

COMMUNICABLE DISEASE CENTER

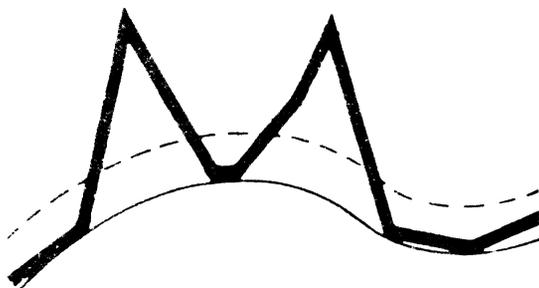
INFLUENZA - RESPIRATORY DISEASE SURVEILLANCE

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PREFACE

Summarized in this report is information received from State Health Departments, university investigators, virology laboratories and other pertinent sources, domestic and foreign. Much 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: Chief, Influenza-Respiratory Diseases Unit*, Epidemiology Branch, Communicable Disease Center, Atlanta, Georgia 30333.

Communicable Disease Center

David J. Sencer, M.D., Chief

Epidemiology Branch

Alexander D. Langmuir, M.D., Chief

Influenza-Respiratory Diseases Unit*

H. Bruce Dull, M.D., Chief
William H. Stuart, M.D.
Kenneth H. Williams, Jr., M.D. (Editor)

Statistics Section

Mrs. Ida L. Sherman, M.S. Acting Chief

In Collaboration with:

Virology Section
Laboratory Branch

Roslyn Q. Robinson, Ph.D.,
Acting Chief

Respirovirus Unit
Serving as International Center
for the Americas

Walter R. Dowdle, M.D.,
Acting Chief

*Staffing for the Unit applicable to 1965-66 influenza season.

INTRODUCTION

As in previous years, the regularly published Morbidity and Mortality Weekly Reports have included pertinent epidemiological data regarding influenza. This present publication is designed as a summary of the year's experience and includes: 1) a review of the 1965-66 influenza experience in the United States; 2) an international summary; 3) a laboratory report, and 4) a collection of epidemic investigation reports.

Data received from official health and research agencies in this country and from publications of the World Health Organization and related organizations concerning international influenza surveillance form the bases of the interpretations and analysis presented here.

I. U.S. SUMMARY

The data used in this report are derived from a number of sources. Weekly correspondence with each of the State Health Departments and weekly review of Pneumonia-Influenza Mortality from 122 U.S. Cities provided the major portion of the information. Supplementing this regularly received data is an "Influenza Appraisal Summary", through which each State epidemiologist summarized his State's experience, indicating initial appearance of influenza, peak incidence, age groups, and epidemic indices most affected. Table 1 and Figures 1-4 are a presentation of this information gathered through May 30, 1966.

Categories of "Geographic Extent" are approximated: a) Isolated - influenza recognized in only a limited number of small well defined population units; b) Regional - influenza recognized in counties comprising less than 50 percent of the State's population; and c) Widespread - influenza recognized in counties comprising more than 50 percent of the State's population.

On the bases of these data, certain generalities regarding the 1965-66 influenza season may be drawn:

1. The presence of influenza (type A and/or B) was identified clinically and epidemiologically in 49 of the 50 States and confirmed by laboratory means in all but one of these. (See Table 1 - complete through May 30, 1966. Kentucky reported isolation of influenza after May 30.)
2. Strains of A2 virus were isolated in 17 States and serologically confirmed in 12 others (29 in all). Strains of influenza B virus were isolated in 25 States and serologically confirmed in 16 others (41 in all). Twenty-one States confirmed the presence of both types A and B. Typically those areas involved by both viruses experienced two waves of increased influenza occurrence, although occasionally both types occurred simultaneously.
3. Influenza B, appearing earlier than type A, was first recognized in the northeast area of the country. Influenza A was first noted on the west coast. (See Figures 1 and 2.) Each virus type then spread centrally from its initial focus, resulting in the widespread patterns of distribution noted. (Figures 3 and 4.)

Influenza B also appeared early in the northwest part of the country and was later succeeded by outbreaks of type A influenza.

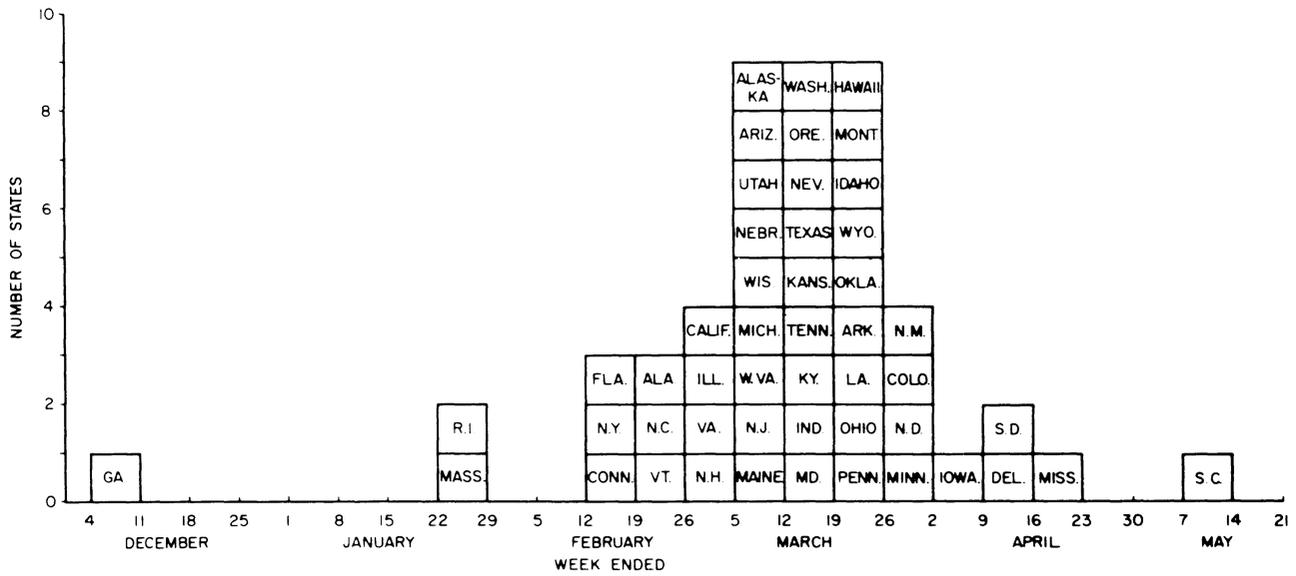
Table 1
 UNITED STATES INFLUENZA (WINTER 1965-66)
 STATE SUMMARY*

