The Occurrence of Influenza In the United States, 1952–53

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UNDER THE SPONSORSHIP of the World Health Organization, a worldwide effort to improve the reporting and epidemiological study of influenza and the exchange of virus strains has been in effect for the last 6 years.

In the United States the WHO Influenza Study Program has been ably supported by investigators and diagnostic laboratories of State and Federal agencies, universities, the Department of Defense, and those associated with the Commission on Influenza of the Armed Forces Epidemiological Board. A previous report described the organization and specific objectives of the program and listed the participating laboratories (*1*). The present report will summarize the information obtained by these investigators and the mortality experience as reported by State and city health officers for the period from July 1, 1952, to June 30, 1953.

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Investigations of recent years have shown that there has been a progressive change in the antigenic pattern of strains of influenza virus, and attempts have been made to indicate this change in the designations of types, such as the widely used term influenza A′. These designations have been convenient but not completely satisfactory. The WHO Expert Committee on Influenza (*2*) recommends:

> the subdivision of the influenza A virus into the following main groups, which are named after their prototype viruses and the date of their isolation:
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> WS (1933)
> PR8 (1934)
> FM1 (1947)
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> The recent A strains, of which FW/1/50 and A/England/1/51 are examples, though different from FM1, are not as divergent as the other main groups and are therefore considered to fall within the FM1 (1947) group. There is also a heterogeneous group of swine influenza viruses related antigenically to the human influenza A viruses.
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> The influenza B viruses should be subdivided into groups with the general characteristics of:
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> Lee (1940)
> Bon (1943)
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> The most recent influenza B strains (1952) appear to diverge from Bon, and it may be found on the basis of future experience that further groups must be created.
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> In influenza C only one antigenic group is so far known, of which 1233 (1947) is the prototype.

Thus, the strains recovered during the 1952–53 season and referred to as A′ because of their similarity to the FW/1/50 of FM1 strains,

which were used most widely for diagnosis, will be designated as belonging to the group FM1 (1947) in this report. Final studies may justify the designation of the 1953 strains as a separate group, but the recommendations of the WHO expert committee will be followed in the present report. Since various influenza strains were used as antigens for the hemagglutination inhibition and complement fixation tests by the different collaborating laboratories, results of serologic diagnoses will be recorded in tables 1 and 2 as either type A or type B with no attempt to differentiate further between strains.

Occurrence of the Disease

No outbreaks of influenza were reported within the United States during the summer and fall of 1952. There were, however, a few cases diagnosed serologically as influenza B and a few cases diagnosed as influenza A (FM1 (1947)) during October and November in military installations in the United States. A small outbreak of influenza-like disease occurred during the latter part of November at a military installation in the Philippine Islands, and several of the cases were subsequently specifically diagnosed as influenza A (FM1 (1947)).

In early December 1952, a definite increase in the incidence of acute respiratory disease occurred among military personnel at Fort Leonard Wood, Mo. The number of cases increased sharply and had reached a relatively high incidence by the last week of December. At this time an influenza A virus closely related to A/FW/1/50 was recovered from a significant number of cases, and serologic studies gave further evidence of influenza infection. However, not all cases studied were established as influenza, and the possibility of the concurrent presence of another disease was studied by military investigators.

