, rises in the 25–44-year age group to 24.0, after which it again drops off; then it registers a comparatively pronounced rise in the last two age groups, reaching a maximum of 57.4 at 75 years and over.

Table 5. *Estimated percentage sampling error of estimated number of deaths and death rates for tuberculosis (all forms), by age, race, and sex: United States 1949*

Race and sex	Total	Under 1 year	1–14 years	15–24 years	25–44 years	45–64 years	65–74 years	75 years and over
All races, both sexes	1.8	17.8	9.0	5.6	2.8	2.7	4.5	6.1
White	1.9	22.4	12.1	8.6	3.6	3.0	4.8	6.5
Nonwhite	3.0	29.5	13.3	7.4	4.6	5.7	12.4	18.6
Male	2.0	25.5	13.3	8.9	3.8	3.0	5.2	8.0
Female	2.7	24.9	12.1	7.2	4.3	6.0	8.6	9.4

Table 6. *Deaths and death rates for tuberculosis (all forms), by age and sex: United States, 1948 and 1949*

[Deaths for 1949 estimated from a 10-percent sample of death certificates classified by the Sixth Revision of the International List. Deaths for 1948 classified by the Fifth Revision. Exclusive of deaths among armed forces overseas. Rates per 100,000 estimated midyear population in each specified group, excluding armed forces overseas]

Sex and year	Total	Under 1 year	1-14 years	15-24 years	25-44 years	45-64 years	65-74 years	75 years and over
				Number				
Both sexes:								
1949	39,032	315	1,248	3,198	12,489	14,008	4,993	2,675
1948	43,833	325	1,311	3,933	14,164	15,902	5,545	2,623
Male:								
1949	25,483	154	565	1,250	7,075	11,187	3,626	1,550
1948	28,552	172	631	1,526	8,035	12,529	3,974	1,666
Female:								
1949	13,549	161	683	1,948	5,414	2,821	1,367	1,125
1948	15,281	153	680	2,407	6,129	3,373	1,571	957
				Rate				
Both sexes:								
1949	26.2	9.6	3.4	14.3	28.3	45.7	65.0	74.6
1948	30.0	10.0	3.7	17.3	32.6	52.9	74.0	76.1
Male:								
1949	34.6	9.2	3.0	11.3	32.9	73.5	97.6	95.1
1948	39.4	10.4	3.5	13.5	37.9	83.5	109.5	105.8
Female:								
1949	18.1	10.1	3.8	17.3	24.0	18.3	34.5	57.4
1948	20.8	9.7	3.9	21.0	27.5	22.4	40.7	51.0

For males, the mortality rate exceeds the rate for females for all age groups, except under 25 years, and the total rate for males is nearly twice as high as the rate for females. Even though the death rate for females rises more steeply than the rate for males from 1–14 years to 15–24, it levels off subsequently, and the two lines formed by the rates cross between the 15–24 and 25–44-year age groups (fig. 3). This same crossing of the two lines has been observed in tuberculosis prevalence rates based on chest X-ray survey data.

For males in the age groups between 25–64, there were significant decreases from 1948 to 1949. In the age groups under 25 and 65 and over, on the other hand, no significant change in mortality was recorded. Tuberculosis mortality rates for females also declined from 1948 to 1949 with decreases occurring in the age groups from 15–64 years. The changes for the remaining age groups among the females which appear as increases for the youngest and oldest groups, are not significant. Consequently, it is concluded that no change in mortality occurred for females under 15 years or 65 years and over.

Provisional Death Rates for 1950

Provisional figures for 1950, based on a 10-percent mortality sample for January through November, show a further pronounced decline in the tuberculosis death rate amounting to 15 percent. The death rate for this period of 1950 was 22.6, as compared with 26.7 for January to November, 1949. Death rates by age for the first 11
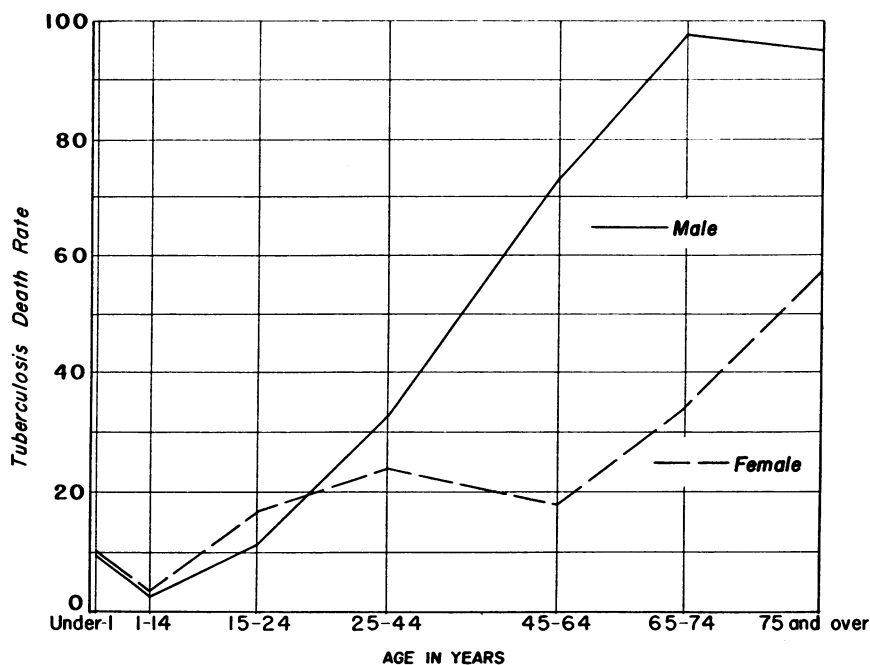
Figure 3.   Estimated death rates for tuberculosis (all forms) by age and sex: United States, 1949.

months of 1950 are shown in table 7 for all forms of tuberculosis, together with comparable ones for the corresponding period of 1949. These figures show significant decreases for each age group except those under 15 years and 75 years and over.   On a relative basis, the decreases range from nearly 9 percent for the group 65–74 years to 23 percent for the 25–44-year age group.   For the youngest and the oldest age groups, however, there is no significant difference between the rates for the two periods.   Since the underlying cause of death was selected and the Sixth Revision used for both the 1949 and the

Table 7.   *Provisional death rates for tuberculosis (all forms), by age: reporting area,*[1] *January–November, 1949 and 1950*

[Based on a 10-percent sample of death certificates received monthly in State Vital Statistics offices.   Exclusive of deaths among armed forces overseas.   Rates on an annual basis per 100,000 estimated population in each specified age group, excluding armed forces overseas]

Age (years)	January–November	
	1950	1949
All ages	22. 6	26. 7
Under 15	3. 7	4. 2
15–24	11. 9	15. 1
25–44	22. 2	28. 9
45–64	41. 2	46. 0
65–74	60. 5	66. 4
75 and over	69. 5	74. 0

[1] The reporting area covered the United States with the exception of certain cities which were excluded in 1 or more months.

1950 data, there is no question of comparability, and the rates can be compared directly.

The decrease in tuberculosis mortality in 1950 appears to have taken place rather generally throughout the United States. Although figures by State cannot be estimated from the 10-percent sample, the provisional death rates for the four geographic regions of the country (table 8) indicate significant decreases in each region. It is likely that the relative decrease was slightly less in the Northeastern States (11 percent) than in the other regions. It is encouraging to see that the death rate for tuberculosis declined further in 1950 and that the rate of decline, which has increased since 1945, was at least as great as in 1949.

Table 8. *Deaths in a 10-percent sample and provisional death rates for tuberculosis (all forms), by geographic region: reporting area,[1] January–November, 1949 and 1950*

[By place of death. Exclusive of deaths among armed forces overseas. Rates on annual basis per 100,000 estimated population excluding armed forces overseas]

Area	January–November		January–November	
	1950	1949	1950	1949
	Number		Rate	
Reporting area	3,124	3,623	22.6	26.7
Northeastern States	875	971	24.0	27.0
North Central States	762	900	18.5	22.1
Southern States	1,108	1,296	26.1	31.0
Western States	379	456	21.1	26.7

[1] The reporting area covered the United States with the exception of certain cities which were excluded in 1 or more months.