Division State	Peak Occurrence	GEOGRAPHIC EXTENT**			Laboratory	Confirmation
					Isolation	Serology
NEW ENGLAND						
Massachusetts	Jan - late	Widespread B	B	B
Rhode Island	Jan - late	Widespread B	-	B
Connecticut	Feb - mid	Widespread B	B	B
Vermont	Feb - late	Widespread B	B	B
New Hampshire	Feb - late	Widespread B	-	B
Maine	Mar - early	Widespread B	B	B
MIDDLE ATLANTIC						
New York	Feb - mid	Isolated B	B	B
New Jersey	Mar - early	Regional B	B	B
(New York City)	Mar - late	Isolated A,B	A ₂ , B	A
Pennsylvania	Mar - late	Isolated B	B	-
SOUTH ATLANTIC						
Georgia	Dec - early	Isolated A,B	B	A, B
Florida	Feb - mid	Widespread B	B	B
North Carolina	Feb - late	Regional B	-	B
Virginia	Feb - late	Isolated A	B	A, B
W. Virginia	Mar - early	Isolated A	-	A, B
Maryland	Mar - mid	Isolated B	B	-
(Washington, D.C.)	Mar - late	Isolated B	B	-
Delaware	Apr - mid	Isolated A	A ₂	-
South Carolina	May - mid	Isolated B	-	B
EAST NORTH CENTRAL						
Illinois	Feb - late	Isolated A	Regional B	A ₂ , B	A, B
Wisconsin	Mar - early	Isolated B	B	B
Michigan	Mar - early	Isolated A	Regional B	A ₂ , B	B
Indiana	Mar - mid	Regional A	-	A
Ohio	Mar - late	Isolated A,B	-	A, B
EAST SOUTH CENTRAL						
Alabama	Feb - late	Isolated B	-	B
Kentucky	Mar - mid	Regional	-	-
Tennessee	Mar - mid	Isolated A	Regional B	B	A, B
Mississippi	Apr - mid	Isolated B	-	B
WEST SOUTH CENTRAL						
Texas	Mar - mid	Isolated A,B	A ₂	A, B
Oklahoma	Mar - late	Regional A,B	A ₂ , B	A, B
Arkansas	Mar - late	Isolated A	-	A
Louisiana	Mar - late	Isolated A,B	A ₂ , B	-
WEST NORTH CENTRAL						
Missouri	-	-
Nebraska	Mar - early	Regional B	B	B
Kansas	Mar - mid	Isolated A,B	A ₂	A, B
Minnesota	Mar - late	Regional B	Widespread A	A ₂	A, B
North Dakota	Mar - late	Isolated B	Widespread A	-	A, B
Iowa	Apr - early	Regional A	A ₂	-
South Dakota	Apr - mid	Isolated B	-	B
MOUNTAIN						
Arizona	Mar - early	Widespread A	-	A
Utah	Mar - early	Isolated A	A ₂	A
Nevada	Mar - mid	Regional	-	-
Idaho	Mar - late	Isolated B	Widespread A	A ₂ , B	A
Montana	Mar - late	Isolated B	Widespread A	A ₂	A, B
Wyoming	Mar - late	Isolated B	-	B
Colorado	Mar - late	Isolated A	A ₂	A
New Mexico	Mar - late	Isolated A	Regional B	-	A, B
PACIFIC						
California	Feb - late	Isolated B	Widespread A	A ₂ , B	A, B
Alaska	Mar - early	Widespread A,B	B	A, B
Washington	Mar - mid	Widespread A,B	B	A, B
Oregon	Mar - mid	Regional B	Widespread A	A ₂ , B	A, B
Hawaii	Mar - late	Widespread A	A ₂	A

*Information from State Health Department Influenza Appraisal Summary, Research Institutions, University Centers and CDC Respiriavirus Laboratory.

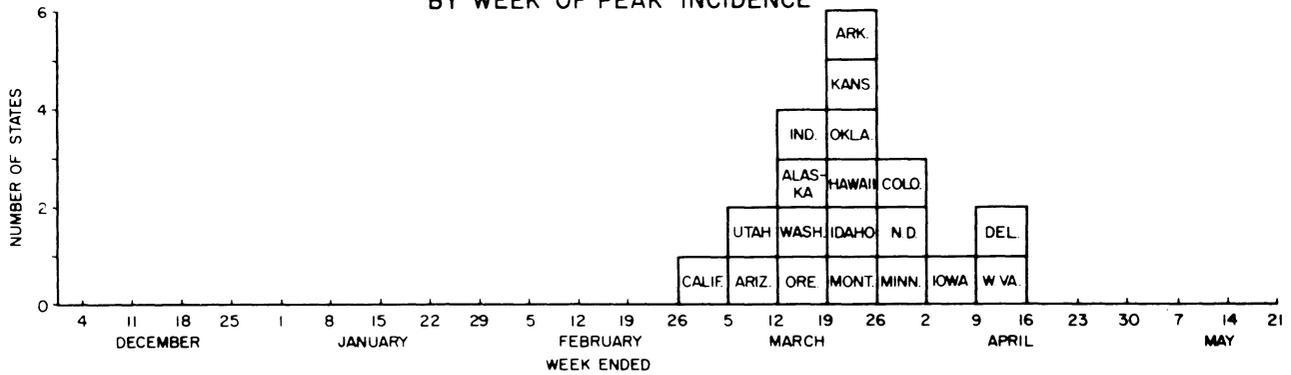
**Terms "Isolated", "Regional", and "Widespread" are defined in text, see p. 1

Figure 1
INFLUENZA
STATES BY WEEK OF REPORTED PEAK INCIDENCE *
1965-1966 WINTER



* AS REPORTED BY STATE EPIDEMIOLOGISTS IN CDC INFLUENZA APPRAISAL SUMMARY.

