1970-1971

REPORT NO. 87
DECEMBER 1971

CENTER FOR DISEASE CONTROL

INFLUENZA - RESPIRATORY DISEASE SURVEILLANCE

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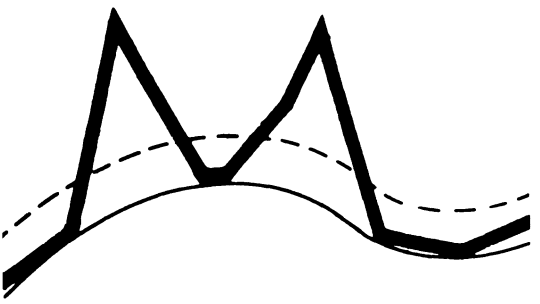

U. S. DEPARTMENT OF
HEALTH, EDUCATION, AND WELFARE
PUBLIC HEALTH SERVICE

PREFACE

Summarized in this report is information received from State Health Departments and other pertinent sources, domestic and foreign. Some of the information is preliminary. It is intended primarily for the use of those with responsibility for disease control activities. Anyone desiring to quote this report should contact the original investigator for confirmation and interpretation.

Contributions to the surveillance report are most welcome. Please address to:

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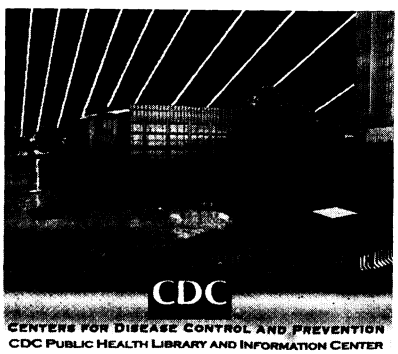

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DHEW Publication No. (HSM) 72-8084

INFLUENZA SURVEILLANCE 1970-71

I. SUMMARY

From March 1970 through July 1971, influenza activity in the United States decreased markedly from the epidemic levels that were present the previous 3 years. However, widespread outbreaks of influenza occurred in a number of states. In a majority of these outbreaks, virus similar to influenza B/Mass/66 was demonstrated to be the responsible agent. Influenza B was also prevalent in Western Europe, while influenza A was more frequently seen in the Southern Hemisphere.

II. METHODS

Basic information on the prevalence of epidemic influenza in the United States was obtained from the 50 states, the District of Columbia, and Puerto Rico.

A. Mortality

Although total reported mortality usually increases in years in which influenza is epidemic, pneumonia and influenza mortality rises more sharply. At the Center for Disease Control, reported pneumonia-influenza deaths for 122 cities are compared weekly with expected values, and excess deaths are used as an index of influenza activity. Increases above the epidemic threshold for 3 successive weeks was found to correlate well with influenza epidemics. Peak deaths from pneumonia and influenza usually followed the peak occurrence of clinical disease by an interval of 3 to 4 weeks. Therefore, these data cannot be used to anticipate epidemics nor is it sensitive to small outbreaks, particularly those of influenza B. Rather the concept of excess mortality is of value for the post-facto assessment of the extent and severity of an influenza epidemic.

The 122 cities that report morbidity and mortality data are major population centers of at least 100,000 inhabitants. They include approximately 70 million people, or roughly one-third of the nation's population. Although statistically valid conclusions concerning excess mortality cannot be generally projected from the 122 cities to the national total population, the data for the 122 cities are roughly comparable from year to year.

B. Morbidity

Weekly or monthly influenza case counts are sent to the CDC by state health departments of 30 states. A variety of reporting procedures are used by these states, and the data obtained are not comparable. However, for any given state, comparison of its data with prior years allows an estimate of current influenza activity.

C. Telephone Surveys

The Respiratory Disease Unit, Viral Diseases Branch, CDC, conducts periodic telephone surveys before, during, and after the peak influenza season to obtain

specific information about occurrence, distribution, severity, and documentation of influenza activity.

D. Supplemental Information

State laboratory reports, epidemic investigations, and direct contacts with state health officials provide additional information.

E. World Health Organization

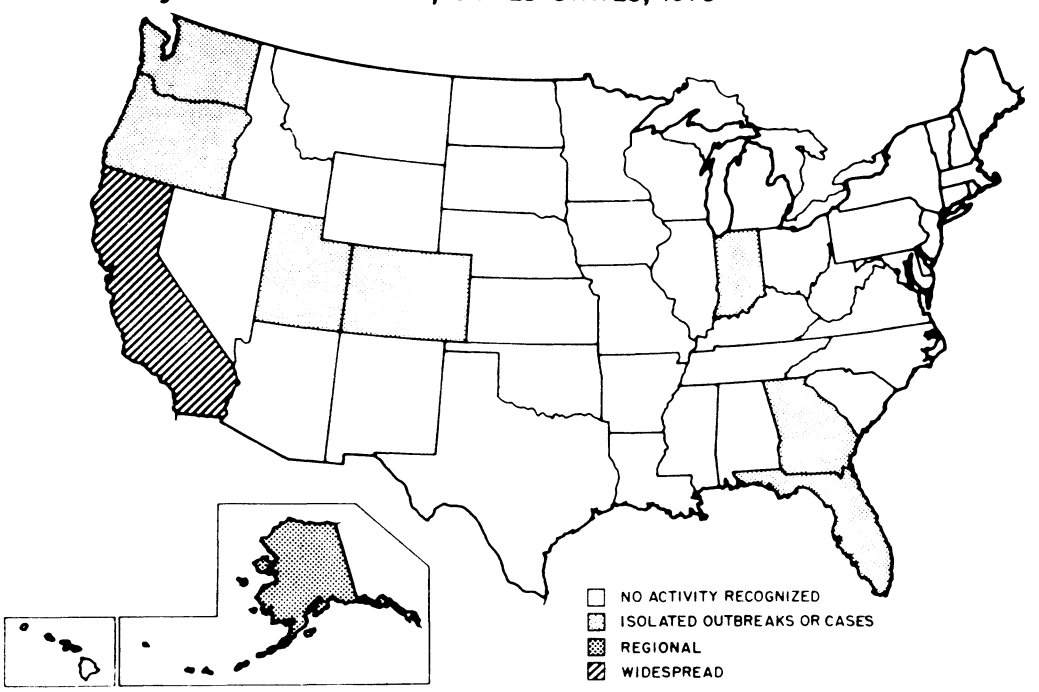
International influenza activity is monitored by the CDC utilizing data obtained from the World Health Organization, the Pan American Health Organization, and national reporting facilities.