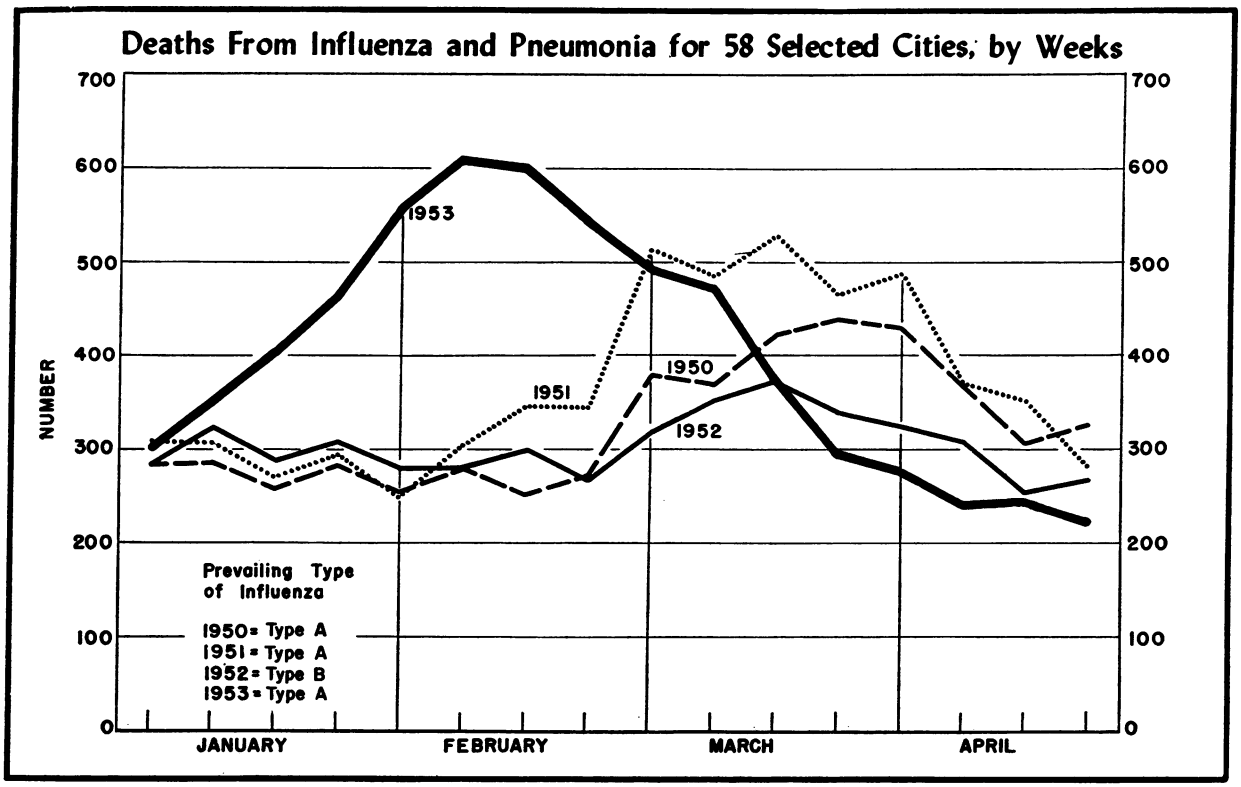
During the same week in December, State health officials reported an influenza-like disease in Pueblo, Colo., and in nearby cities. This also was subsequently confirmed by laboratory tests as influenza A (FM1 (1947)). In Watauga County, N. C., a sharp epidemic with a high incidence rate was reported during the same week and shown to be influenza of the same type. Reports from the military services indicated an increased number of cases of influenza in the Far East in late November and December, principally in Okinawa and Japan.

January 1953 Epidemic

During the first week of January 1953, influenza occurred in epidemic form in Iowa, Indiana, South Dakota, Oklahoma, Missouri, and Florida. By the second and third weeks of the month the outbreaks were reported from most of the middle western and southern States. Texas was particularly affected, and a large number of cases was reported. Subsequent reports indicate that influenza virus A (FM1 (1947)) was frequently recovered. The southeastern part of the United States also was affected, and the disease was specifically diagnosed in the Washington, D. C., area, Norfolk, Va., and other parts of Virginia. The northeast and New England area had a few localized outbreaks particularly noticeable in military installations, but the incidence generally was not high. On the west coast, the incidence was slightly increased but did not approach that of the midwest. Sharp outbreaks in the military and civilian population in Alaska began the first week of January and persisted throughout the month. Toward the end of January the incidence of disease in affected military installations fell off although in civilian populations incidence remained high, and new areas within the midwest and south reported outbreaks. In many of these areas schools were closed because of the illness among pupils and teachers. By the end of the month, the incidence was declining in most areas.

In the early part of February 1953, the incidence continued to decline, and during the remainder of February and early in March, a few scattered outbreaks of influenza occurred, particularly in schools and institutions, not only in the midwest and the south but also in the northeast and the west. No outbreaks have been reported in the United States since that time.

The total number of isolations of influenza virus and positive serologic tests reported from all participating laboratories in the continental United States and the Territory of Alaska by date of onset or collection for each month are presented in table 1. In addition, 16 strains of



influenza A were recovered and 355 positive serologic tests for influenza A were made for which the month of onset or collection date was not exactly known although presumably it was during the period of highest incidence. Not shown in the table is a record of a case of influenza C diagnosed by the complement fixation test which occurred in Chicago during December 1952.

These data indicate that influenza A (FM1 (1947)) was almost exclusively the cause of the epidemic and that only a few sporadic influenza B infections occurred. That January was the month of highest incidence of specifically diagnosed cases conforms to the reports of the occurrence of outbreak received from health officers.

Dr. Clayton G. Loosli of the collaborating laboratory at the University of Chicago has submitted the following observations on the clinical features of the disease:

Although generally reported as mild, influenza infections produced in some cases severe prostration. They were sudden in onset beginning with high fever, marked headache, malaise, muscle aches and pains, and dry cough. Some had photophobia. A few having etiological or serologic evidence of influenza also had symptoms of the common cold, sinusitis or a pneumonitis. Three cases with typical findings of pneumococcal pneumonia due to pneumococcus types I, II or III also had etiological or serologic evidence of influenza. The average hospital stay of uncomplicated influenza was approximately 5 days.