Figure 2
STATES REPORTING PREDOMINANT OR WIDESPREAD TYPE A
BY WEEK OF PEAK INCIDENCE



STATES REPORTING PREDOMINANT OR WIDESPREAD TYPE B
BY WEEK OF PEAK INCIDENCE

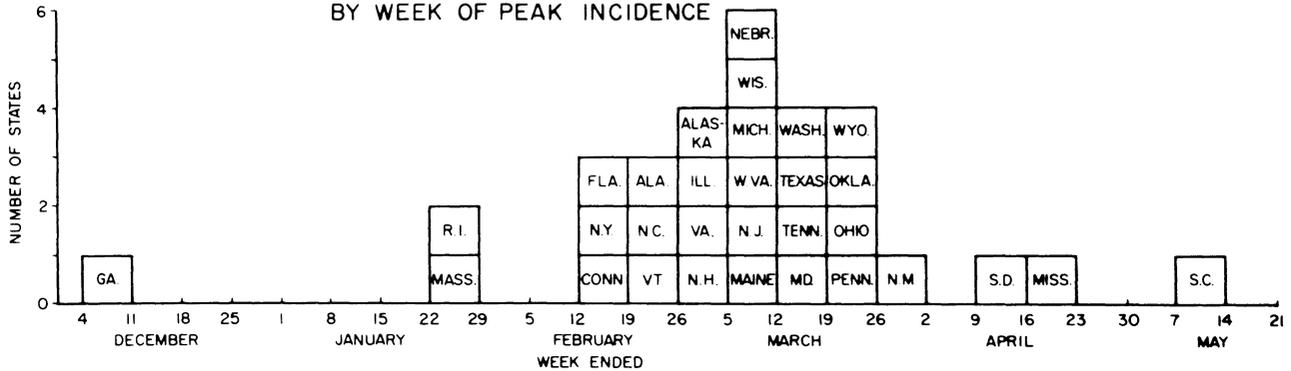


Figure 3

DISTRIBUTION OF INFLUENZA A

UNITED STATES
1965 - 66

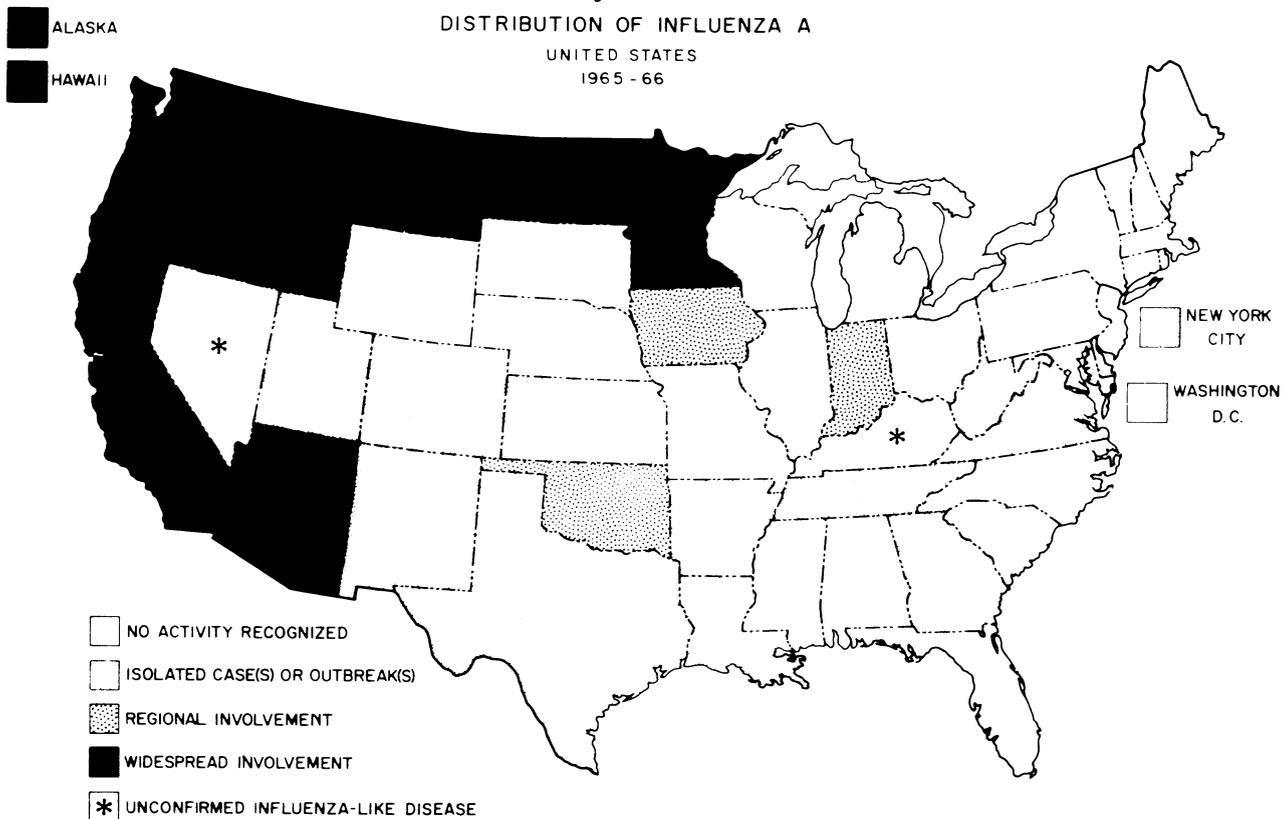


Figure 4

DISTRIBUTION OF INFLUENZA B

UNITED STATES
1965 - 66

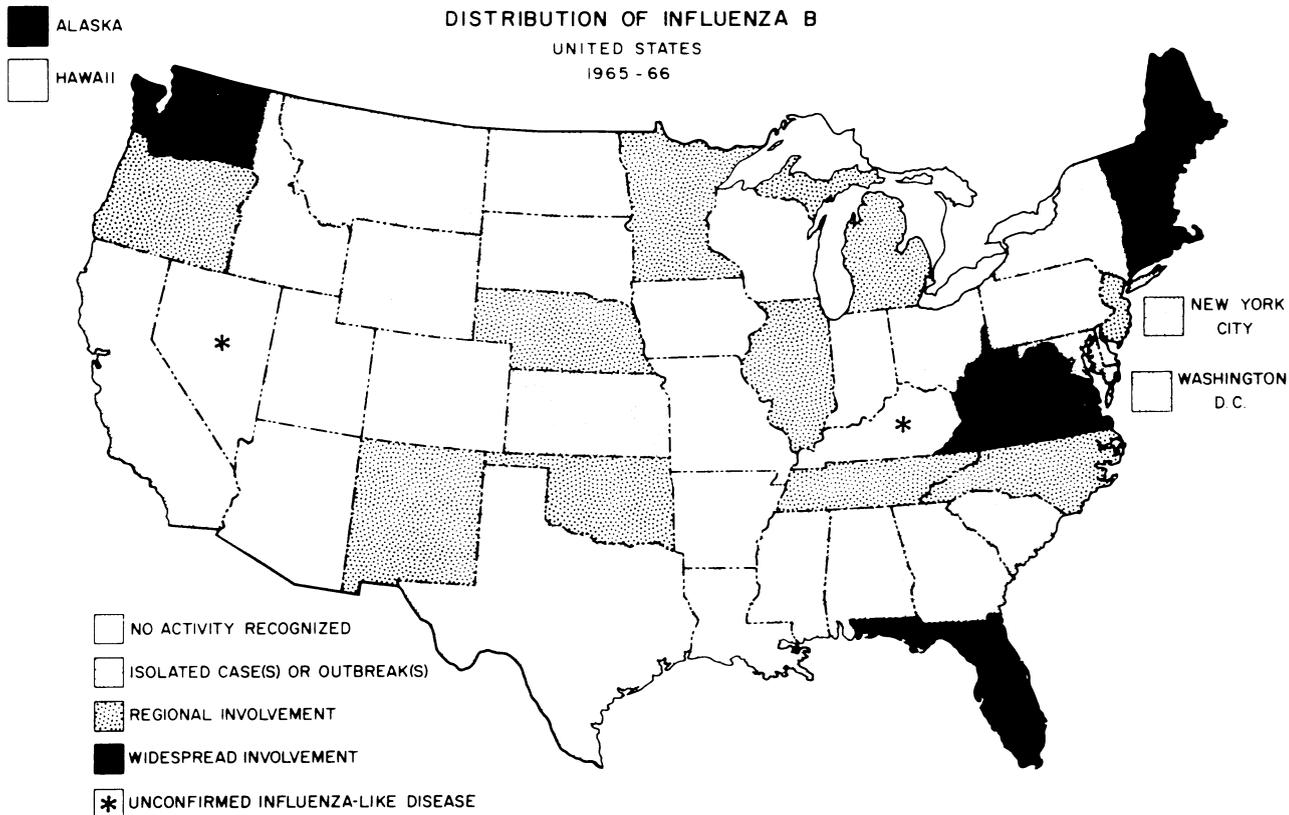
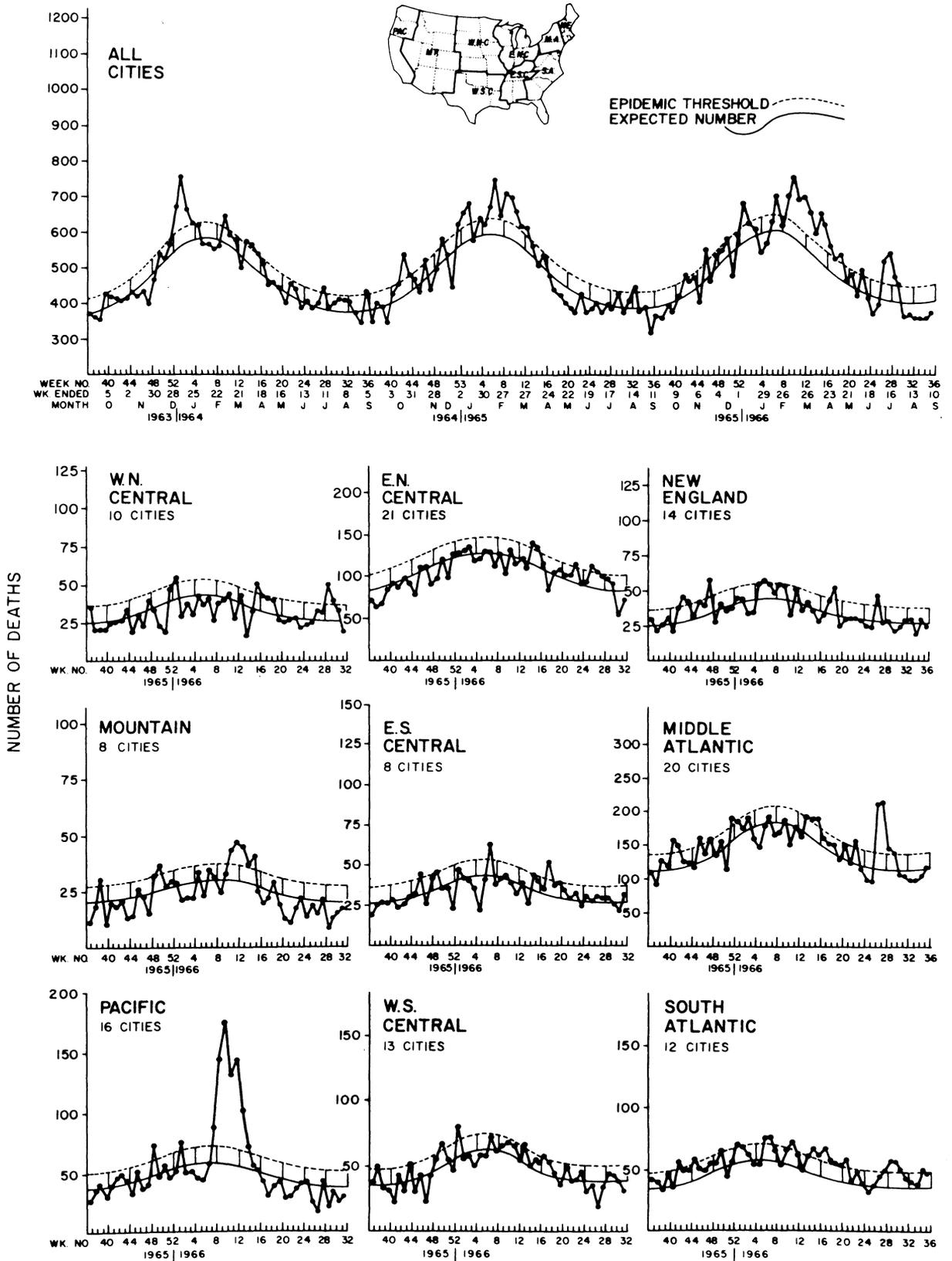


Figure 5.

PNEUMONIA-INFLUENZA DEATHS IN 122 UNITED STATES CITIES



4. Pneumonia-influenza mortality reported to CDC by 122 cooperating United States cities showed minor excursions above the epidemic threshold from mid-February to mid-May. (See Figure 5.) The excess mortality was almost entirely contributed by the State of California, where every parameter of influenza mortality (increased school and industrial absenteeism, hospital admissions, and outpatient visits) reached levels not exceeded since 1960 when A2 influenza also occurred in the State.
5. As in past experience, areas involved with type A2 influenza experienced considerably more excess mortality than those where type B influenza was identified. In areas where type B virus was most widespread, school absenteeism without an equivalent rise in industrial absenteeism or hospital admissions was noted, reflecting the usually younger age group involved.
6. Despite a continuous antigenic drift of type A2 viruses, all isolates were clearly related to strains identified in the past several years. The broadly reacting antibody stimulated by certain of the contemporary strains may have a bearing on the selection of virus types for vaccine purposes. (See Laboratory Report.) No great shift in influenza B antigenic pattern was noted.

II. INTERNATIONAL SUMMARY