NOTE: The Northeast includes the New England and Middle Atlantic States; the North Central Region includes the East and West North Central States; the South includes the South Atlantic, East and West South Central States; the West includes the Mountain and Pacific States.

Source and Limitations of the Data

The 10-percent sample of death certificates, from which the estimates for 1949 and provisional figures for the first 11 months of 1949 and 1950 have been made, is a systematic sample drawn monthly in State vital statistics offices. Transcripts of the sample death certificates are forwarded to the Public Health Service where shipments from each State and the District of Columbia are classified and tabulated monthly.

Estimates of the 1949 tuberculosis deaths are made by multiplying the sample frequency by 10 and adjusting the figure proportionately, together with data for other causes, to an independent estimate of all deaths based on a complete count of death certificates received in State vital statistics offices. Correction for bias in the sample returns is made in proportion to the underreporting or overreporting in the 1947 and 1948 samples, which have been compared with the final

figures for those years. The estimated percentage errors shown in table 5 reflect random sampling variation.

The death rates discussed in this report are based on the estimated population of the United States, excluding members of the armed forces overseas. The population estimates are provided by the Bureau of the Census. For the war years, the difference between the population excluding the armed forces abroad and that which includes them makes an appreciable difference in the death rates for tuberculosis. However, for more recent years, the difference is very small. In 1949, there was a difference of less than 1 percent between rates for males computed from the two populations, and no difference in the crude death rate for tuberculosis (7).

Summary

This report presents data on tuberculosis mortality in the United States derived from a 10-percent sample of death certificates in 1949 and the first 11 months of 1950, summarizing the changes which have occurred. Included is a discussion of factors which affect the comparability of data for 1949 and 1950 with those for earlier years, and provisional comparability ratios are given. In 1949 4 percent fewer deaths are assigned to tuberculosis (all forms) by the classification procedures and the Sixth Revision of the International List of Causes of Death introduced in 1949 than by the Fifth Revision and Joint Cause Manual.

Deaths from all forms of tuberculosis in 1949 are estimated to have numbered 39,000. The estimated death rate of 26.2 per 100,000 population is 9 percent lower than the rate for 1948, after allowance is made for the changes in classification procedure. A further decrease of approximately 15 percent appears to have occurred in 1950. Changes in mortality from both respiratory tuberculosis, which accounts for 93 percent of the deaths, and nonrespiratory forms of the disease contribute to the decreases.

Among white persons, decreases in the death rate between 1948 and 1949 appear to have been confined to the 15–64-year age groups. In the preliminary figures, no change was recorded in the death rates for nonwhite groups.

Death rates by age for both males and females, irrespective of race, were lower in 1949 than in 1948. The significant decreases were those recorded for males between the ages of 25 and 64 years and for females aged 15 to 64 years. In comparing age-specific tuberculosis death rates for January through November 1950 with those for the corresponding period of 1949, it is found that decreases occurred for persons between 15 and 64 years of age. The four geographic regions shared in the decreases in the death rate for all forms of tuberculosis in 1950.

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Experimental Error in the Determination of Tuberculin Sensitivity

By Sven Nissen Meyer, M.D., Anna Hougen, Cand. Act., and Phyllis Edwards, M.D.*

In tuberculin testing, the size of a skin reaction is frequently used as a measure of the degree of tuberculin sensitivity without regard for the unavoidable errors in the technical procedures of giving and reading the tests.

With an exactly measured dose of tuberculin, a uniform technique, and faultless measurement of the reaction, a theoretically exact determination of sensitivity should be possible. In practice, however, such conditions can never be fulfilled. Small uncontrollable variations will always occur in the amount of tuberculin injected, in the depth of injections, and in defining the boundary of the induration.

These variations will cause the observed value of a reaction to deviate more or less from what may be called "the true level of sensitivity" of the person in question. Sometimes a whole series of small errors may act in one direction, causing the observed value to deviate considerably from the true. More often the individual errors of an observation will be partly positive, partly negative, and thus to some extent neutralize each other, resulting in a smaller total error. It is for this reason that experimental errors, expressed as positive or negative deviations from the true value, usually have an approximately normal distribution with a mean around zero.

An estimate of the "true value" of sensitivity and of the quantitative importance of experimental error would be obtained if it were possible to measure the sensitivity many times by repeated tests in the same person. The mean of all the observations, provided their distribution is fairly symmetrical, would then be an estimate of the true value of the sensitivity level. The standard deviation of the distribution, or its variance (i. e., the square of the standard deviation) would be a measure of the magnitude of variations caused by all uncontrollable factors in the technique employed. This measure would depend explicitly on the investigator; a careful tester would, by using the same method routinely, obtain a smaller dispersion in his results than a less precise worker.

Testing the same person many times is, however, rarely feasible and in addition it is possible that sensitivity may change during, or

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even be changed by, the investigation. Information about experimental error can be obtained, however, by simultaneously giving duplicate tests with the same dose of tuberculin to a sufficient number of persons. The two corresponding reactions will reflect the same level of sensitivity and the difference between them will be caused by all experimental errors involved in both tests. The distribution of these differences in the total group will have a variance twice as large as the variance of the experimental error.

The total experimental error can be regarded as a sum of two main components—errors made in giving tuberculin and errors made in reading the reactions. An estimate of the latter can be obtained when one person makes duplicate readings of the same reactions. The distribution of the difference between the two readings, as with duplicate reactions, will have a variance twice as large as the variance of the reading error.

In a series of studies designed primarily for other purposes and conducted during May–November 1950 by the Tuberculosis Research Office of the World Health Organization, it was frequently possible to perform duplicate testing and reading. These data have been utilized in order to estimate the magnitude of errors involved in determining tuberculin sensitivity.

Technique

The same general principles for technique and procedure were used in six different investigations from which material is derived for this study.

The research personnel were nurses who had been carefully trained and were considerably experienced in the type of work being performed. Throughout each study the same individual gave all the tests and another read all the reactions in order to minimize variations due to personal differences in technique. To avoid bias in reading reactions, the reader had no knowledge of the dose of tuberculin used, previous reactions of the subject, or observations made by another reader when there was one. Secretaries recorded all results, so the readers had no opportunity to see the cards.

Tests in all six studies were given by the Mantoux method, intradermally in the dorsal forearm, injecting 0.1 cc. of carefully measured PPD, batch RT XIX–XX–XXI,[1] freshly prepared at the State Serum Institute in Copenhagen. When one person was given two tests simultaneously, care was taken that the injections were placed as symmetrically as possible on the two arms.

All readings were made after 3 days. Erythema was first measured and recorded, then the reaction was carefully palpated and the transverse diameter of the induration was measured. Finally, qualitative

[1] RT XXII used in Study III.

characteristics of reactions were observed by classifying induration into four groups or types.[2] Type I is a very dense, hard, elevated induration, occasionally accompanied by ulceration. Type IV, at the other extreme, is a soft, indefinite reaction, perhaps best described as an edematous swelling which might be missed unless carefully palpated. Types II and III represent intermediate degrees of density. Type II is characterized as less dense and less elevated than type I. Type III indurations are those which, though clearly and definitely palpable, are not so dense as type II.

Material

Study I, planned specifically to investigate experimental error in tuberculin testing, was carried out in Oslo among 114 persons, of whom 99 were medical students and 15 were office personnel. There were 91 men and 23 women ranging in age from 19 to 49 years with 85 percent between 20 and 29 years. Seventy-five men and 18 women were previously BCG-vaccinated; the rest stated that they had been positive to previous tuberculin tests.

The study plan was to give all persons two simultaneous tests with 10 T.U.,[3] one test in each arm. Since 32 persons refused this dose, they were tested with 5 T.U. in each arm, and the other 82 received 10 T.U. as planned. Taking groups of about 30 persons, 2 readers first read all reactions in the left arm, then all reactions in the right arm, and, finally, each reaction in the left arm again.

Study II, designed primarily to determine the degree of reaction to different doses of tuberculin, was carried out in a rural district of Denmark among 557 school children ranging in age from 8 to 15 years. Each child was given 5 T.U. in one arm, alternating between left and right. In the other arm one of the following doses was given: 1 T.U., 5 T.U., 10 T.U. Reading of the reaction on the left arm was followed immediately by reading of the right arm. The 124 out of 375 previously BCG-vaccinated children, who, because of the randomization of tests, received 5 T.U. in both arms, were used for the present purpose. Ninety-nine out of these 124 children were also read by a second reader in reading a sample of the entire group of 557.