III. RESULTS

A. General Data

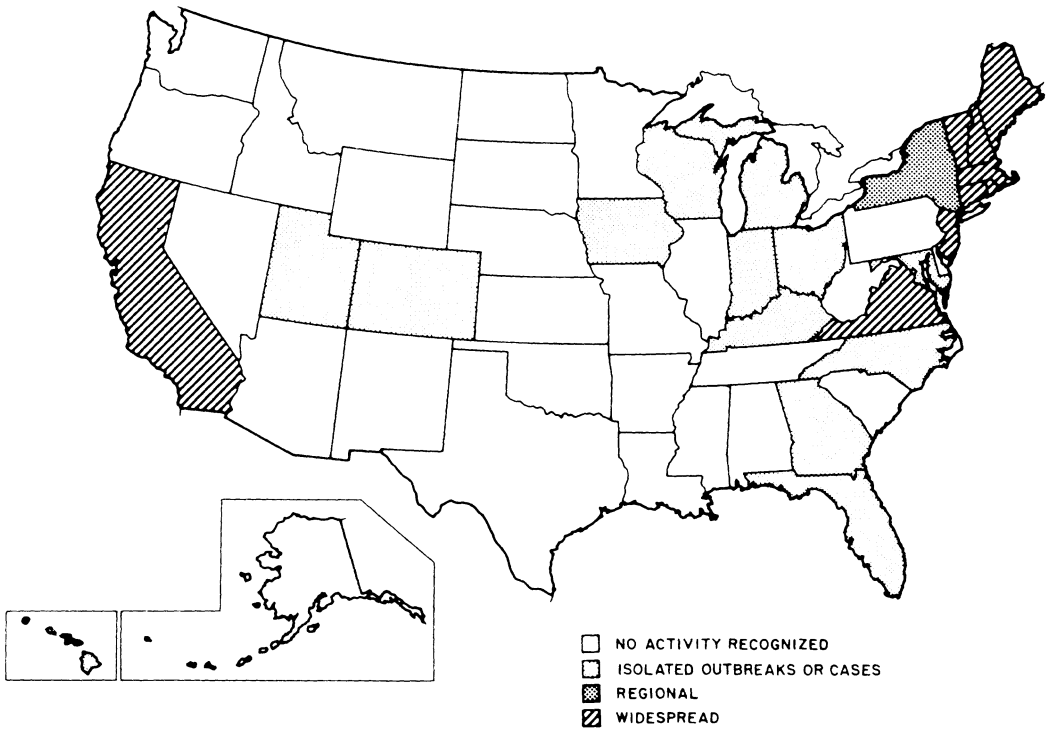
Twelve states reported regional* or widespread* influenza activity in 1970-71, compared with 18 in 1969-70. (Figure 1 and 2) The earliest activity was reported in August 1970 from Puerto Rico. National case reporting was maximum in early February. The disease was localized, and urban areas appeared to have been spared. In contrast to the last 2-3 years, influenza B was responsible for most of the activity.

Figure 1 **INFLUENZA A, UNITED STATES, 1970**



*The CDC classified extent of influenza in 4 categories: 1) Isolated cases, 2) Isolated outbreaks, 3) Regional involvement (outbreaks recognized in contiguous counties, but altogether involving counties comprising less than 1/2 of the state's population), and 4) Widespread involvement (more than 1/2 of the counties or more than 1/2 of the population).

Figure 2 INFLUENZA B, UNITED STATES, 1970



From March 1970 through July 1971, no sustained increase was noted in pneumonia-influenza mortality in 122 United States cities (Figure 3). In the first 2 weeks in January the slight elevation above the epidemic threshold in "Mortality from all Causes" represents a reporting artifact which is seen annually, attributable to end of year reporting. Although there was no general increase in pneumonia-influenza deaths throughout the United States, slight increases above the epidemic threshold level occurred in New England during the first 3 weeks of February 1971, in the Middle Atlantic region in early February, the South Atlantic region in late February, and the Pacific region in March and April. The increased mortality in the first three regions was associated with isolated outbreaks of influenza A_2. Outbreaks of influenza-like illness in other regions were not accompanied by sustained increases in pneumonia-influenza deaths above the epidemic threshold. (Figure 4)