Reports published in the WHO Weekly Epidemiological Record and received by the WHO Influenza Center for the Americas at CDC form the basis for the 1965-66 International Influenza Summary. (See Table 2.) Because of the inherent differences in reporting, these data can be expected to give only a general appraisal, and omissions and minor inconsistencies may represent as yet unpublished data and incomplete reports.

Of the 23 countries reporting the identification of outbreaks of influenza from late spring 1965, 18 reported evidence of type B virus activity, 18 noted type A, and 13 demonstrated both types. In many countries, these two types often appeared in relatively contiguous areas. Ten of the 23 countries reported a predominance of type B influenza, 6 noted mostly type A, and the remaining 6 appeared to have equivalent amounts of both types.

In general, the observed clinical characteristics of influenza were considered to be mild. Type B outbreaks in particular were repeatedly noted to involve primarily school children. Heightened school absenteeism was generally recognized as one of the best indices of incipient community epidemics.

Type A influenza likewise involved children in many areas, but tended to be less confined to the younger age groups. In fact, in the countries where both virus types were identified, it was sometimes possible to relate the adult illnesses and often the fatal cases to type A infections. In both Great Britain and the United States, these general relationships were observed.

In Europe, where the most consistent reporting was available, type B influenza appeared first in central regions from early to middle fall and first spread locally but later, by the end of the year, toward the western and northern parts of the continent. In late December and in the subsequent months, type A2 virus activity appeared generally to increase and to cause concurrent epidemics.