Mortality From Influenza

As shown in the accompanying chart, the number of deaths from influenza and pneumonia in 58 selected cities, which are located in all parts of the United States, began to increase early in January 1953. These diagnoses are based on records of death certificates and do not represent laboratory confirmed cases of influenza. The peak was reached in early February; after that time the number gradually declined. In the 3 years immediately preceding 1953, an increase in deaths from influenza and pneumonia did not begin until late in February, and in none of these years was the number reported as great as it was in 1953.

Data from 8 of the 58 cities were obtained on

Table 1. Isolations of influenza virus and positive serologic tests (any technique) reported by civilian and military laboratories participating in the United States and Alaska

Month and year	Isolation of virus		Positive serologic tests	
	A	B	A	B
December 1952	10	------	29	4
January 1953	237	1	1, 148	22
February 1953	105	------	1, 059	9
March 1953	27	------	377	1
April 1953	------	------	69	3
May 1953	------	------	21	------
Total	379	1	2, 703	39

numbers of deaths from influenza and pneumonia during January and February 1953 for broad age groups. The deaths from influenza and pneumonia combined showed the following distribution: 22.7 percent were in persons under 15 years; 1.3 percent were in the 15- to 24-year group; 28.4 percent in the 25- to 64-year group; and 47.5 percent were 65 years and over. The percentage distribution of influenza deaths was 7.7, 2.3, 20.9, and 69.0, respectively, for the above age groups. These data indicate that persons in the older age groups felt the impact of the influenza epidemic more than younger persons.

Table 2. Estimated death rates per 100,000 population for the United States from all causes, and from influenza, pneumonia, and major cardiovascular-renal diseases, for certain months of 1951, 1952, and 1953

Month and year	All causes	Influenza	Pneumonia	Cardiovascular-renal diseases
December 1952	1, 014. 2	3. 4	31. 4	547. 5
January 1953	1, 083. 1	17. 1	48. 4	590. 7
February 1953	1, 131. 6	26. 4	49. 5	610. 7
March 1953	1, 021. 2	13. 6	34. 0	561. 7
April 1953	955. 7	5. 0	26. 0	524. 2
December 1951	1, 027. 2	3. 1	31. 0	551. 7
January 1952	1, 003. 5	4. 6	36. 6	544. 3
February 1952	1, 015. 6	8. 4	34. 7	538. 0
March 1952	1, 059. 5	12. 3	41. 0	568. 4
April 1952	982. 4	5. 4	31. 5	530. 5

The figures in table 2 have been taken from the *Monthly Vital Statistics Reports* of the National Office of Vital Statistics, Public Health Service. They show the estimated death rates for the United States from all causes; and from influenza, pneumonia, and major cardiovascular-renal diseases, based on a 10-percent sample; and they cover the periods when influenza was prevalent in 1952 and 1953. Although these data are subject to random sampling errors, they show that the number of deaths from all causes and from pneumonia and cardiovascular-renal diseases increased moderately and that those from influenza increased markedly during the period when influenza was known to be prevalent. The increases were greater in 1953 than in 1952; this is consistent with the data for 58 cities shown on page 1143.

Antigenic Analysis of 1953 Strains

The WHO Strain Study Center for the Americas, located in the laboratory of Dr. T. P. Magill, State University Medical Center at New York, Brooklyn, N. Y., has studied the antigenic characteristics of 24 strains of influenza virus isolated late in 1952 and in 1953 by various workers. Hemagglutination inhibition tests were performed with these strains and cholera-filtrate treated rabbit antiserums which were prepared against 8 chronologically separated strains:

A/PR8/34.
A/New York/41 (Coyle).
A/FM1/47.
A/Nederland/1/49.
A/FW/1/50.
A/England/1/51.
A/New York/1/53.
A/Ohio/1/53.

These tests showed clearly that the recently isolated strains were influenza A virus but were different antigenically from previously isolated ones. The 1952–53 strains possessed some antigenic components which were undetectable or inconspicuous in the older strains. They differed in practically the same degree from A/FM1/47, the prototype of the A′ strains, as the latter differs from the PR8 strain in 1934. This adds further evidence to the concept of an apparently orderly alteration in the antigenic

The Virus, the Cell, and the Potentialities of Influenza

"I have spoken throughout as if influenza virus were no more than a laboratory tool, a subtle probe with which to explore the finer structure and functioning of the living cell. It would not be fitting, however, to end this lecture without reminding you that influenza virus is also an important agent of disease. It is a virus which even in 1951 killed heavily amongst the old people of Europe and in 1918–19 generated the third great plague of recorded history. Of all virus diseases influenza is probably that in which mutational changes in the virus are of greatest human importance. We can only guess what type of virus was responsible in 1918–19 and what changes took place during the course of the pandemic. But even in the period since the human virus was first isolated in 1933 there have been striking changes in the immunological character of both influenza A and B viruses. Some of us believe that the influenza virus' chief means of survival is its capacity for constant mutation to new serological patterns, and those of us who have had anything to do with the production of influenza vaccines know very well how that capacity can nullify the most painstaking work. Infectious disease today has lost most of its terrors, and in America at least the peaks of mortality that always marks the passage of an influenza epidemic are becoming smaller. But no one yet can say whether or when we shall see another pandemic outbreak of influenza. Until we know the answer to that question we should not be too complacent about our powers to deal with acute infectious disease. Even the most academic-seeming of investigations, like those I have described, may one day become matters of life and death."