Figure 3

PNEUMONIA-INFLUENZA DEATHS IN 122 UNITED STATES CITIES

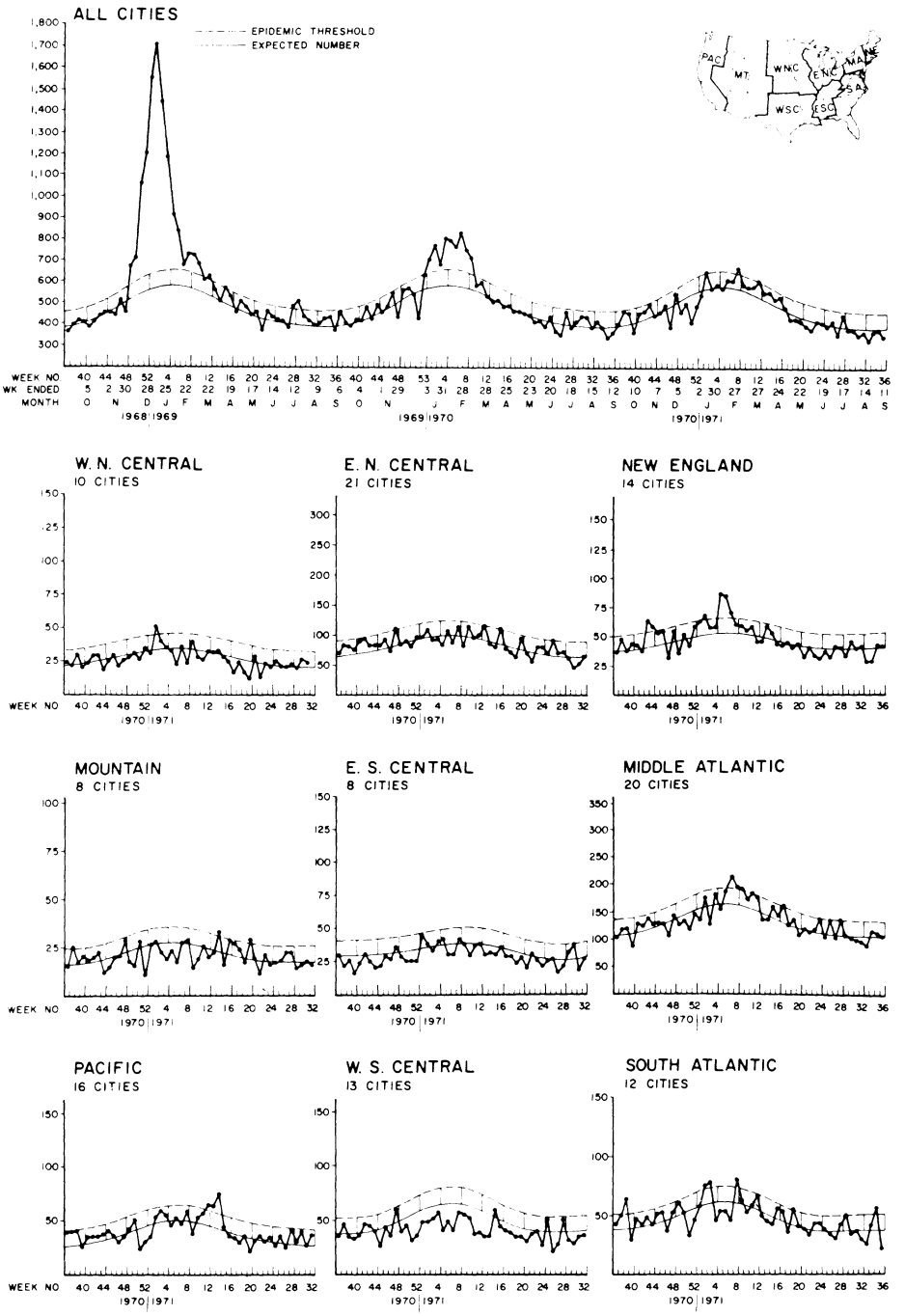
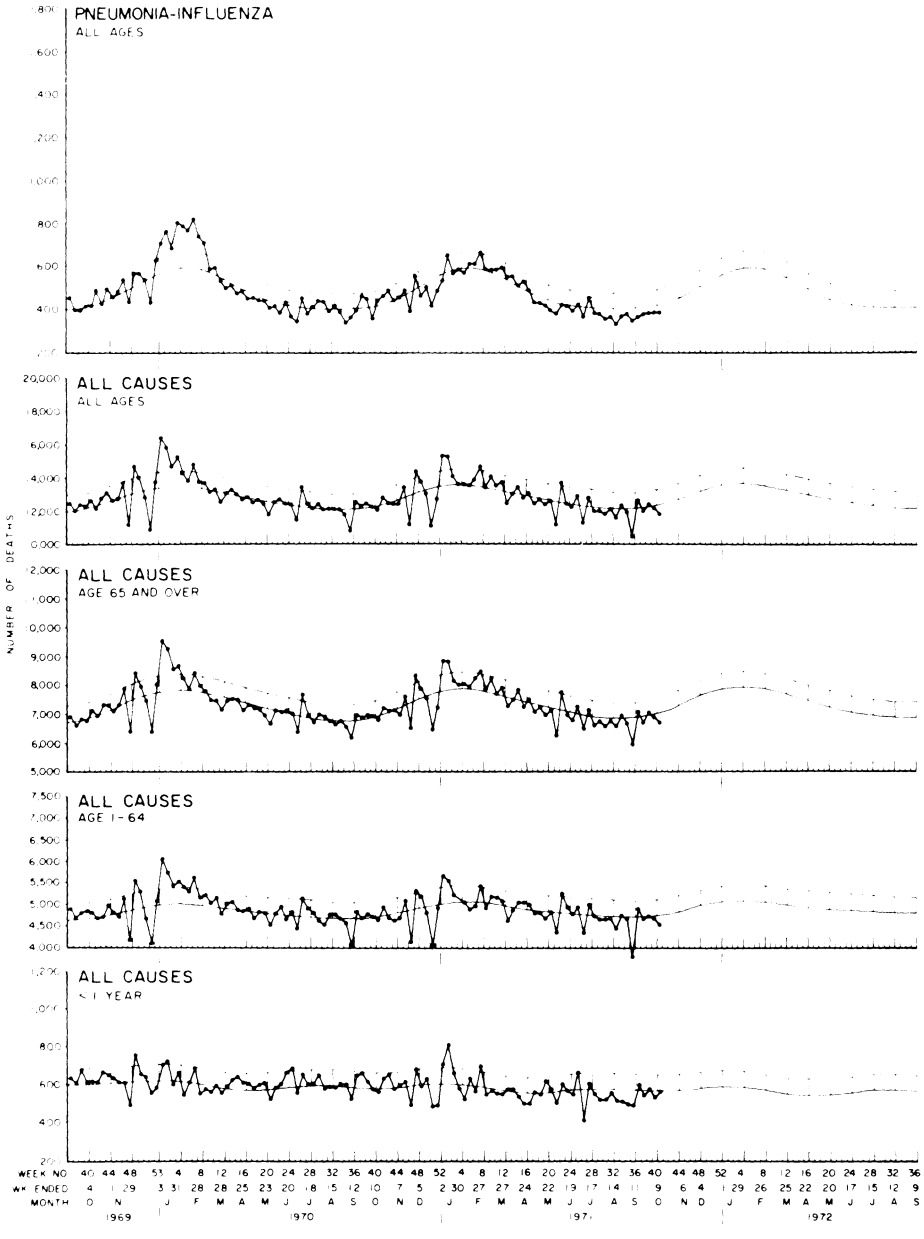


Figure 4

MORTALITY IN 122 UNITED STATES CITIES



B. State Reports

1. Puerto Rico

Puerto Rico was the first reporting area to confirm A_2/Hong Kong influenza from specimens taken on September 30, 1970. Case reporting began in early August with the opening of school and reached its peak in September; an estimated 8,000 people were involved.

In 1970-71, cases occurred in western parts of the island which are relatively sparsely populated; 12.7 percent of the population lives on 25-30 percent of the land area. The major towns were not affected. Some of the affected areas had influenza activity in both 1968 and 1969, so that this outbreak did not occur in a completely susceptible area. It is postulated that these areas were not as thoroughly exposed previously as were areas where transportation is better and where living conditions are more crowded.

Influenza epidemics occurred in Puerto Rico in 1967 and 1968. In 1967, A virus was thought responsible; the outbreak began in December, peaked in January, and involved 107,000 people. A_2/Hong Kong caused the epidemic in 1968. The disease appeared in September, peaked in October, and continued until February. In 1969, scattered outbreaks occurred, predominantly in the east and south.

Severity of the disease in 1970-71 was difficult to compare with previous years, but was probably similar. Specific age data is not available, but observers were impressed that children and young adults were significantly affected. (Reported by Dr. Rafael Correa Coronas, Assistant Secretary of Health, and Dr. Paul Blake, EIS officer).

2. New England

Massachusetts experienced a widespread outbreak of influenza that was recognized January 11, 1971, in the eastern part of the state and moved steadily west, reaching a peak in the last half of January in eastern Massachusetts and in the first week of February in the western part of the state. Influenza B was isolated from a number of patients. School age children were predominantly affected; there was no increase in industrial absenteeism. In Connecticut a widespread outbreak of influenza involved primarily school-age children, and absenteeism from schools reached 20-30 percent. Eighteen isolations of influenza B virus were reported and additional sero-conversions were documented. There were no isolations of influenza A_2. The peak incidence occurred in the week ending February 13. In New Hampshire there was also widespread influenza activity, occurring for the most part in the southern part of the state with peak activity in the week ending February 6. Vermont had an increase in influenza-like illness in at least 12 of 14 counties. School absenteeism rose during the first week in February. Rhode Island also reported widespread influenza involving school children, and sero-conversion to influenza B was documented in at least 10 patients. The peak of school absenteeism occurred in the week ending February 5.