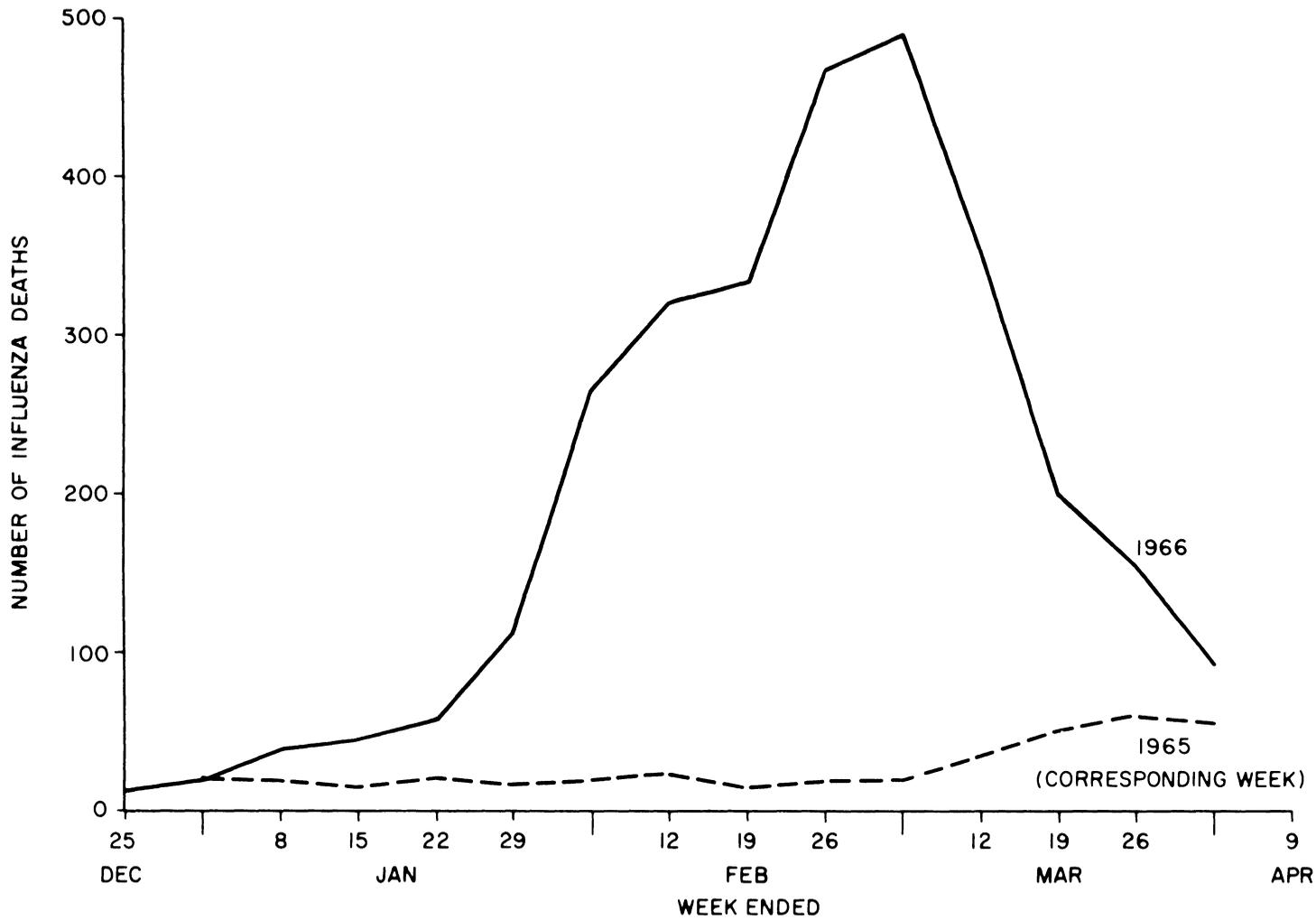
Elsewhere in the world, patterns of spread were difficult to trace in detail, but the widespread seeding of both types A2 and B influenza virus strains was clearly evident throughout the season.

TABLE 2

INTERNATIONAL INFLUENZA SUMMARY - 1965-66

Country	First Recognized	Laboratory Confirmation		Predominant Virus Type(S)
		Isolation	Serology	
<u>EUROPE</u>				
Czechoslovakia	Sept. 1965	B	B	B
Hungary	Nov. 1965	B	B	B
Bulgaria	Dec. 1965	A2,B	A,B,C	B
Romania	Dec. 1965	B	A,B	B
England-Wales	Jan. 1966	A2,B	A,B	A,B
Scotland	Jan. 1966	B	A,B	B
Netherlands	Jan. 1966	A2,B	A,B	B
France	Jan. 1966	A2,B	A,B	A,B
E. Germany	Feb. 1966	-	B	B
Sweden	Feb. 1966	A2	A,B	A,B
U.S.S.R.	Feb. 1966	-	B	B
Switzerland	Mar. 1966	-	A	A
Denmark	Mar. 1966	-	A,B	A
Fed.Rep.-Germany	Apr. 1966	A,B	A,B	A,B
Finland	Apr. 1966	-	-	A
<u>AFRICA</u>				
Senegal	Jan. 1966	-	A	A
<u>ASIA-OCEANIA</u>				
Australia	Apr. 1966	B	B	B
Philippines	May 1965	A2	A	A
Thailand	Oct. 1965	A2	A	A
Japan	Dec. 1965	A2,B	A,B	B
Hong Kong	Jan. 1966	A2,B	A,B	-
<u>NORTH AMERICA</u>				
U.S.A.	Nov. 1965	A2,B	A,B	A,B
Canada	Feb. 1966	A2	A,B	A,B

Figure 6
INFLUENZA IN ENGLAND AND WALES
DEATHS PER WEEK
1965 - 1966



Great Britain

Outbreaks of influenza, predominantly involving school-age populations, were first reported from northern parts of England and from southern Scotland in middle January. Type B influenza virus was readily identified as the etiologic agent in many of the school-centered outbreaks. Quite commonly the illness was mild, but high attack rates of up to 50 percent or more were experienced.

Coincident with the recoveries of type B influenza virus from school children were demonstrations of type A2 influenza virus activity, not characteristically related to the school outbreaks, but rather to adult cases observed in nursing homes and geriatric wards of some hospitals.

Subsequent to the initial January appearance of the virus, during the next two months, influenza spread southward in England and northward into much of Scotland. Noteworthy was the paucity of evidence that London was substantially involved in the outbreak. There, only minor reflections in increased influenza mortality were observed although British news media reported some influenza-like illnesses occurring among school children in areas adjacent to the city.

Of particular interest because of the dual virus nature of the epidemic (especially in view of the frequent observation that many adult and fatal cases of influenza were associated with type A2 virus) is a review of the reported weekly number of deaths from England and Wales registered as being due to influenza. Figure 6 presents weekly numbers of influenza deaths for 1966 in contrast to 1965, a non-epidemic year. The week of peak mortality in 1966 shows more than 20 times the number of deaths reported in the comparable week of 1965. Less marked but significant increases in numbers of deaths attributed to pneumonia and bronchitis paralleled the rise in influenza deaths. The peak occurrences of the three categories essentially coincided.