Study III, a somewhat similar study, was carried out at a mental hospital in Denmark. Of the 1,852 patients tested, there were 839 men and 1,013 women ranging in age from 15 to 95 years. Two teams were used, team I testing the relatively quiet, ambulatory patients and team II testing the unruly or bedridden patients. All patients were given 5 T.U. in one arm, alternating between left and

[2] Method developed by Carroll E. Palmer, M. D., Director, Tuberculosis Research Office, World Health Organization.

[3] 1 T.U.=0.00002 mgm. reference standard PPD or 0.01 mgm. international standard O.T.

right. In the other arm one of the following doses was given if tested by team I: 1.25 T.U., 2.5 T.U., 5 T.U., 10 T.U., or if tested by team II: 5 T.U. Reactions on both arms were read at one time, one reader for each team. The 297 patients given 5 T.U. in both arms by team I and the 179 patients tested by team II were used for analysis.

Study IV, designed to determine the sensitivity level among tuberculosis patients,[4] was carried out at a tuberculosis hospital in Copenhagen. Of the 245 patients tested with 1 T.U., there were 118 women and 127 men ranging in age from 14 to 76 years. The readings were made by 2 readers independently and in a sample (104 patients) the reactions were read twice by both readers.

Study V, a large-scale series of projects, still in progress among school children in rural Denmark, is concerned with variations in vaccines as reflected by post-vaccination allergy. At the time of the 9-week retesting of project IV [5] with 5 T.U., two readers frequently were available to read the reactions independently. Material from 2 such occasions is used in this paper, group A comprising 182 children and group B, 191.

Study VI, conducted in Mexico, was made by a special research team using 2 different vaccines and retesting after 10 weeks with 10 T.U. On 1 day of the retesting among 253 school children, tuberculin reactions were read by 2 readers.

A summary is given in table 1 of the material used from the six studies described above showing, for each study, the number of observations, mean size of induration, and standard deviation of the distribution of reactions read by each reader.

Results

Starting with the observations made by reader I in study I, where the reactions on the left and right arms were read separately, a correlation of reactions according to size of induration is shown in table 2. The size of reactions on the left arm is indicated by the horizontal scale, on the right arm by the vertical scale. Reactions which are of the same size on both arms are located along a diagonal line drawn from the upper left-hand corner to the lower right. Spread of observations on either side of this line expresses variations due to experimental error.

If the size of induration on the right arm of each individual is subtracted from the size of induration on his left arm, the differences for the 114 persons will be distributed as shown below:

[4] Edwards, Phyllis, and Guld, Johs.: Tuberculin sensitivity: A study of 245 tuberculous patients. Acta tuberc. Scandinav. (in press).

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Table 1. *Basic data of 6 studies used for investigation of experimental errors in tuberculin reactions with Mantoux tests*

Study No.	Number tested	Reader	First reading Left arm		First reading Right arm		Number tested	Second reading Left arm	
			Mean induration in mm.	Standard deviation	Mean induration in mm.	Standard deviation		Mean induration in mm.	Standard deviation
1	114	I	10.2	5.8	10.1	5.5	114	10.6	5.9
	114	II	9.8	6.1	9.0	5.5	114	9.3	5.4
2	124	I	7.5	5.2	7.2	5.1			
	99	II	7.4	5.5	7.4	5.4			
3	[1]297	I	11.8	6.0	11.1	5.3			
	[2]179	I	10.5	6.3	9.9	6.1			
4	245	I	13.2	4.2			104	13.7	3.4
	245	II	12.4	3.6			104	11.9	3.3
5a	182	I	11.3	4.7					
	182	II	11.5	5.0					
5b	191	I	12.4	4.9					
	191	II	11.4	4.8					
6	253	I	14.8	5.5					
	253	II	11.9	4.4					

[1] Team I.
[2] Team II.

NOTE: For the empty boxes no observations have been made.

Difference in size of induration in mm.

−8 −7 −6 −5 −4 −3 −2 −1 0 1 2 3 4 5 6 7 8

Number of persons

1 0 1 1 1 9 9 19 29 18 9 7 7 2 0 0 1

This distribution has a mean of 0.1, which is close to zero, as would be expected. The variance of this distribution is $S_D^2 = 5.47$. The variance of the experimental error will be half of 5.47, i. e., $S_E^2 = 2.74$, because each difference is composed of errors made in two tests. The standard error of an observed value of a reaction will be the square root of 2.74, $S_E = \sqrt{2.74} = 1.65$.

Similar analyses were made for all the data used for this investigation and the results are presented in summary form in the tables that follow.

Table 3 concerns those studies where each individual received two tests with the same dose of tuberculin. It shows the mean difference in size of induration between reactions on left and right arms, the variance of the distribution of these differences, and estimated standard deviation for the total experimental error. It will be noted that the latter varies between 1.65 mm. and 2.27 mm.; the largest figure was obtained from testing of unruly and bedridden mental patients.

Table 2. *Distribution by size of induration in right arm and left arm, 114 persons, study I, reader I*

Mm. of induration on left arm

Mm. of induration on right arm	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	Total
0	2	1		1																						4
1																										
2	1		2	1	1																					5
3			2	2	1	1																				6
4			1		2	1																				4
5				1	3	4			2		1			1												12
6						1			1	1	1	1														5
7									2		1	1		1												5
8			1				1		1	1		1														5
9				1					2		2	1	2	1												9
10										1	1		2													4
11											1	1	1	2	2											8
12												2	1	2	2	1										8
13												1		3	3	1										9
14														1	1	1										3
15											1			1	1	1		1	1		1					6
16																	1		1							2
17									1							1	1			1			1			4
18														1				4	2				1			8
19															1	1	1						1			4
20																			1							1
21																										
22																						1				1
23																										
24																									1	1
Total	3	1	6	6	6	9	6	1	9	4	10	5	8	9	6	2	2	4	8	3	1	1	3	---	1	114

Table 3. *Analysis of differences for studies where duplicate tests have been performed on the 2 arms (left arm minus right arm)*

Study No.	Number tested	Reader	Mean differences in mm.	S_D^2	$\sqrt{1/2\,S_D^2}$
1	114	I	0.1	5.47	1.65
	114	II	0.8	6.29	1.78
2	99	I	0.4	8.99	2.12
	99	II	0.0	8.26	2.03
3	[1] 297	I	0.7	7.83	1.98
	[2] 179	I	0.6	10.28	2.27

[1] Team I.
[2] Team II.

In a corresponding way, table 4 concerns studies where reactions were read twice by the same reader. The estimated standard deviation for the reading error varies between 1.09 mm. and 1.43 mm. These figures are in general smaller than those on the preceding table as they pertain only to the reading error since the errors in giving tests are not involved in duplicate readings.

Table 5 concerns studies in which a number of reactions have been read once by each of two persons. The difference between their results and the variance of the distribution of the differences have been computed as on preceding tables. In this case, the variance is equal to the sum of the reading variance for each reader, and it is not possible

Table 4. *Analysis of differences for studies where duplicate readings of the same reaction have been made by the same reader (first reading minus second reading)*

Study No.	Number tested	Reader	Mean differences in mm.	S_D^2	$\sqrt{1/2\ S_D^2}$
1	114	I	−0.4	3.68	1.36
	114	II	+0.5	4.12	1.43
4	104	I	−0.5	3.38	1.30
	104	II	+0.3	2.38	1.09

to determine what proportion of the total variance belongs to each reader. In order to calculate the standard deviation of the reading error, the assumption has been made that the reading variance is the same for both readers.