3. Middle Atlantic Region

New Jersey reported widespread outbreaks of influenza caused by influenza B virus. School age children were mainly affected with 15-20 percent school absenteeism. Peak activity occurred in the first week of February. New York State reported influenza B activity in its eastern counties,

documented by sero-conversions and isolations. A number of schools were closed. Scattered cases of influenza-like illness occurred in New York City, and sero-conversions to both influenza B and A were documented.

4. Elsewhere in the United States

In Virginia, widespread influenza was reported with the closing of some schools, and 15-20 percent absenteeism in others. Viruses similar to influenza B/Mass/66 were isolated from three patients. In the District of Columbia, isolation of influenza B was frequent in January and February. Pre-school children appeared to be most involved.

Isolated outbreaks of influenza B were also reported from Colorado, Florida, Georgia, Indiana, Kentucky, Maryland, Michigan, New Mexico, North Carolina, Ohio, Utah, and Wisconsin.

Regional outbreaks of influenza A_2/Hong Kong were reported from Alaska, California, and Hawaii. Isolated A_2 activity was noted in Colorado, Florida, Georgia, Indiana, Oregon, Utah, and Washington. California and Hawaii also reported isolations of influenza B and noted marked increases in influenza-like illness in the latter part of February.

Arizona, Kentucky, Montana, New Mexico, Pennsylvania, and Tennessee reported seasonal increases in influenza-like illness without much school or industrial absenteeism and without viral isolation or documented sero-conversions to influenza.

C. Influenza Outside the United States

Influenza was widespread in both the Northern and Southern Hemispheres during their respective winter seasons. Relatively little influenza A was recovered, and only sporadic cases or localized outbreaks were documented. Virus isolation yielded strains which were antigenically similar to A_2/Hong Kong/68. Only A_2/England/878/69 and A_2/Nagasaki/1/70 have showed any significant (fourfold or greater) decrease in reactivity with the A_2/Hong Kong/8/68 antisera.

Influenza B activity throughout Western Europe was reported to the WHO. Significant influenza B isolates accounted for the preponderance of cases, and isolates were all inhibited by B/Massachusetts/3/66 reference antisera. The disease was generally mild, often involved school children, and tended to peak in March or April. In France cases occurred, and a February peak was noted.

Countries in the Southern Hemisphere generally reported influenza A activity in the period April-September 1970.[1] Widespread disease was reported from Argentina, Australia, Fiji, New Guinea, Panama, Papua, Senegal, and Tahiti. Influenza B was isolated in Australia, Ceylon, Fiji, and South Africa.

In the Northern Territory of Australia, severe epidemics associated with virus A_2/Hong Kong/68 occurred in 1968 and 1969. Therefore, this was the third consecutive year that epidemic influenza was associated with this virus in Australia.

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[1]WER 46:353, No. 34

Localized outbreaks of influenza were associated with A_2/Hong Kong/68 in Hong Kong and Malaysia. In Hong Kong, A_2 virus was isolated from mid-March 1971 to June 1971. In Taiwan, A_2/Hong Kong/68 was isolated during a period of slightly increased incidence between March 11 and June 30, 1971.

A_2 viruses isolated throughout the world were similar to the original strain A_2/Hong Kong/1/68 or A_2/Hong Kong/8/68. However, there were isolations in Hong Kong and New Zealand of the variant A_2/England/878/69 which is slightly different antigenically. All strains of virus B were antigenically similar to the variant which was prevalent from 1967 to 1970 (B/Switzerland/265/67, B/Rome/1/67, and B/Massachusetts/3/66).

IV. DISCUSSION

The absence of major influenza A activity in the United States in 1970-71 epidemic season was not entirely unexpected, coming after 3 consecutive years of influenza A. However, isolations of influenza A were made throughout the United States in individual cases; several large isolated outbreaks occurred.

Localized and regional outbreaks of influenza B that occurred in the past year were of relatively small magnitude and were not unexpected. During the period 1965-66, widespread influenza B activity was documented along with influenza A on both the east and west coast. The activity of influenza B in the Atlantic and New England states, coming as it did 5 years after their last documented epidemic, is in keeping with the usual 3-6 year cycle of influenza B.

V. STATE LABORATORY REPORTS

A. New York*

The state laboratories in New York collect monthly sera from eight geographic areas in the state excluding New York City. Ten sera from each district are examined for prevalence of complement fixing antibody against influenza A and B. The sera are available from pre-employment and pre-marital blood tests and are from a population who are mostly in their twenties and thirties. The specimens are selected from a group of 20-100 in each district to give a roughly equal sex distribution and a comparable age grouping; the population is predominantly rural and small town. The results are shown in Table 1.