During the 11-week period encompassing the height of increased influenza mortality (beginning with the week ending January 22, 1966), it is of interest to look at the 2,800 influenza deaths in terms of age distribution. Approximately 95 percent of them occurred in individuals 45 years of age or more, some 80 percent in those of 65 years or more, and nearly 60 percent in persons 75 years or more. Information is not available regarding the proportion of fatal cases with chronic illness or other possibly predisposing conditions.

Senegal, West Africa*

Introduction

Near the end of December, an outbreak of suspect influenza was reported from the Region of the Fleuve in a remote sector of northeastern Senegal. A little later, other foci appeared in nearby areas to the east in the Region of Diourbel; however, here the epidemic did not appear to present the same degree of severity.

*Translated from the Study by Dr. Yves Robin, Chief, Virology Service, Pasteur Institute, Dakar, from the report to the Sixth Technical Conference of the S.C.C.G.E., Bobo-Dionlasso, Upper Volta, March 21, 1966.

Investigations

In the Region of the Fleuve, the two counties of Matam and Podor were particularly hard hit. A thorough epidemiologic inquiry was conducted there which will serve as a basis for defining the morbidity and mortality rates of the epidemic.

Twenty-seven villages representing a population of about 13,500 inhabitants were investigated. The physicians conducted 7,086 examinations of ambulatory patients and made 262 visits to bed patients. Three hundred twenty-four deaths were recorded. From a thorough survey in the villages the attack rate was found to be 54 percent. The case fatality ratio reached 4.4 percent and the general mortality rate was 2.4 percent, or 2,400 per 100,000 population. There was no age group specially afflicted.

Investigations revealed that the epidemic began during the second half of December, that it peaked during mid-January, and that its abrupt decline began as early as January 20.

Elsewhere the disease did not assume the same degree of severity. In the Region of the Fleuve, there had been noted a considerable number of malignant, severely toxic forms (accompanied by complications), which killed the patient in two or three days. The complications were of the pulmonary, digestive, and especially neurologic type, the latter presenting with meningeal signs. Initially, the neurologic complications brought about the fear that an epidemic of cerebrospinal meningitis was being experienced. In certain villages, the concurrence of localized measles outbreaks probably contributed to the high mortality rate. But this does not explain the general severity of the epidemic throughout the entire region. It would seem that climatic conditions might have intervened, with a drop of nocturnal temperatures and the "harmattan" (cold winter winds) that blew from mid-December to January. In the regions to the east, the disease again afflicted more than half of the exposed population, yet the same mortality rate was not approached.

Laboratory Studies

The initial serologic study performed with the help of the complement fixation tests using soluble antigens, determined that the epidemic was due to a type A influenza virus. The overall percentage of serologic rises, interpreted as indicating recent infection, attained 55 percent. Thus, one is able to demonstrate again and confirm the previously calculated attack rates which were obtained from the interview survey in the villages.

The hemagglutination inhibition test confirmed that it was indeed type A influenza. It would seem likely that the few small titer rises observed with type B antigen could be attributed to an anamnestic cross-reaction. To date no viral isolations have been made.

Summary

Between December and February an epidemic of type A influenza was experienced in the Republic of Senegal. Attack rates in all affected regions appeared to be near 50 percent. In the two counties in which the disease first appeared the disease was inexplicably attended by an extremely high case-fatality ratio.

III. LABORATORY REPORT

This is to report on the antigenic analysis of current influenza A and B viruses. The strains examined were chosen as representative of the total received at the World Health Organization International Influenza Center for the Americas according to their reactions in preliminary tests and on a geographic basis.

Results of a single reciprocal hemagglutination inhibition test using RDE-treated immune chicken sera and allantoic fluid antigens are presented in the enclosed tables. For purposes of discussion, cross reactions which are eight-fold lower than the homologous antiserum titer are arbitrarily regarded as evidence of dissimilarity and are indicated by open areas in the table; more closely related strains are indicated by shaded areas. A zero or dash in the table is used to indicate titers of less than 1:10.

Type A influenza viruses isolated during the 1965-66 "influenza season" and examined at the IICA appear to comprise a relatively homogeneous group showing variable relationships to strains isolated in earlier years. If one considers the reactivity of certain antisera with all viruses it appears that most contemporary strains show significant degrees of "antigenic drift" away from the A2/Japan/305/57 prototype. However, all contemporary strains are clearly related to the A2/Japan/170/62 strain of virus. It may also be noted that the A2/Taiwan/1/64 strain, which previously has been observed to differ from both A2/Jap/305/57 and A2/Jap/170/62, appears to be closely related to the A2/Itsukaichi/1/65 strain first isolated in December 1964. It is of interest that the A2/Thailand/385/65 strain which was first isolated in October 1965 is quite closely related to type A viruses isolated in the United States during the following five month period.

If one considers reactivity of current strains with all antisera, there is little evidence of dissimilarity. The differences observed with the A2/California/1/66 strain appear to be the result of lowered avidity of the test antigen (note that the A2/Cal/1/66 antiserum titer is lower with the homologous antigen than with heterologous antigen) rather than to true antigenic differences.

Certain of the contemporary strains such as A2/Thailand/385/65, A2/Canada/1/66, A2/Iowa/1/66, and A2/Montana/1/66 stimulate the production of very broadly reacting antibody, while A2/Albany/3/65 and A2/Iowa/1/66 react broadly with all test antisera. These characteristics have a bearing on the selection of virus strains for vaccine or diagnostic reagents.

Type B influenza viruses isolated during the 1965-66 season show variable relationships to viruses isolated in earlier years. All contemporary strains tested are clearly different for the B/Lee/40 virus but each strain bears a distinct relationship to one or more viruses isolated since that time. The B/Wash/1/66, B/Mich/1/66, B/Albany/1/66, B/Georgia/1/65 and 66, and B/Oregon/1/66 strains are related on the basis of one-way cross reactions to B/Great Lakes/1739/54 virus. Antisera prepared from most current strains react with both B/Maryland/1/59 and B/Singapore/3/64 viruses. However, if one considers reactivity of current strains with B/Maryland/1/59 and B/Singapore/3/64 antisera, there is clear evidence of dissimilarity, with most strains more clearly resembling B/Singapore/3/64. Three viruses, B/Taiwan/2/62, B/India/363/64 and B/Colorado/2/65 were previously thought to be quite different from other type B viruses. While the B/Taiwan/2/62 virus remains somewhat different, it is apparent that most current strains show clear relationships to B/Colorado/2/65 and B/India/363/64 viruses.

The above properties tend to bind the type B viruses closer together rather than permitting orderly subdivision, and a clear line of transition is impossible to define.