Table 5. *Analysis of differences for studies where the same reactions have been read by two readers (reader I minus reader II)*

Study No.	Number tested	Arm	Mean differences in mm.	S_D^2	$\sqrt{1/2\ S_D^2}$
1	114	Left	0.4	5.45	1.65
	114	Right	1.1	5.32	1.63
2	99	Left	0.6	4.43	1.49
	99	Right	0.1	2.63	1.15
4	245	Left	0.8	4.47	1.50
5a	182	Left	−0.2	3.24	1.27
5b	191	Left	1.0	2.66	1.15
6	253	Left	2.9	4.97	1.58

From two of the studies where duplicate tests as well as duplicate readings are available, it is possible to obtain some idea of the relative amount of the total error attributable to each of its two components: giving of tests and reading of tests. In study I, about two-thirds of the variance of the total error is due to reading errors. In study II, however, reading errors account for scarcely half of the variance of the total error.

If the materials in these studies are grouped by dose of tuberculin (5 and 10 T.U. used in study I), by type of reaction (I–IV), and source of the allergy (from BCG or natural infection), no significant variation in the total error and reading error can be demonstrated.

The observed size of reactions to Mantoux 1–10 T.U. thus appears to have a standard error of roughly 2 mm. This figure was computed from all reactions, large and small, in each of the six studies, and represents a very general result which must be qualified. It is not possible from this material to determine whether this standard error of 2 mm. applies equally at all levels of sensitivity.

It clearly cannot apply to very small reactions because negative

deviations cannot possibly be of the same size as positive deviations the closer the true value of the reaction comes to zero. At these lower levels, the distribution of errors will, therefore, be skewed. It is reasonable to assume that a reaction somewhere near the middle of the total range should have a fairly normal distribution of errors. Under this assumption if the true value of a reaction is 10 mm., there would be a 95 percent chance that the observed value would be between 6–14 mm.; 5 out of 100 would be either above 14 mm. or below 6 mm. In very practical terms this means that if the lowest level for a positive reaction is at 6 mm., essentially all reactions whose true value is 10 mm. would be called positive. It would be desirable to know whether such limits could also be applied to reactions at a lower sensitivity level, particularly around the 5–6 mm. level where the border line is commonly placed between positive and negative. Unfortunately, it is not possible to say from this material what the standard error at this level may be, as it becomes progressively less certain as reactions approach zero.

Another method of analysis may be used, however, to obtain pertinent information concerning how often experimental errors may cause a reaction apparently to change from negative to positive or vice versa. Table 6 is designed to illustrate the frequency of such changes. The material from all studies in which two tests with the same dose of tuberculin were given has been grouped according to size of induration on the left arm. For each of these groups the distribution is shown by size of induration on the right arm.

Table 6. *Analysis of duplicate Mantoux tests grouped by size of induration on left arm and distributed by size of induration on right arm, 714 persons*

Mm. induration to test on left arm	Number tested	Distribution by size of induration to test on right arm				
		0–2 mm.	3–5 mm.	6–8 mm.	9–11 mm.	12 mm. and over
0	53	46	7	0	0	0
1	6	3	3	0	0	0
2	25	12	12	1	0	0
3	30	8	17	4	1	0
4	34	6	23	4	1	0
5	32	3	21	6	2	0
Total 0–5 mm	180	78	83	15	4	0
Percentage	100	43.4	46.1	8.3	2.2	0
6	27	0	7	13	5	2
7	21	1	2	12	5	1
8	37	0	6	20	8	3
9	32	0	3	11	12	6
Total 6–9 mm	117	1	18	56	30	12
Percentage	100	0.8	15.4	47.9	25.6	10.3
10–14	227	1	5	20	96	105
15–19	146	0	2	2	12	130
20 mm. and over	44	0	0	1	1	42
Total 10 mm. and over	417	1	7	23	109	277
Percentage	100	0.2	1.7	5.5	26.2	66.4

If 6 mm. is arbitrarily taken as the lower limit for a positive reaction, it will be noted that of 180 persons whose first test was negative, 10.5 percent would be called positive on the second test. This change in classification occurred essentially among persons whose response to the first test was 3–5 mm. of induration.

Conversely, of 117 persons showing 6–9 mm. of induration at the first test, i. e., positive to usual criteria, 16.2 percent would be negative to the other test. Of 417 persons with strong positive reactions (10 or more mm.) to the first test, 1.9 percent would be called negative to the second test.

It may be pertinent to point out that differences of such a degree have been shown in these research studies where duplicate tests were given by the same tester with careful attention to uniform technique and read by the same experienced reader. Greater differences due to experimental error must be expected when repeated tests are performed by different individuals in routine examinations.

Summary

1. The paper reports the results of an investigation on the measurement of experimental error in giving and reading Mantoux tests.

2. Duplicate tests with 0.00002 mg., 0.0001 mg. or 0.0002 mg. PPD, and/or duplicate readings of the same reactions were made in 100–300 persons in each of six different studies carried out by the Tuberculosis Research Office, May–November 1950.

3. The total experimental error of an observed size of induration, composed of errors made in giving and reading tests, has a standard deviation of roughly 2 mm.; the reading error alone is about 1.3 mm.

4. In low levels of tuberculin sensitivity where the distribution of errors cannot be considered normal, the estimate of the standard deviation of the total experimental error does not apply.

5. Data are presented to show how frequently experimental errors can cause a reaction to appear to change from negative to positive and vice versa. Using 6 mm. of induration as the border line, positive reactions on the right arm were recorded in 10.5 percent of the persons who had negative reactions on the left arm, essentially among those with 3–5 mm. of induration. Conversely, 16.2 percent of the weak positive reactors, those with 6–9 mm. of induration on the left arm, had a negative reaction on the right arm. Of the strong positive reactors—more than 10 mm. of induration—1.9 percent were negative on the second test.

Atabrine Therapy of Histoplasma Infections in Mice

By Charlotte C. Campbell and Samuel Saslaw, M.D.[*]

The specific agents now generally available for the therapy of bacterial and rickettsial infections have not been effective in diseases of mycotic origin other than nocardiosis and actinomycosis. The search therefore continues for nontoxic drugs which may be effective in the treatment of such diseases as coccidioidomycosis, histoplasmosis, cryptococcosis, blastomycosis, and other potentially systemic mycoses.

Bockman (1) has reported that atabrine (quinacrine hydrochloride) in concentrations of 25 to 50 mg. percent is fungicidal for *Cryptococcus neoformans in vitro*, but concludes that the high concentrations which are required for fungicidal action rule out *in vivo* study. Since the work done at this laboratory with several strains of *Histoplasma capsulatum* appeared to confirm the fact that relatively high concentrations of the drug are necessary for growth inhibition, it was felt that the drug's *in vitro* degree of activity against histoplasma organisms was insufficient to promise effectiveness *in vivo*.

Parmer (2), however, has demonstrated that, 4 hours after intramuscular administration of atabrine in rabbits, the spleens, livers, rib marrow, and lymph nodes of these animals yielded much higher concentrations of the drug than did plasma. Since these macrophage-rich tissues are typically involved in human histoplasmosis, the potentialities of atabrine as a therapeutic agent for the disease were deemed worthy of investigation. Studies were therefore undertaken to determine the effects of atabrine therapy on the mortality rates of mice experimentally infected with histoplasma organisms.

Preliminary Studies

In Vitro Experiments

In order to determine the effects of atabrine on the growth of *H. capsulatum*, preliminary experiments were performed in Mycophil [1] broth containing the following respective concentrations of the drug: 0.075, 0.15, 0.30, 0.60, and 0.75 mg. per ml. The broth was dispensed to tubes in 10 ml. quantities and inoculated with 0.1 ml. suspensions of 5-day-old cultures, after which the tubes were incubated for 60 days at 28° C. Inoculated broth which contained no atabrine was used

as a growth control. The contents of all the tubes were subcultured in broth without atabrine at weekly intervals.

Three strains of *H. capsulatum* were used in these studies. Growth occurred in the control tube which contained no atabrine and in the one containing 0.075 mg. per ml., but none was apparent in the tubes containing higher concentrations of the drug. Subcultures from the 0.15 and 0.30 mg. per ml. concentrations were consistently negative over the 8-week period of incubation.

Toxicity Tests

Groups of 10 mice each were given daily intraperitoneal injections of 0.5 ml. aqueous solutions of atabrine for 30 consecutive days. Daily dosages for the respective groups amounted to 0.075, 0.15, 0.30, 0.60, 0.75, and 0.90 mg. There were no deaths among the mice receiving 0.75 mg. of the drug for the 30-day test period. At the end of this period, however, 40 percent of the test mice receiving the 0.90 mg. dosage had succumbed.