TABLE 1

NEW YORK DATA

PERCENT INCIDENCE OF CF ANTIBODY AGAINST A & B

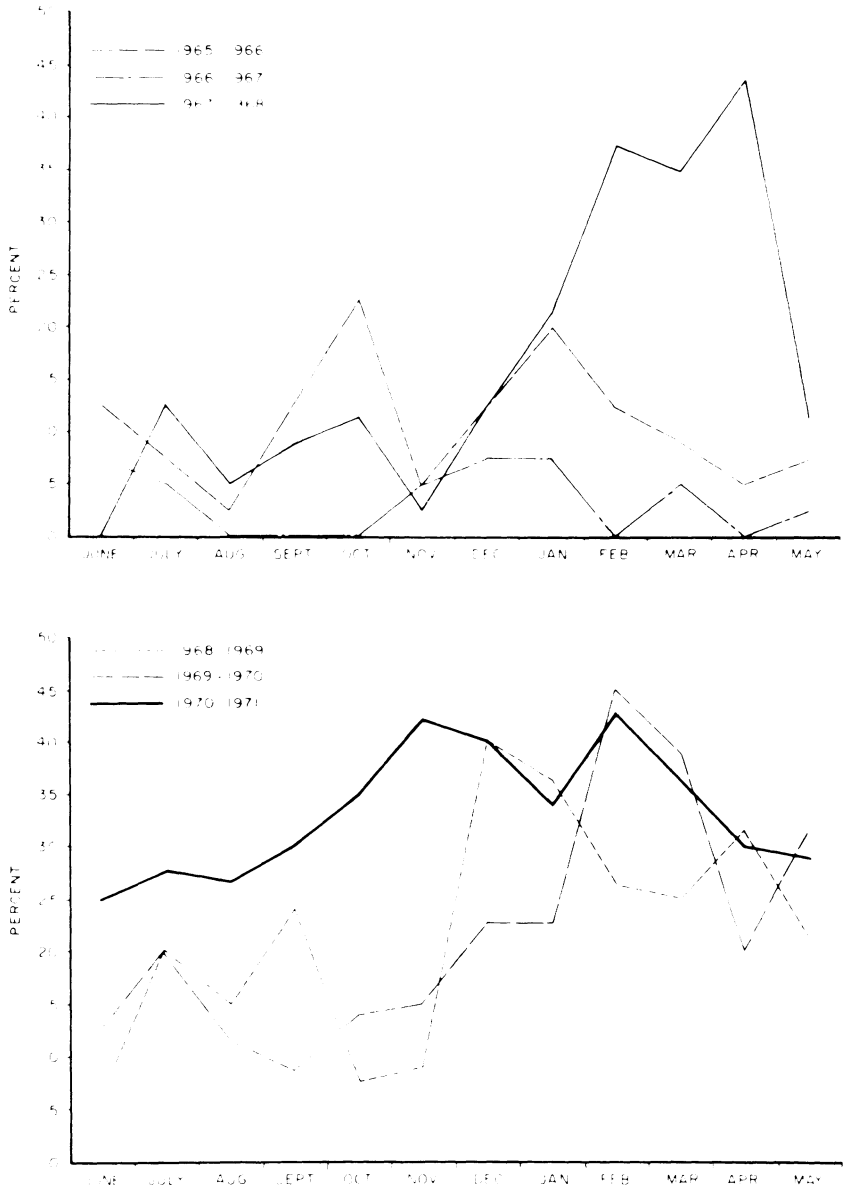
	MAY	JUNE	JULY	AUG.	SEPT.	OCT.	NOV.	DEC.	JAN.	FEB.	MARCH	APR.
A	31	25	28	26	30	35	43	40	34	43	36	30
B	4	1	5	4	3	1	5	10	4	6	13	18

*Reported by Rudolf Deibel, M.D., Director, Virus Laboratories, New York State Department of Health, Albany, New York.

New York State had regional outbreaks of influenza B which peaked in early February. Increased incidence of CF antibody to influenza B appeared in March. In previous years, there has been a lag time of about 4-6 weeks from peak disease activity to increased antibody levels. There was minimal influenza A activity in New York last year, and CF titers to A did not show a significant change throughout the year.

The level of antibody to influenza A in New York State has usually peaked in February and declined in the summer (Figure 5). In 1970, high antibody levels continued into the fall. The persistance of high titer CF antibody to influenza A followed 3 years of widespread influenza A activity and was associated with limited clinical disease. It is not known if this type of data will have prognostic significance.

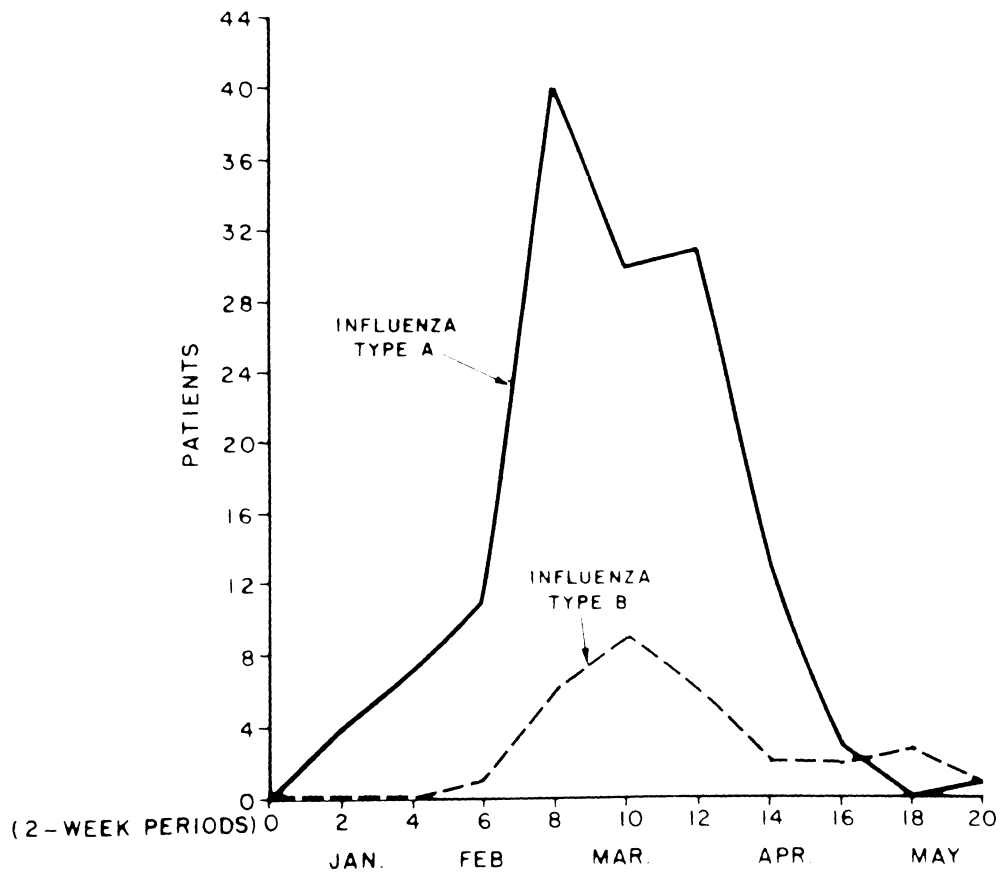
Figure 5 INCIDENCE OF COMPLEMENT FIXING ANTIBODY AGAINST INFLUENZA A IN RESIDENTS OF NEW YORK (EXCLUDING NEW YORK CITY), 1965-1971



B. California*

The State Department of Public Health Laboratory of California maintains an acute respiratory disease surveillance system. Laboratory confirmation of infection was documented in 24 persons by A_2 virus isolation and by B isolation in 3. Figure 6 shows a peak of sero-conversion in early March. More than half of the laboratory-confirmed cases of influenza B occurred in persons 20 years old. Most documented A_2 cases, however, were in persons over 50 years old.

Figure 6 **NUMBER OF PATIENTS WITH 4X OR > INFLUENZA ANTIBODY RISES, BY DATE OF ONSET, VIRAL AND RICKETTSIAL DISEASE LABORATORY, CALIFORNIA, 1971**



*Reported by Edwin H. Lennette, M.D., Chief, Viral-Rickettsial Disease Laboratory, and James Chin, M.D., Bureau of Communicable Disease Control, California State Department of Public Health, Berkeley, California.