Certain of the contemporary strains such as B/Washington/1/66, B/Michigan/1/66 and B/Albany/1/66 stimulate the production of very broadly reacting antibody while others, such as B/G1/I-7/66 and B/Singapore/3/64 react broadly with most antisera. These properties may have an important bearing on the selection of strains of virus for vaccine or diagnostic reagent production. All contemporary strains of type B influenza require treatment of sera with receptor destroying enzyme for removal of non-specific inhibitors.

HEMAGGLUTINATION INHIBITION: TYPE A INFLUENZA VIRUSES

ANTIGENS CHICKEN ANTISERA	ANTIGENS																			
	A/PR/8	A1/FM/1	A2/Jap/305	A2/Jap/170	A2/Tw/1	A2/Its/1	A2/Thai/385	A2/Alb/3	A2/Berk/1	A2/Cal/1	A2/Cal/3	A2/Can/1	A2/De1/1	A2/Iowa/1	A2/Mon/1	A2/UC/1	A2/Panama/1/65	A2/Alaska/1/66	A2/Cal/10/66	A2/Kansas/1/66
A/PR/8/34	640	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
A1/FM/1/47	0	240	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
A2/Jap/305/57	0	0	640	160	160	160	80	320	80	40	80	80	80	320	160	320	40	80	160	80
A2/Jap/170/62	0	0	640	640	320	320	320	1280	160	160	320	320	160	>1280	640	640	160	320	640	320
A2/Taiwan/1/64	0	0	40	40	320	640	80	240	40	10	80	40	80	320	160	80	40	40	80	40
A2/Itsukaichi/1/65	0	0	80	40	>1280	1280	80	640	80	10	160	160	80	>1280	160	160	80	80	160	160
A2/Thailand/385/65	0	0	160	320	320	640	640	>1280	320	160	640	640	320	>1280	1280	640	640	1280	1280	320
A2/Albany/3/65	0	0	640	480	>1280	1280	160	1280	320	320	320	320	240	>1280	640	>1280	160	320	640	320
A2/Berkley/1/66	10	0	80	80	320	320	320	320	320	20	160	320	160	640	640	640	160	320	640	160
A2/California/1/66	0	0	40	160	320	320	160	640	160	80	160	320	160	640	640	640	160	320	640	160
A2/California/3/66	0	0	80	80	320	640	320	1280	320	80	640	640	320	>1280	>1280	640	320	320	1280	480
A2/Canada/1/66	10	0	80	320	320	640	640	1280	640	160	640	640	320	>1280	>1280	>1280	320	1280	1280	480
A2/Delaware/1/66	0	0	20	80	160	160	160	80	160	20	320	160	320	160	320	80	80	80	320	160
A2/Iowa/1/66	0	0	160	320	640	640	160	640	320	160	320	320	320	640	240	480	160	320	320	160
A2/Montana/1/66	0	0	160	480	320	320	640	640	320	160	640	640	640	>1280	640	640	320	320	640	640
A2/Univ. of Chicago/1/66	0	0	40	160	240	320	320	320	160	160	320	320	160	640	320	640	80	320	320	160

Hemagglutination Inhibition Tests With Type B Influenza Viruses

Chicken Antisera	Antigens																				
	B/Lee/40	B/GL/54	B/Md/59	B/Tai/62	B/India/64	B/Sing/64	B/Col/64	B/Ga/65	B/Ga/66	B/NYC/66	B/Va/66	B/Mass/66	B/Alaska/66	B/Wash/66	B/Mich/66	B/Alb/66	B/Maine/66	B/Wisc/66	B/UC/66	B/Oreg/66	B/GL/66
B/Lee/40	640	20	20	- *	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
B/Great Lakes/1739/54	80	640	80	40	40	80	160	160	160	80	80	40	80	40	80	40	40	80	80	160	40
B/Maryland/1/59	20	40	320	-	40	160	10	20	40	80	40	40	40	20	40	40	20	40	80	40	80
B/Taiwan/2/62	10	20	40	320	20	80	80	40	40	40	20	40	20	20	40	20	20	20	40	40	80
B/India/363/64	-	-	40	-	160	40	10	20	20	320	160	80	160	160	20	160	160	320	20	20	20
B/Singapore/3/64	-	20	160	10	80	320	20	160	160	80	40	160	40	20	80	40	40	40	40	160	160
B/Colorado/2/64	-	20	40	80	80	80	160	40	80	80	40	80	80	20	40	40	40	80	80	40	80
B/Georgia/1/65	10	40	40	10	40	160	80	320	320	160	80	40	80	40	160	80	40	80	80	320	320
B/Georgia/1/66	10	40	40	20	40	80	40	320	320	80	40	40	80	20	320	80	20	80	320	320	160
B/New York City/1/66	-	20	40	10	80	40	20	20	40	160	80	80	160	80	20	160	160	160	20	40	40
B/Virginia/1/66	-	10	40	10	320	80	40	80	80	160	160	160	160	160	80	160	160	320	80	80	80
B/Massachusetts/1/66	-	40	80	20	320	160	40	80	80	320	160	320	320	160	80	320	320	320	80	160	80
B/Alaska/1/66	-	10	40	80	80	320	80	160	320	320	320	320	320	160	160	320	160	320	320	320	320
B/Washington/1/66	10	40	160	40	160	160	40	80	80	320	160	160	320	160	80	320	160	320	80	160	160
B/Michigan/1/66	-	80	80	20	80	320	320	320	320	320	80	160	160	80	320	160	160	160	320	640	320
B/Albany/1/66	-	40	40	20	160	80	40	80	80	320	160	160	320	160	40	160	160	160	80	80	80
B/Maine/1/66	-	20	40	40	160	80	80	80	80	320	160	160	320	160	80	320	160	320	80	80	160
B/Wisconsin/1/66	-	20	320	40	640	320	80	80	80	640	320	640	1280	320	80	1280	640	640	80	160	160
B/Univ.Chicago/1/66	-	20	320	20	80	320	40	640	640	160	80	80	160	80	320	160	160	160	160	160	640
B/Oregon/1/66	-	80	320	40	160	640	80	1280	1280	160	80	160	160	80	640	160	160	160	1280	1280	1280
B/Great Lakes/1/7/66	-	20	80	10	40	160	40	160	160	40	40	40	40	20	160	40	40	40	160	160	320

IV. SPECIAL REPORTS

1. Epidemic Investigations

Polk, Nebraska

On March 19, 1966 an outbreak of acute febrile respiratory disease among high school students was reported from Polk, Nebraska to the State Health Department by a local physician. Accompanying the report was a request for assistance in an epidemic investigation. The Communicable Disease Center was contacted, and in conjunction with the State Health Department a joint investigation of the outbreak was undertaken.

The epidemic occurred in Polk, Nebraska, located approximately 60 miles northwest of Lincoln, Nebraska. Polk is predominantly an agricultural, rural community with a population of approximately 800 persons. It is moderately prosperous with most of the population living in single well-kept dwellings.

The striking feature of the epidemic was greatly increased absenteeism among high school students. During the 3-day period from March 7 through March 10, 52 percent of the high school students were absent with a maximum absenteeism of 37 percent occurring on March 10. Considerably less absenteeism was observed in the local elementary schools.