—Sir F. MACFARLANE BURNET, F. R. S.,
in the Second R. E. Dyer Lecture, 1952,
Public Health Service Publication No. 328.

composition of influenza viruses by the appearance or dominance of new components and the suppression or disappearance of others as determined by this method of analysis.

Influenza Outside the United States

From outbreaks of influenza in South Africa during June 1952 and on Bahrein Island during September, strains of influenza A (FM1 (1947)) were isolated and identified by the laboratories cooperating in the World Health Organization study. Influenza was next reported in the Far East, Japan, and the Philippine Islands in early December 1952. Approximately coincident with highest occurrence of the disease in the United States during January 1953, outbreaks appeared in France, Germany, and southern England, later extending to the Scandinavian countries, Switzerland, and Austria, and to scattered parts of Europe. The isolated strains were all influenza A (FM1 (1947)), similar to those isolated in 1951 and to those recovered in the United States during January 1953.

Later in 1953, during May, June, and July, influenza appeared in the Central American and South American countries in large outbreaks, but virus strains have not yet been completely studied. Mortality reports indicate that the death rates in European and South American countries were similar to those in the United States with some increase in the death rate from influenza and pneumonia, but the increase was largely confined to the older age groups of the population.

Comments and Conclusions

The broad picture presented here shows that influenza A was widespread throughout the Americas and Europe during the 1953 season, although some large areas were spared. It is to be remembered that during the previous season in 1952, influenza B was locally prevalent although not of such high incidence in North

America and Europe, and influenza A was practically absent. During the winter 1950–51, influenza A caused serious epidemics in northern England, particularly in Liverpool, and in northern Europe during December and January, and it was also occurring in the northeastern part of the United States. In the 1953 experience, the highest incidence in the United States was in the midwest and the south. The northeastern part which had been affected 2 years previously was largely spared with the exception of some military installations.

The mortality data here presented indicate that a significant number of deaths resulted from influenza and that this disease is still an important cause of deaths. This is true in spite of the availability of antibiotics and their presumed widespread use in the treatment of severe respiratory infections. Perhaps too complete confidence in the ability of the antibiotics to reduce deaths from pneumonia, and thus alleviate the chief danger from influenza, may not be justified.

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Current lists of laboratories and observers participating in the Influenza Study Program in the United States may be obtained from the Influenza Information Center, National Institutes of Health, Public Health Service, Bethesda, Md.

REFERENCES

(*1*) Davis, D. J.: World Health Organization influenza study program in the United States. Pub. Health Rep. 67: 1185–1189 (1952).

(*2*) World Health Organization Expert Committee on Influenza: First Report. WHO Techn. Rep. Ser. 64. Geneva, April 1953. 32 pp.

Labeling Salt in Food

Two actions to protect persons suffering from some types of heart disease, or from high blood pressure, who are on "low-sodium" or "low-salt" diets have been taken by the Food and Drug Administration of the Department of Health, Education, and Welfare.

The aim of these actions is to improve the labeling of special dietary foods, and of certain frozen vegetables commonly used in low-salt diets, so that patients and physicians will be better able to calculate the sodium intake from such foods. In one action, the Department published a statement of policy in the Federal Register, to the effect that it will henceforth expect all labels of frozen vegetables to declare the presence of salt whether added directly or indirectly to these products.

FDA explained that frozen vegetables are quite commonly supposed to be salt-free, and on that account are largely used in low-salt diets. Actually, salt brine is used in the preparation of certain of these vegetables, particularly frozen peas and frozen lima beans, as a means of quality separation (the younger and more tender peas or beans will float in the brine). This process may add a substantial amount of salt to the frozen product, which would be of significance to persons seeking to restrict their salt intake.

In the second action, the Secretary of the Department of Health, Education, and Welfare gave notice in the Federal Register that public hearings will be started on December 15, 1953, in Washington, to amend the FDA dietary food labeling regulations so as to require label declaration of sodium in low-sodium foods on the basis of their sodium content in milligrams of sodium per 100 gm. (roughly one small serving) of the food. FDA said that the declaration of sodium content on this basis conforms with the recommendation of the American Heart Association and the Council on Foods and Nutrition of the American Medical Association.