VI. SUPPLEMENTARY REPORTS

A. Influenza B - Albuquerque, New Mexico*

In February and March 1971, an outbreak of respiratory illness due to influenza B occurred at the Albuquerque Indian School (AIS), Albuquerque, New Mexico. Students range in age from 9 to 21 years and live in three large dormitories on campus. Approximately 60 percent of the students attend classes on the AIS campus, while the remainder go to public schools in Albuquerque.

The epidemic began the week of February 14 and persisted through the week of March 28. The attack rate for the entire school was 29 percent (161 of 559). Fifty-four percent of the illnesses occurred in the 2-week period between February 28 and March 13.

Most students experienced a relatively mild illness with sore throat (97 percent), cough (86 percent), headache (77 percent), and fever (63 percent). Myalgia was distinctly uncommon, occurring in only 1 percent of the cases. There was one death associated with the outbreak; a 16-year-old girl developed symptoms compatible with influenza and died 8 days later of a superimposed staphylococcal pneumonia.

Influenza virus B was isolated from 16 ill students and one asymptomatic student. Influenza virus A_2/Hong Kong strain, was isolated from three ill students. Forty-five sero-conversions to influenza B and nine to influenza A/Hong Kong were documented.

In a dormitory where students had a clinical attack rate of 43 percent, an effort was made to determine the overall incidence of clinical and sub-clinical infection with influenza B. Paired sera were obtained from as many students as possible regardless of a history of illness; 82 of 100 were sampled. Sero-conversions were noted in 65 percent of those ill (24 of 37) and in 33 percent of those not ill (15 of 45).

Absenteeism rates in Albuquerque public schools were generally low during the epidemic period and did not differ from previous years except among high school students. In the latter group, absenteeism ranged from 8.9 to 11.1 percent in February, March, and April 1971, which was about 3 percent higher than for the same period in 1969 and 1970. Peak absenteeism in the public high schools occurred some 4 weeks after the AIS outbreak had subsided.

B. Influenza A_2 - St. Paul Island, Alaska**

An epidemic of influenza A occurred on St. Paul Island, Alaska, in April 1971. St. Paul is a compact community of 500 persons, primarily Aleuts, on the Pribilof

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*Reported by Elisa Hurtado, M.D., Physician-in-charge, Student Health Clinic, Albuquerque Indian School, Albuquerque, New Mexico; M. Colin Jordan, M.D., EIS officer, Gary R. Nobel, M.D., Chief, Virus Disease Section, and Gary E. Tegtmeier, Ph.D., Chief, Serology Unit, Virus Disease Section, Ecological Investigations Program, Kansas City, Kansas.

**Reported by Jonathon Rodnick, M.D., Medical Officer, St. Paul Island, Indian Health Service; Elmer Feltz, Virology Unit, Arctic Health Research Center, College, Alaska; Respiratory Viral Unit, CDC, Atlanta, Georgia; Alaska Activities, Ecological Investigation Program; and Elizabeth Price, M.D., Chief, Community Health, Alaska Department of Health & Welfare; and an EIS officer.

Islands in the Bering Sea. The only regular contact with the outside is once or twice weekly airplane service to the Alaska mainland.

Febrile respiratory illness typical of influenza first appeared in early April. The peak of school absenteeism occurred in late April. The island physician estimated that 20 percent of the population of all ages was affected. There were two cases of pneumonia, but no deaths occurred.

Twelve isolates of A_2 influenza virus were made between March 12 and May 13, 1971. Two patients from whom virus was isolated in April had greater than fourfold titer rises to A_2 antigen. One influenza A_2 strain was isolated from a patient in Anchorage in April 1971. Since St. Paul Island had no significant influenza activity during the three previous influenza seasons, the population was probably susceptible to A_2 influenza.

Widespread influenza morbidity and multiple isolations of influenza B occurred on the Alaska mainland in March and April 1971.

C. Reye's Syndrome - New England and New York State

In January and February 1971, 36 children in New England and New York State were hospitalized with acute encephalopathy diagnosed as Reye's syndrome, apparently representing a marked increase in the number of cases for the same period in previous years. Twenty-one of the patients were from Massachusetts, five were from Connecticut, three each from Rhode Island and Vermont, two from New York, and one each from Maine and New Hampshire (Figure 3). Cases were distributed over wide areas within the states, and there were only two instances in which two or more of the patients lived in the same town. The ages of the patients ranged from 3 to 16 years, median 11 years (Table 1). Thirteen patients were male and 24 female. The distribution of cases by age and sex were roughly comparable for all states. A total of 21 (58 percent) of the 36 children died; the case fatality rate in the 10-14 age group (72 percent) was higher than in the other age groups.

For 18 of the 36 cases of acute encephalopathy, a diagnosis of liver abnormality was made on the basis of fatty degeneration of the liver at autopsy. On two cases, changes noted in liver biopsy specimens accounted for the diagnosis. The remaining cases were diagnosed on the basis of abnormal serum glutamic oxaloacetic transaminase or serum glutamic pyruvic transaminase values with minimal or no elevation of the total bilirubin.

Specimens for serologic testing and/or virus isolation studies were obtained from 12 patients. A fourfold increase in serologic titers against influenza B was noted for two of the patients. Single hemagglutination-inhibition (HI) titers against influenza B of 1:256 and 1:512 were documented for four others. For another patient, a single HI titer of 1:512 against influenza B was recorded, and this virus was recovered from a throat swab specimen. No other agent has been associated with these cases. Virus isolation studies and serologic tests were negative for the remaining five patients.

Note:

Although other viruses have been associated with Reye's syndrome (most notably varicella), the Viral Diseases Branch is instituting a surveillance program this year for this disease and hopes to make available epidemic aid for cases occurring simultaneously with influenza B. Moreover, we hope to intensify our case finding of Reye's syndrome with the cooperation of the state epidemiologists and interested clinical specialists. Participation in this program is invited.

In addition, the Neurotropic Disease Unit of the Viral Diseases Branch has been concerned with the neurologic sequelae of influenzal disease. Since Leigh's description in 1946 of encephalitis, myelitis, and polyneuritis following influenza B, numerous other reports have appeared. Encephalitis has been described during the acute stage of influenza illness and also after the patient appears to have entered the recovery phase. Polyneuropathy of the Guillain-Landry-Barré type was frequently noted in England in the 1969-70 epidemic and usually appeared in the recovery phase. Transverse myelitis is less frequently described. In our experience, neurologic disorders have been related to epidemic influenza rather than to sporadic cases. We are most interested in obtaining reports of such sequelae of influenza.

VII. LABORATORY REPORT - WHO INTERNATIONAL INFLUENZA CENTER FOR THE AMERICAS

A. Hemagglutination-inhibition Characteristics of Influenza A and B Strains Isolated During 1970-71

From July 1970 to July 1971, very few influenza A isolations were reported from the Western Hemisphere. Those viruses that were recovered from patients in sporadic cases or localized outbreaks continued to resemble the 1968 Hong Kong pandemic strain (Table 2). Only A_2/England/878/69 and A_2/Nagasaki/1/70 showed any significant (fourfold or greater) decrease in activity with either the recombinant virus antisera or the parent A_2/Hong Kong/8/68 antisera.