Therapeutic Studies

Young, white Swiss mice (Bagg strain), weighing 14 to 17 gm. were used throughout these studies. In each experiment, the animals were divided at random into groups of five, and each group was placed in an individual jar. Infections were induced by the intraperitoneal injection of approximately 3.5 million yeast-phase organisms (strain G–8) [2] suspended in 0.5 ml. of 5 percent hog gastric mucin, as described previously by the authors (*3*).

Among the animals in the treatment groups, atabrine therapy was begun on the day of infection. Daily injections of the drug were given intraperitoneally for 30 days. In one group of experiments, however, atabrine therapy was delayed for 1 week following infection. All drug dosages were delivered in 0.5 ml. sterile, distilled water.

Animals serving as drug controls in each experiment received 30 injections of the highest concentration of atabrine used in the experiment. Those serving as organism, or infected, controls received only a single challenge inoculum suspended in 0.5 ml. of mucin. A representative number (2 of each group of 5) of the infected animals that died during the course of the experiments, as well as of those surviving at the end of the studies, were necropsied and their spleens cultured for *H. capsulatum*.

Dosage Level Experiments

These experiments were conducted for two purposes: (1) to determine whether the mortality rates of infected mice could be reduced

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[2] A human isolate identified at this institution.

by the daily parenteral administration of atabrine, and (2) to establish the drug level at which such a reduction could be demonstrated. Of a total of 215 mice infected, five groups of 35 were given daily atabrine dosages of 0.075, 0.15, 0.30, 0.60, and 0.75 mg., respectively, for a period of 30 days. (These amounts, selected on the basis of the preliminary studies, approximated 5, 10, 20, 40, and 50 mg. per kg. of body weight, respectively.) The remaining 40 mice received no atabrine and served as organism controls.

Table 1 compares the results obtained at each atabrine dosage level in treated animals with those obtained among the untreated controls. Since the majority of deaths occurred during the second and third weeks following infection, the relatively few deaths within the first 3 days after inoculation were attributed to nonspecific causes and were not included in the statistical evaluations. In treated mice receiving the three smaller drug dosages, the respective death rates were 24 out of 35 (68.6 percent), 26 out of 34 (76.5 percent), and 26 out of 35 (74.3 percent). None of these rates differs appreciably from the rate among the untreated controls (26 out of 39, or 66.7 percent).

Table 1. *Comparative mortality among mice receiving atabrine and those receiving no atabrine following infection with* H. capsulatum (*atabrine dosages of 0.075–0.75 mg.*)

Experimental subjects	Daily ata- brine dosage (mg.)	Number dead/number observed				Death rates (percent)			
		After 7 days	After 14 days	After 21 days	After 30 days	After 7 days	After 14 days	After 21 days	After 30 days
Infected animals (dosage approximately 3.5 million yeast-phase *H. capsulatum* organisms suspended in 0.5 ml. of 5 percent hog gastric mucin).	[1]None	2/39	15/39	24/39	26/39	5.1	38.5	61.5	66.7
	0.075	2/35	19/35	23/35	24/35	5.7	54.3	65.7	68.6
	.15	4/34	15/34	24/34	26/34	11.8	44.1	70.6	76.5
	.30	0/35	12/35	25/35	26/35	0	34.3	71.4	74.3
	.60	0/33	2/33	7/33	14/33	0	6.1	21.2	42.4
	.75	0/32	3/32	8/32	9/32	0	9.4	25.0	28.1
Drug controls [2]	.75	0/10	0/10	0/10	0/10	0	0	0	0

[1] Infected controls.
[2] Uninfected animals. Received atabrine only.

However, lower mortality rates were observed in the groups receiving atabrine dosages of 0.60 mg. and 0.75 mg., respectively. In the former, the death rate was 42.4 percent (14 deaths among 33 animals), while in the latter, it was only 28.1 percent (9 deaths among 32 animals). At necropsy, no gross lesions were observed in either the treated or untreated animals, but *H. capsulatum* was consistently isolated from the spleens of mice in both groups.

While there were no deaths among the drug-control animals receiving daily atabrine injections of 0.75 mg. during both the preliminary and the therapeutic studies, there were indications (such as failure to grow or gain weight) to suggest that this dosage approached the level of maximum tolerance. In subsequent studies, therefore, atabrine dosages in excess of 0.60 mg. were excluded.

In an effort to determine the variations which could be expected in biologic studies of this type, five separate additional studies were conducted in which the preceding experiments were repeated in modified form. Freshly prepared suspensions of the organism in mucin and atabrine dosage schedules of 0.30 and 0.60 mg. were employed in each experiment.

The results of the five separate studies, which were conducted at monthly intervals, are shown in table 2. The combined death rate for the untreated control groups was 74.4 percent (145 out of 195), which is significantly greater than the rates of 58.0 percent (83 out of 143), and 41.1 percent (58 out of 141), for the groups receiving 0.30 and 0.60 mg. of atabrine, respectively. As in the preceding experiments, the peak of mortality occurred during the second and third weeks following infection, and relatively few animals died during the fourth week. No mice in either the treated or untreated groups died after the thirtieth day. Again, necropsy of infected animals revealed no gross lesions, but positive cultures were obtained from the spleens of both treated and untreated animals.

Table 2. *Comparative mortality among mice receiving atabrine and those receiving no atabrine following infection with* H. capsulatum *(atabrine dosages of 0.3 and 0.6 mg.); results of 5 separate experiments*

Experimental subjects	Daily atabrine dosage (mg.)	Experiment 1	Experiment 2	Experiment 3	Experiment 4	Experiment 5	All experiments
		Number dead/number observed [1]					
Infected animals (dosage approximately 3.5 million yeast-phase *H. capsulatum* organisms suspended in 0.5 ml. of 5 percent hog gastric mucin)	[2] None	26/39	34/40	28/37	26/39	31/40	145/195
	0.30	26/35	11/20	14/20	5/19	27/49	83/143
	.60	14/34	11/20	8/18	7/20	18/50	58/141
Drug controls [3]	.60	0/10	0/10	0/10	0/10	0/10	0/50
		Death rates (percent)[1]					
Infected animals (dosage approximately 3.5 million yeast-phase *H. capsulatum* organisms suspended in 0.5 ml. of 5 percent hog gastric mucin)	[2] None	66.6	85.0	75.7	66.6	77.5	74.4
	0.30	74.3	55.0	70.0	26.3	55.1	58.0
	.60	41.1	55.0	44.4	35.0	36.0	41.1
Drug controls [3]	.60	0	0	0	0	0	0

[1] 30 days after infection.
[2] Infected controls.
[3] Uninfected animals. Received atabrine only.

Delayed Treatment Experiments

In order to determine the effect of atabrine on well-established histoplasma infections, 100 mice were infected with *H. capsulatum*, and treatment was not initiated until 7 days later when the animals appeared to be very ill. At that time, daily injections of 0.60 mg. of atabrine were begun in 50 of the mice.

As indicated in table 3, 43 of the 50 treated mice (86.0 percent) died, as compared with 39 of 48 untreated controls (81.3 percent). Thus, the mortality rate of mice with established infections did not appear to be altered by atabrine treatment.

Table 3. *Comparative mortality among mice receiving delayed atabrine therapy and those receiving no therapy following infection with* H. capsulatum *(daily atabrine dosage of 0.60 mg. begun 7 days following infection)*

| Experimental subjects	Daily atabrine dosage (mg.)	Number dead/number observed				Death rates (percent)			
		After 7 days	After 14 days	After 21 days	After 30 days	After 7 days	After 14 days	After 21 days	After 30 days
Infected animals (dosage approximately 3.5 million yeast-phase *H. capsulatum* organisms suspended in 0.5 ml. of 5 percent hog gastric mucin).	[1] None	1/48	28/48	33/48	39/48	2.1	58.3	68.7	81.3
	0.60	1/50	29/50	38/50	43/50	2.0	58.0	76.0	86.0
Drug controls [2]_____	.60	0/10	0/10	0/10	0/10	0	0	0	0

[1] Infected controls.
[2] Uninfected animals. Received atabrine only.