TABLE 2

Hemagglutination inhibition reactions* of type A2 influenza viruses, 1968-71

Antigens	Antisera†	Aichi$_H$ - Sw$_N$	HK$_H$ - NWS$_N$	A2/HK/8/68	A2/England/878/69	A2/Chile/474/70	A2/Wash/2/70	A2/Mayo Cl/1/71	A2/Miss/1/71
A2/Hong Kong/8/68		890	1780	1280	1780	1280	1280	890	1280
A2/England/878/69		**160****	640	445	1780	**320**	445	**220**	640
A2/Chile/474/70		445	890	640	890	640	890	445	640
A2/Fukuoka/1/70		890	1280	1280	1780	890	1780	890	1280
A2/Ibaragi/15/70		320	640	445	**445**	**320**	640	320	445
A2/Jamaica/728/70		1280	1780	890	1780	1780	2560	1780	1280
A2/Nagasaki/1/70		**160**	**220**	**320**	**320**	**320**	445	**160**	**320**
A2/Osaka/1/70		320	640	445	890	**320**	890	**160**	**320**
A2/Puerto Rico/54/70		640	1280	640	1280	640	1280	445	640
A2/Rio/1/70		640	1280	1280	1280	640	1280	640	890
A2/Taiwan/888/70		640	1280	1280	1280	1280	1280	1280	1280
A2/Washington/2/70		320	1280	640	1280	640	1280	640	640
A2/Georgia/7/71		640	1280	1280	1280	1280	1280	1280	1280
A2/Hawaii/2/71		640	1280	1280	1280	890	1280	1280	1280
A2/Iowa/2/71		640	1280	1280	1280	890	1280	1280	1280
A2/Mayo Clinic/1/71		445	1780	890	890	890	640	1280	1280
A2/Mississippi/1/71		445	640	445	890	890	640	890	890
A2/New York City/1/71		445	640	640	890	640	890	445	640
A2/Oregon/1/71		890	1280	890	890	890	1280	1280	1280
A2/San Diego/1/71		640	1280	1280	1280	890	1780	1280	1280
A2/Utah/1/71		445	890	640	640	640	640	640	640

*Geometric mean HI titers of duplicate tests.

†Chicken antisera treated with receptor destroying enzyme.

**Shading indicates fourfold or greater difference from homologous antiserum titer.

Influenza type B accounted for the bulk of the influenza activity in 1970-71. Some areas had their first major outbreaks of influenza B in 5 years. All the isolates were inhibited by B/Massachusetts/3/66 reference antisera (representing the current influenza B vaccine component), but there was a general drift away from this strain (Table 3). Over one-third of the isolates showed a significant decrease in reactivity (fourfold or greater) with B/Massachusetts/3/66 antisera. Among the current strains, B/Czech/28/71, B/Mass/1/71, and B/New Hampshire/1/71 appear to stimulate the most broadly reactive antibody response.

TABLE 3

Hemagglutination inhibition reactions* of type B influenza viruses, 1940-71

Antigens \ Antisera†	B/Lee/40	B/GL/1739/54	B/Md/1/59	B/Taiwan/2/62	B/Mass/3/66	B/Czech/28/70	B/Canada/200/70	B/Mich/1/71	B/Mass/1/71	B/NH/1/71
B/Lee/40	320	40	80	<	<	<	<	<	<	<
B/GL/1739/54	10**	320	60	14	50	10	7	20	80	40
B/Maryland/1/59	20	160	1280	28	100	<	28	28	80	112
B/Taiwan/2/62	<	<	<	320	40	80	7	10	28	28
B/Mass/3/66	<	10	40	14	320	112	56	20	80	40
B/Czech/28/70	<	8	28	7	80	112	40	14	20	10
B/Canada/200/70	10	10	40	14	160	112	80	14	80	80
B/Dakar/1/70	<	20	<	10	50	80	20	20	56	56
B/Berkeley/1/71	<	7	77	7	40	40	10	20	56	56
B/Connecticut/1/71	<	20	10	10	160	160	80	112	160	224
B/Wash D.C./1/71	<	20	7	20	120	80	28	112	112	112
B/Florida/1/71	<	16	7	10	80	40	20	80	80	160
B/Georgia/1/71	<	20	10	20	320	160	40	224	160	160
B/GL/166/71	<	14	<	10	112	56	20	28	112	160
B/Hawaii/2/71	<	40	20	20	160	112	40	112	112	160
B/Mass/1/71	<	20	<	14	112	56	20	40	160	320
B/Mayo Clinic/1/71	<	14	<	10	80	40	20	40	112	224
B/Michigan/1/71	<	28	20	28	224	224	40	320	160	160
B/New Hampshire/1/71	<	10	<	10	80	56	14	20	112	224
B/New Jersey/1/71	<	14	10	20	160	160	40	112	160	112
B/SUNY/1/71	<	14	10	20	224	112	28	112	160	224
B/Texas/2/71	<	20	10	20	160	112	40	112	160	160
B/Univ.Maryland/1/71	<	10	7	10	80	56	20	56	160	224
B/Yale/1/70	<	20	7	20	160	112	28	80	112	160

*Geometric mean HI titers of duplicate tests. < = equal to or less than 1:5.

†Chicken antisera treated with receptor destroying enzyme.

**Shading indicates fourfold or greater difference from homologous antiserum titer.

B. Revision of Influenza Virus Nomenclature

There are several inadequacies in the present system of influenza virus nomenclature. The influenza A subtype designation does not take into account both the surface antigens -- the hemagglutinin and the neuraminidase -- and therefore, does not fully describe the antigenic character of the virus.

For example, A_2/Hong Kong/1/68 and A_2/Tokyo/3/67 have the same hemagglutinin but different neuraminidase antigens; this difference is not evident in the present terminology.

Moreover, the present system does not distinguish the antigenic relationship between viruses isolated for different animal species. Many avian influenza viruses, for example, possess neuraminidase antigens in common with viruses from man or horses. Finally, it is not clear with the present nomenclature when strains from different species are identical. For example, A/swine/Taiwan/1/70 has the same antigens as the A_2/Hong Kong/68.

A study group sponsored by the World Health Organization met in Geneva in September 1971 and recommended a system of nomenclature consisting of a strain designation and a description of the hemagglutinin and neuraminidase antigens.

The strain designation for human types A, B, or C remains the same except for the omission of subtypes for type A. The antigenic description follows the strain designation and includes in parenthesis an index describing the antigenic character of the hemagglutinin and an index describing the neuraminidase. Under the revised system the full designation of some human strains are, for example, A/PR/8/34 (H0N1), A/FM/1/47 (H1N1), A/Japan/305/57 (H2N2), and A/Hong Kong/8/68 (H3N2).