Discussion

The studies described in this report indicate that the mortality rates of mice experimentally infected with *H. capsulatum* are reduced significantly following daily intraperitoneal injections of atabrine begun at the time of infection. Moreover, survival rates appear to increase with increasing dosages of the drug up to and including the dosage level of 0.75 mg. per day (approximately 50 mg. per kg. of body weight). This parallelism is demonstrated graphically in the chart.

As noted, viable histoplasma organisms were consistently recovered from all treated survivors, regardless of the atabrine dosage level employed. This would appear to suggest that the drug exerts a suppressive rather than a lethal effect on *H. capsulatum in vivo*, and may account in part for the failure of the drug to reduce mortality rates among mice included in the delayed treatment studies. Since massive doses of the organism were suspended in a virulence "enhancing" menstruum and introduced by an artificial route to induce mortality in excess of 50 percent in untreated animals, it is possible that the infectious process was too firmly established to respond to atabrine when therapy was begun. This type of infection may be comparable to widely disseminated histoplasmosis in man, and since the macrophage tissues are heavily parasitized in such advanced infections, it probably would be most difficult to demonstrate the effectiveness of any therapeutic agent.

On the other hand, the possibility that atabrine may be of value in the therapy of less overwhelming infections is encouraged by
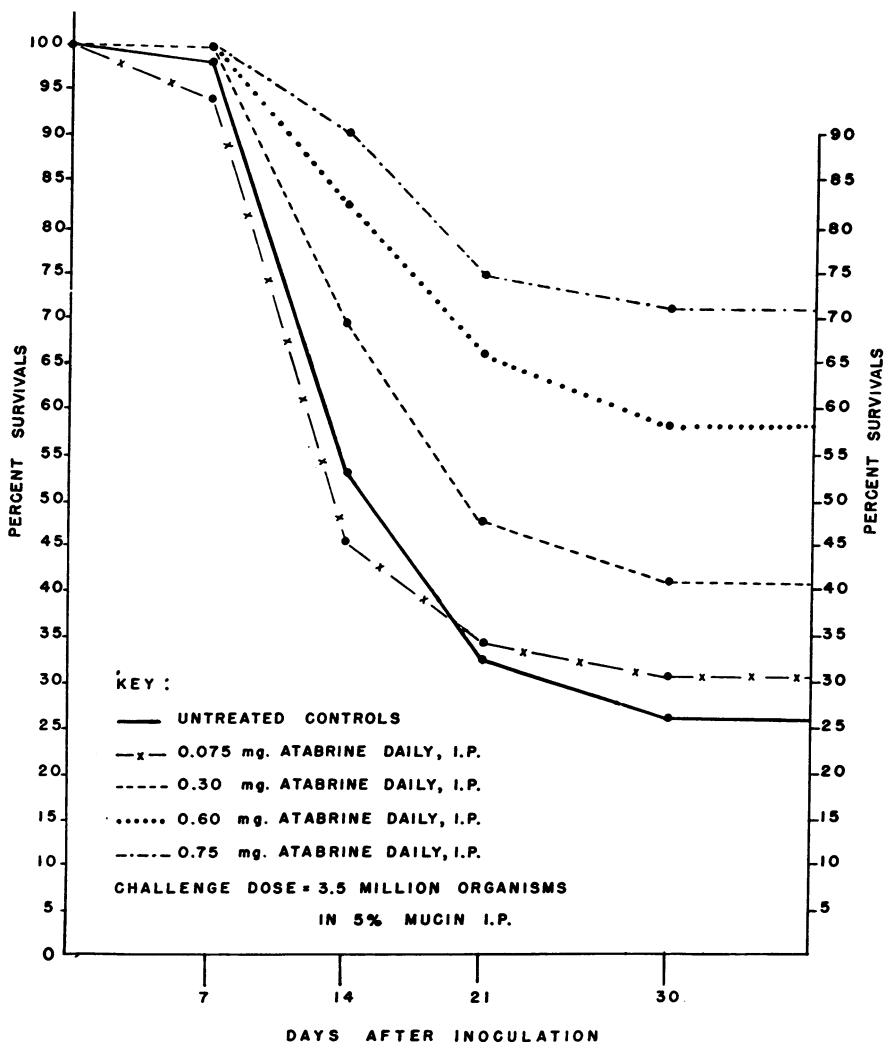
Effect of increasing doses of atabrine on survival rate of mice infected with *H. capsulatum*.

Parmer's (*2*) observation that high concentrations of atabrine are deposited in the reticulo-endothelial tissue, which *H. capsulatum* so readily involves. Four hours after rabbits had received intramuscular injections of 8 mg. of atabrine per kg. of body weight, Parmer found that 40.62, 7.78, 5.08, and 3.70 mg. per kg., respectively, could be recovered from the spleen, liver, rib marrow, and lymph nodes. Plasma levels, however, were only 0.08 mg. per kg. of body weight.

In further experiments with patients, moreover, Parmer found that bone-marrow concentrations were as high as 20 times those of plasma.

In the present studies, the effective atabrine dosage for experimental mouse infections corresponded to 40 mg. per kg. of body weight,

which, on an equivalent weight basis, would suggest a daily dosage for humans of 2.8 gm. for an average 70 kg. individual. Obviously, this would be a toxic regimen, but it is possible that the differences between experimental histoplasma infections in mice and spontaneous infections in humans may permit the use of lower atabrine dosages in the latter. Moreover, it is also possible that the successful suppression of histoplasma infections may be influenced by the early administration of the drug before widespread involvement of tissue has occurred, as is the case in plasmodial infections.

Because proved cases of human histoplasmosis do recover spontaneously (4–7), and, furthermore, because of the disease's diversity of symptoms and its severity in man, the ultimate evaluation of any therapeutic agent for the disease will be quite difficult to accomplish. Nevertheless, since no other nontoxic agents for the treatment of this disease exist, the findings reported in this paper would suggest that atabrine be given clinical trial in human histoplasma infections.

One further observation should be noted regarding the studies reported in this paper. During the experiments, the authors received the impression that male mice were more susceptible than females to histoplasma infection. Available data were therefore segregated according to sex, and the resulting summary (table 4) showed an over-all mortality rate for both treated and untreated males which was 26.1 percent higher than that for females. In the untreated control groups alone, deaths among males totaled 49 out of 58 (84.5 percent), as against 34 out of 60 (56.7 percent) among females. Moreover, in the groups receiving atabrine dosages varying from 0.075 to 0.60 mg., male deaths totaled 93 out of 149 (62.4 percent), whereas the female deaths amounted to only 65 out of 149 (43.6 percent). These differences are significant and suggest that the sex of animals should be considered in evaluating studies of this nature.

Table 4. *Comparative mortality, by sex, among mice receiving atabrine and those receiving no atabrine following infection with* H. capsulatum

Experimental subjects	Daily atabrine dosage (mg.)	Number dead/ number observed		Death rates (percent)	
		Males	Females	Males	Females
Infected animals (dosage approximately 3.5 million yeast-phase *H. capsulatum* organisms suspended in 0.5 ml. of 5 percent hog gastric mucin.	None 0.075– 0.60	49/58 93/149	34/60 65/149	84.5 62.4	56.7 43.6
Totals_____	_____	142/207	99/209	68.6	47.4

Summary

1. Atabrine concentrations of 0.15 mg. per ml. inhibited the growth of *H. capsulatum in vitro*.

2. Mice experimentally infected with yeast-phase histoplasma organisms were given daily intraperitoneal injections of atabrine in dosages of 0.075, 0.15, 0.30, 0.60, and 0.75 mg., respectively, for 30 consecutive days. The animals treated with the daily dosage of of 0.60 mg. experienced a survival rate of 58.9 percent compared with 26.1 percent for untreated controls. The daily administration of 0.75 mg. of the drug resulted in a survival rate of 71.9 percent as against 33.3 percent for the control group. When therapy (0.60 mg. of atabrine) was withheld for 1 week following infection, mortality rates proved to be roughly the same for both treated and untreated animals. *H. capsulatum* was isolated from the spleens of both treated and untreated mice, indicating that the effect of atabrine is suppressive rather than lethal.

3. The results of these studies suggest the trial of atabrine in the therapy of human histoplasmosis.

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! Incidence 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 April 14, 1951]

Influenza

In collaboration with the Influenza Information Center, National Institutes of Health, the following report on influenza has been prepared.

The total number of influenza cases reported for the current week was 5,898, a 19-percent decrease from the 7,253 reported last week and a 12-percent decrease from the 6,737 cases reported for the corresponding week last year.

Dr. A. A. Jenkins, Utah State Department of Health, reports a total of 265 cases of influenza in Davis County (population 30,000) for the week ended March 9. Since that time, paired blood specimens have been examined by Dr. Glenn R. Leymaster, University of Utah College of Medicine. The diagnosis has been confirmed in three out of six serum specimens. The total number of reported cases in Utah for March was 538.

Dr. J. V. Irons of the Texas State Department of Health Laboratories, reports influenza A-prime virus isolated recently from two cases of influenza in Austin. Of nine cases clinically diagnosed as influenza, three have shown significant titer rises against influenza A and A-prime and two have shown significant titer rises only against influenza A-prime.

Dr. E. H. Lennette, Director of the Regional Laboratory at Berkeley, Calif., reports significant antibody rises in the complement fixation test against influenza A in 36 paired serum specimens tested from March 31 to April 13.

The laboratory at the National Institutes of Health reports an increase in titer for influenza A-prime in one paired serum specimen and for both A and A-prime in three paired sera obtained at a military installation in Virginia.

Epidemiological Reports

Conjunctivitis

Dr. D. S. Fleming, Minnesota Department of Health, reports an outbreak of 24 cases of acute conjunctivitis between February 7 and March 12 among 400 employees of a produce plant. Twenty-three of

the cases occurred among 90 employees eviscerating chickens. No cases were known in family or community contacts.

The conjunctivitis was limited to one eye with severe edema of the lids and considerable involvement of the ocular conjunctiva. The cornea was not involved. Recovery was usually complete in a week with no sequela. There were no constitutional symptoms. The condition did not cause any loss of time from employment. Bacteriological studies from swabs of the conjunctiva were not productive. Virus studies are being carried out at this time.

Infectious Hepatitis

Dr. W. J. Murphy, Director, Division of Epidemiology, Georgia Department of Public Health, reports six cases of infectious hepatitis among children at one school during February and March. The children ranged in ages from 6 to 13 years. No evidence suggested a common source of infection.

Dr. A. W. Freeman, Maryland State Department of Health, reports outbreaks of infectious jaundice. Twenty-seven cases have occurred in one State institution since January 1. Person-to-person spread is indicated but not a common origin. Another outbreak seemingly centered about public schools is now under investigation.

Botulism

Dr. W. L. Halverson, California Director of Public Health, telegraphed a report of one case of botulism diagnosed April 2, 3 days after the patient tasted home-canned string beans that appeared spoiled and musty in odor. The patient is expected to recover. After one bean was eaten, the remainder of the jar was thrown to chickens and ducks. Twenty to thirty of the fowl died of typical limberneck

Food Poisoning

The Food and Drug Officer, United States Food and Drug Administration, adds the following to the report last week of Dr. W. L. Halverson. Investigation of the food poisoning outbreak among 25 diners at a popular resort area restaurant occurred March 10 at Palm Springs, Calif. Apparently the item eaten in common was corned beef obtained from a Los Angeles sausage company. Specimens of the corned beef sent to the State laboratory at Berkeley showed a heavy growth of coagulase-positive staphylococcus organisms. One patient, a doctor, sent a specimen to a Chicago laboratory. All investigations have not yet been completed by State and other agencies concerned.

Dr. W. L. Halverson reported by telegraph, an outbreak of food poisoning involving eight cases in two families with suspected source reported as chicken sandwiches. No food was available for laboratory examination.

Comparative Data For Cases of Specified Reportable Diseases: United States

[Numbers after diseases are International List numbers, 1948 revision]

Disease	Total for week ended—		5-year median, 1946–50	Seasonal low week	Cumulative total since seasonal low week		5-year median, 1945–46 through 1949–50	Cumulative total for calendar year—		5-year median, 1946–50
	Apr. 14, 1951	Apr. 15, 1950			1950–51	1949–50		1951	1950	
Anthrax (062)	1	1	1	(¹)	(¹)	(¹)	(¹)	23	9	15
Diphtheria (055)	60	107	127	27th	4,242	6,504	9,334	1,335	2,233	2,976
Encephalitis, acute infectious (082)	20	24	7	(¹)	(¹)	(¹)	(¹)	222	199	119
Influenza (480–483)	5,898	6,737	1,597	30th	117,891	132,582	132,582	103,349	121,998	121,998
Measles (085)	23,253	12,248	25,616	35th	249,154	138,692	285,679	220,453	119,562	250,733
Meningitis, meningococcal (057.0)	138	104	104	37th	2,597	2,356	2,294	1,636	1,442	1,322
Pneumonia (490–493)	1,676	2,348	(²)	(¹)	(¹)	(¹)	(¹)	³ 30,333	38,638	(²)
Poliomyelitis, acute (080)	71	62	32	11th	216	249	119	1,428	1,380	721
Rocky Mountain spotted fever (104)	------	------	3	(¹)	(¹)	(¹)	(¹)	4	16	13
Scarlet fever (050) ⁴	2,140	1,513	2,234	32d	49,903	42,834	64,215	34,212	26,395	40,604
Smallpox (084)	------	------	2	35th	13	39	58	5	18	37
Tularemia (059)	13	16	16	(¹)	(¹)	(¹)	(¹)	210	316	316
Typhoid and paratyphoid fever (040, 041) ⁵	39	39	43	11th	156	171	176	591	681	681
Whooping cough (056)	1,454	2,467	2,149	39th	44,833	60,221	60,221	23,231	38,685	32,906

¹ Not computed.
² Data not available.
³ Addition: Florida, week ended Mar. 31, 39 cases.
⁴ Including cases reported as streptococcal sore throat.
⁵ Including cases reported as salmonellosis.

Reported Cases of Selected Communicable Diseases: United States, Week Ended Apr. 14, 1951

[Numbers under diseases are International List numbers, 1948 revision]

Area	Diphtheria (055)	Encephalitis, infectious (082)	Influenza (480–483)	Measles (085)	Meningitis, meningococcal (057.0)	Pneumonia (490–493)	Poliomyelitis (080)
United States	60	20	5,898	23,253	138	1,676	71
New England	3	4	191	911	5	66	1
Maine			165	24	1	34	
New Hampshire			9	26		4	
Vermont				170			
Massachusetts	3	4		435	2		
Rhode Island			2	2		1	
Connecticut			15	254	2	27	1
Middle Atlantic	7	6	495	3,114	19	233	3
New York	4	5	¹473	1,071	10	65	1
New Jersey		1	22	591	1	74	1
Pennsylvania	3			1,452	8	94	1
East North Central	8	6	36	3,972	31	157	7
Ohio	4			1,479	16		
Indiana	1	1	7	269	3	17	
Illinois		2	20	460	6	87	3
Michigan	3	2	9	458	2	53	4
Wisconsin		1		1,306	4		
West North Central	3	1	90	1,092	7	42	14
Minnesota	1		3	87	3	11	3
Iowa				92	2	2	5
Missouri	1		10	236	2	3	
North Dakota	1	1	26	69		21	
South Dakota			47	45		4	5
Nebraksa				39			1
Kansas			4	524		1	
South Atlantic	18	1	2,359	1,786	20	219	10
Delaware			1	14			
Maryland	3		16	110	2	36	
District of Columbia				38	1	13	
Virginia	5	1	717	776	3	70	1
West Virginia	1		1,142	158	2	19	
North Carolina	3			140	9		2
South Carolina	3		92	103	1	54	
Georgia			391	339	2	27	2
Florida	3			108			5
East South Central	7	1	325	838	13	162	8
Kentucky			120	504	5	7	3
Tennessee	5		158	99	5		1
Alabama	1			143	1	93	2
Mississippi	1	1	47	92	2	62	2
West South Central	11	1	1,309	5,271	27	622	11
Arkansas	1		1,173	335	3	103	
Louisiana	1		7	58	3	74	2
Oklahoma	4		129	608	5	48	1
Texas	5	1		4,270	16	397	8
Mountain	1		854	1,643	4	90	7
Montana			40	38			
Idaho				190	1		
Wyoming			1	32		5	
Colorado			43	467	1	25	6
New Mexico				40	1	19	
Arizona	1		770	784	1	41	1
Utah				81			
Nevada				11			
Pacific	2		239	4,626	12	85	10
Washington			57	659	3		
Oregon			91	815	2	29	1
California	2		91	3,152	7	56	9
Alaska			173		1		
Hawaii			3	8			

¹ New York City only.
Anthrax: Massachusetts, 1 case.