Influenza A virus recombinants (antigenic hybrids) which contain the hemagglutinin derived from one parent and the neuraminidase from another can also be described.

C. Collection and Shipment of Specimens for the Diagnosis of Viral Respiratory Diseases

The following recommendations for handling clinical specimens are used by the Center for Disease Control and may differ slightly for other laboratories. A rapid diagnosis of influenza outbreak may be made in many instances by the careful selection of unpaired acute and convalescent sera. This technique is outlined in the "Influenza-Respiratory Disease Surveillance Report", number 84, 1968.

1. Time of specimen collection:

For virus isolation. That specimens be collected within the first 48 hours after onset of symptoms. Although viruses may often be isolated after longer time periods, the likelihood of recovering most respiratory disease viruses is greatly diminished after 3 days.

For serodiagnosis. That blood specimens be collected during the acute stage of the disease, usually at the time specimens are collected for isolation, and again during convalescence 2-3 weeks later.

2. Collection of specimens:

For virus isolation. That specimens be collected from the oropharynx and nasal membranes with dry cotton swabs. Swabs from both sites may be com-

bined in a single screw-capped tube containing 5 ml cold trytose broth (pH 7.0-7.2) with 0.5 percent gelatin. Formalin preserved specimens are not acceptable.

For serodiagnosis. That serum be separated from the clot, lightly centrifuged to remove any remaining erythrocytes and stored at 4° C or -20° C until the acute and convalescent specimens can be examined simultaneously in the appropriate serologic tests.

3. Shipment of specimens:

For virus isolation. That specimens be placed on wet ice for transport to the laboratory for processing. If the time between collection and inoculation into an appropriate system for virus isolation exceeds 48 hours, specimens should be sealed tightly and shipped on dry ice.

For serodiagnosis. Sera may be shipped frozen or dry ice or at ambient temperatures provided specimens have been collected and processed aseptically.

For identification and confirmation. That viruses be shipped in the cold, lypholized, frozen on dry ice, or on living tissue at ambient temperatures. However, respiratory syncytial virus as well as some enteroviruses do not lypholize well and some myxoviruses demonstrate a loss in titer upon freezing. For these reasons, shipment of unknown viruses on living tissue is preferred and may be accomplished as follows. Inoculate viruses into tubes of susceptible tissue cultures and incubate overnight. Fill tubes with maintenance medium and pack for immediate shipment. No refrigeration is required. Tissue cultures should survive 1-2 weeks in transit under normal conditions.

RECOMMENDATION OF THE PUBLIC HEALTH SERVICE ADVISORY COMMITTEE ON IMMUNIZATION PRACTICES

INFLUENZA VACCINE

INTRODUCTION

Influenza occurs in the United States every year, but the incidence and geographic extent vary widely. Periodically, it appears in epidemic form as a result of antigenic variation in prevalent viruses and the relative susceptibility of the population. Both type A and type B influenza viruses undergo antigen changes. Antigen shifts usually occur slowly, but occasionally they are rapid and abrupt. Epidemics caused by type A influenza viruses occur more frequently and are generally more severe than those caused by type B.

Inactivated influenza vaccines* have not been used to control epidemic influenza in the general population. Their effectiveness is variable, and protection is relatively brief. Nevertheless, since they are the only available influenza preventives, they should be given to chronically ill patients and possibly to older persons in general. These two groups appear to be more vulnerable than others to serious cases of influenza and its complications. Because some influenza occurs each year, annual immunization of "high risk" patients is indicated as a routine procedure regardless of the amount of influenza expected in any specific geographic area.

INFLUENZA VIRUS VACCINES

The Division of Biologics Standards, National Institutes of Health, reviews influenza vaccine formulation regularly and recommends reformulation, when indicated, to include contemporary antigens. Strains of influenza A examined in the United States and abroad in 1970-71 did not differ significantly from the Hong Kong strain, A2/Aichi/2/68. For 1971-72, the composition of the vaccine will remain the same as the bivalent vaccine recommended for 1970-71. The adult dose of inactivated influenza vaccine will contain 400 chick cell agglutinating (CCA) units of type A2 antigen (A2/Aichi/2/68) and 300 CCA units of type B antigen (B/Mass/3/66).

Highly purified vaccines will be available from most manufacturers. These vaccines are equivalent in potency to earlier vaccines, but since they contain less nonviral protein, they are the recommended products where available. Patients who have had severe local or systemic reactions to influenza vaccine should experience less discomfort when highly purified vaccine is used.

VACCINE USAGE

General Recommendations

Annual vaccination is recommended for persons who have chronic debilitating conditions: 1) congenital and rheumatic heart disease, especially mitral stenosis; 2) cardiovascular disorders, such as arteriosclerotic and hypertensive heart disease, particularly with evidence of cardiac insufficiency; 3) chronic bronchopulmonary diseases, such as asthma, chronic bronchitis, cystic fibrosis, bronchiectasis, emphysema, and advanced tuberculosis; 4) diabetes mellitus and other chronic metabolic disorders.

Although the value of routinely immunizing all older age persons is less clear, those patients who have incipient or potentially chronic disease, particularly affecting cardiovascular and bronchopulmonary systems, should also be considered for annual immunization.

Immunizations of persons who provide essential community services may also be considered if local priorities justify. However, before undertaking such programs, responsible physicians must take into account a number of reasonable constraints: difficulties inherent in predicting influenza epidemics, variability of vaccine effectiveness, incidence of adverse side effects, cost, availability of vaccine, and risk of diverting vaccine from those with chronic debilitating conditions who are at risk.

Schedule

The primary series consists of 2 doses administered subcutaneously, preferably 6 to 8 weeks apart. (Dose volume for adults and a detailed schedule for children are specified in the manufacturers' labeling.) Persons who have had 1 or more doses of vaccine containing the Hong Kong strain antigen (all influenza vaccines since 1968-69) need only a single subcutaneous booster dose of bivalent vaccine. All others should receive the full primary series. Vaccination should be scheduled for completion by mid-November.

Precautions

Influenza vaccine is prepared from viruses grown in embryonated eggs and ordinarly should not be administered to persons clearly hypersensitive to egg protein, ingested or injected.

*The official name of the currently available product is Influenza Virus Vaccine, Bivalent.

Published MMWR: Vol. 20, No. 29, 1971.

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Key to all disease surveillance activities are the physicians who serve as State epidemiologists. They are responsible for collecting, interpreting, and transmitting data and epidemiological information from their individual States; their contributions to this report are gratefully acknowledged. In addition, valuable contributions are made by State Laboratory Directors; we are indebted to them for their valuable support.

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