Reported Cases of Selected Communicable Diseases: United States, Week Ended Apr. 14, 1951—Continued

[Numbers under diseases are International List numbers, 1948 revision]

Area	Rocky Mountain spotted fever (104)	Scarlet fever (050)	Small-pox (084)	Tulare-mia (059)	Typhoid and para-typhoid fever [1] (040, 041)	Whoop-ing cough (056)	Rabies in animals
United States		**2,140**		**13**	**39**	**1,454**	**163**
New England		**188**				**99**	
Maine		14				18	
New Hampshire		[2] 7				9	
Vermont		7				9	
Massachusetts		129				42	
Rhode Island		3				12	
Connecticut		28				9	
Middle Atlantic		**392**			**7**	**213**	**14**
New York		[2] 222			4	88	10
New Jersey		42				72	
Pennsylvania		128			. 3	53	4
East North Central		**733**			**2**	**184**	**38**
Ohio		236			1	46	8
Indiana		68				19	14
Illinois		62			1	28	4
Michigan		312				59	11
Wisconsin		55				32	1
West North Central		**119**				**64**	**14**
Minnesota		24				2	3
Iowa		13				4	6
Missouri		48				12	3
North Dakota						2	
South Dakota		4				4	
Nebraska		4				2	2
Kansas		26				38	
South Atlantic		**168**		**4**	**4**	**316**	**20**
Delaware						1	
Maryland		30			3	4	
District of Columbia		10				6	
Virginia		16				71	7
West Virginia		24				30	1
North Carolina		62				56	
South Carolina		5				17	4
Georgia		4		4	1	103	8
Florida		[2] 17				28	
East South Central		**85**		**3**	**6**	**49**	**32**
Kentucky		35			1	7	12
Tennessee		35		3	1	8	10
Alabama		9			4	27	9
Mississippi		6				7	1
West South Central		**48**		**5**	**9**	**377**	**38**
Arkansas		3			1	48	1
Louisiana		4			3	7	
Oklahoma		11		1		22	
Texas		30		4	5	300	37
Mountain		**132**		**1**	**7**	**101**	**3**
Montana		10		1		7	3
Idaho		38			2	5	
Wyoming						11	
Colorado		13			2	17	
New Mexico		1			1	7	
Arizona		3			2	51	
Utah		[2] 67				3	
Nevada							
Pacific		**275**			**4**	**51**	**4**
Washington		62				5	1
Oregon		23				9	
California		[2] 190			4	37	3
Alaska		1					
Hawaii							

[1] Including cases reported as salmonellosis.
[2] Including cases reported as streptococcal sore throat.

FOREIGN REPORTS

AUSTRALIA

Encephalitis. An outbreak of encephalitis, now called "Murray Valley encephalitis," has occurred in Murray Valley, in northwest Victoria. Prior to February 28, a total of 39 cases with 10 deaths had been observed. A virus, as yet unidentified, has been recovered by Dr. F. M. Burnet, who states that it is unlike the poliomyelitis or myxomatosis viruses. The latter is found in rabbits in that area. Dr. Burnet states that Murray Valley encephalitis appears to have an animal reservoir.

CANADA

Reported Cases of Certain Diseases—Week Ended March 31, 1951

Disease	Total	New-found-land	Prince Ed-ward Island	Nova Scotia	New Bruns-wick	Que-bec	On-tario	Mani-toba	Sas-katch-ewan	Al-berta	Brit-ish Co-lum-bia
Brucellosis	2					1		1			
Chickenpox	689	2		17		154	339	22	13	55	87
Diphtheria	4					3	1				
Dysentery, bacillary.	7					4					3
German measles	358			102		38	120		4	34	60
Influenza	1,180			81	22		140	81			856
Measles	1,050	3		113	2	163	560	118	7	45	39
Meningitis, meningococcal.	6			1			4				1
Mumps	759			10	1	186	239	33	63	103	124
Scarlet fever	241			1		57	36	26	17	69	35
Tuberculosis (all forms).	187	1		3	9	106	38	11	6	13	
Typhoid and para-typhoid fever.	7				3	1	1				2
Venereal diseases:											
Gonorrhea	239	2		4	7	79	36	17	16	33	45
Syphilis	112	3		7	1	60	15	5	8	3	10
Primary	6					2	1		1	1	1
Secondary	10					4	4	1	1		
Other	96	3		7	1	54	10	4	6	2	9
Whooping cough	132	6				36	51	5	6	20	8

FINLAND

Reported Cases of Certain Diseases—February 1951

Disease	Cases	Disease	Cases
Diphtheria	47	Typhoid fever	7
Meningitis, meningococcal	9	Venereal diseases:	
Paratyphoid fever	31	Gonorrhea	366
Poliomyelitis	8	Syphilis	42
Scarlet fever	2,191		

Reported Cases of Certain Diseases—January 1951

Disease	Cases	Disease	Cases
Brucellosis	1	Pneumonia (all forms)	4,411
Diphtheria	17	Poliomyelitis	26
Dysentery, unspecified	1	Rheumatic fever	88
Erysipelas	306	Scabies	997
Gastroenteritis	2,099	Scarlet fever	90
Hepatitis, infectious	48	Tuberculosis (all forms)	287
Impetigo contagiosa	1,506	Venereal diseases:	
Influenza	10,831	Gonorrhea	179
Malaria	2	Syphilis	60
Measles	2,518	Other forms	2
Meningitis, meningococcal	12	Weil's disease	7
Mumps	60	Whooping cough	1,574

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

The following reports include only items of unusual incidence or of special interest and the occurrence of these diseases, except yellow fever, in localities which had not recently reported cases. All reports of yellow fever are published currently. A table showing the accumulated figures for these diseases for the year to date is published in the PUBLIC HEALTH REPORTS for the last Friday in each month.

Cholera

India (French). During the week ended March 24, 1951, 28 cases of cholera were reported in Pondicherry as compared with 11 the previous week.

Smallpox

Burma. For the week ended April 7, 1951, 14 cases of smallpox were reported in Moulmein and 6 cases were reported in Akyab.

India. During the week ended April 7, 1951, smallpox was reported in ports of India as follows: Calcutta, 498 cases; Madras, 133; Bombay, 49; and Masulipatnam, 14. For the week ended March 31, 181 cases were reported in the airport of Nagpur and 12 cases were reported in Ahmedabad.

India (French). During the week ended March 24, 1951, 191 cases of smallpox were reported in Pondicherry as compared with 293 for the previous week. For the week ended March 24, two cases were reported in Karikal.

Indochina. During the week ended April 7, 1951, 47 cases of smallpox were reported in Haiphong, Viet Nam, and 23 cases were reported in Hanoi. For the previous week these ports reported 29 and 15 cases, respectively.

Typhus Fever

Iran. For the week ended March 31, 1951, typhus fever was reported in airports of Iran as follows: Zabol, 28 cases; Teheran, 2; and Zandjan, 2.

Transjordan. For the week ended March 31, 1951, 21 cases of typhus fever were reported in Transjordan as compared with 7 the previous week.

Brazil. In connection with the recent outbreak of jungle yellow fever in Brazil one death each has been confirmed in Goias State on the following dates: December 23, 1950, January 5 and 14, February 1 and 9, and March 18, 1951. In Mato Grosso State one death was confirmed on January 18, 1951.

Ecuador. Eighteen cases of jungle fever were reported in Santo Domingo de los Colorados, which is about 80 miles west of Quito.

Nigeria. During the week ended March 23, 1951, one suspected case of yellow fever was reported in Sabon. This is a new focus for the disease. The patient died in a hospital in Ibadan on March 23.