Utilizing family names selected from the high school absentee census, a telephone survey evaluating the extent of the epidemic was conducted on March 12 and 13. Seventy-three families (involving a total study population of 233 individuals) were contacted. The data from this survey form the basis for Table 1, which illustrates incidence of respiratory disease by history of week of symptom onset; and Table 2, which demonstrates age specific attack rates.

Table 1

INCIDENCE OF ACUTE RESPIRATORY DISEASE BY WEEK OF ONSET
FROM TELEPHONE SURVEY OF 73 FAMILIES (233 PERSONS)

<u>Week</u> <u>Beginning</u>	<u>Number Ill</u>	<u>Percentage</u>
2/21	4	1.7
2/28	10	4.3
3/7	39	16.7
Total 2/21-3/13	53	27.7

Table 2

ATTACK RATES BY AGE
FROM TELEPHONE SURVEY OF 73 FAMILIES (233 PERSONS)

<u>Age</u> <u>Years</u>	<u>Number Ill</u>	<u>Number in</u> <u>Age Group</u>	<u>Percentage</u>
0-4	2	14	14
5-9	9	22	41
10-14	9	24	38
15-19	17	30	57
20-39	3	39	8
40-59	8	59	14
≥ 60	5	45	11
All Ages	53	233	22.7

The overall attack rate in the study population was 22.7 percent with the highest age specific attack rate occurring in the 15 to 19 year age group. The telephone survey and the epidemic investigation would appear to have been conducted near the time of peak incidence of the respiratory disease. Both of these factors may be due to the fact that families were selected for the telephone survey from the high school absentee census.

The most frequently noted symptoms among clinical cases were malaise, myalgia, headache, chills, and fever. Rhinorrhea, ocular pain, and conjunctivitis occurred occasionally. It was noted that some children experienced fever, headache and mild sore throat on the first day of illness, with clinical relapse on the third day following apparent recovery. The illness generally lasted from four days to one week; residual fatigue was a common sequellae.

Twenty-four throat washings and serum specimens were obtained for laboratory analysis from acutely ill people. In addition, nasal smears were collected for fluorescent antibody studies. The results of the fluorescent antibody studies were inconclusive, but type B influenza virus was isolated from throat washings utilizing monkey kidney tissue culture.

In summary, an outbreak of respiratory illness observed in a rural Nebraska high school was subsequently determined by laboratory studies to be due to a type B influenza virus. The clinical illness was typical of influenza, but through a peculiar epidemiologic characteristic, the epidemic involved predominantly the high school age group.

(Reported by Dr. E. A. Rogers, Nebraska State Director of Health, and by EIS officers assigned to the Omaha-Douglas County Health Department and the Kansas City Field Station.)

Brunswick, Maine

An outbreak of influenza was suspected at the U.S. Naval Air Station, Brunswick, Maine, during the week ending February 12. At that time 30 percent of cases reporting to military sick call and 45-50 percent of dependent personnel appearing at the outpatient department were found to have influenza-like symptoms. This proportion of cases with respiratory symptoms was considerably above that normally expected at this time of year. Moreover, schools in the surrounding communities were experiencing high absenteeism because of influenza-like disease. Brunswick Junior High School, for example, had closed on February 8, when 60 percent of the students were reported to have been ill.

Approximately 80 percent of the active duty military personnel stationed at the Brunswick Naval Air Station were immunized with military formula influenza vaccine in October, November, and December 1965.

During the month of February 720 military personnel reported at sick call, a number only slightly above that seen the previous three months. However, two hundred and sixty-two of them (36.4%) presented with influenza-like symptoms, a proportion greater than in November (14.8%), December (6.7%) or January (22.7%).

In an attempt to determine the extent of the outbreak at the Base, the available medical records of 492 of the 720 military personnel reporting at sick call during the period February 1-28 (68.3%) were reviewed. On the basis of presenting symptomatology (sore throat, rhinorrhea, nasal congestion, general malaise, fever, myalgia), and the clinical impressions of the examining physicians, 195 of the 492 (36.6%) were classified as having had an influenza-like respiratory illness. At the time of their initial visits, a temperature of 100° F. or more was present in 40 of the 195 (20.5%), 13 of whom were subsequently hospitalized.

A review of the influenza vaccination histories of all 492 patients revealed that one-third had not received influenza vaccine, a proportion found to be constant among all those reporting to sick call whether they had presented with an influenza-like illness or with non-respiratory complaints. Thus the illnesses in the vaccinated and unvaccinated personnel showed almost identical distributions by type of illness, i.e., respiratory with or without temperatures $\geq 100^{\circ}\text{F}$. and non-respiratory. (See Table 1.)

Table 1

VACCINATION HISTORIES
FROM A REVIEW OF 492 AVAILABLE RECORDS OF 720 SICK CALL VISITS
February 1-28, 1966

<u>Type of Illness</u>	<u>Vaccinated Personnel</u>		<u>Vaccinated Personnel</u>		<u>Total</u>
	<u>Number</u>	<u>Percentage</u>	<u>Number</u>	<u>Percentage</u>	
Respiratory					
\geq Max. Temp. - 100°F .	28	8	12	8	40
$<$ Max. Temp. - 100°F .	105	32	50	31	155
Non-Respiratory	199	60	98	61	297
Total	332	100%	160	100%	492

The peak of the outbreak, as based on the number of visits to military sick call for influenza-like illness, appeared to have occurred during the week ending February 26.

At the time of the epidemic investigation, between February 21 and 23, 23 patients reported to sick call with respiratory illnesses, 18 of whom had temperatures of 100°F . or more. Of the 18, only three had received influenza vaccine three months previously.

Acute serum specimens and throat washings for viral isolations were collected from all 23; convalescent sera were obtained from those 16 who were available 2-3 weeks later.

One virus isolated from throat washings has been identified as type B influenza. Laboratory studies on the paired serum specimens showed a four-fold diagnostic antibody increase to influenza B (by HI and/or CF) in 9 of the 16.

In summary, a small outbreak of a clinically mild influenza-like illness occurred at the Naval Air Station in Brunswick, Maine with peak incidence during the last week in February. Influenza virus, type B, was identified as the etiologic agent.

In this study on the basis of the available information, it was not possible to demonstrate effectiveness of the military vaccine. Those who had received vaccine reported to sick call with influenza-like symptoms (with or without temperatures of 100°F . or more) as frequently as those who had not received the vaccine.

(Reported by Captain R. J. Martin, M.C., Senior Medical Officer, Naval Air Station Hospital, Brunswick, Maine; Captain Jack Millar, Director, Preventive Medicine Division, Department of the Navy, Washington, D.C., and an epidemiological team from CDC.)

Washington State

On February 8, the Division of Epidemiology, Washington State Department of Health, became aware of an increase in absenteeism approaching 15 percent at a large junior high school located in Olympia. Since respiratory symptoms suggested that epidemic

influenza might be making its first clinical appearance in the State during the 1965-66 season, an immediate investigation was instituted by the State Health Department. It was thought that this outbreak might afford an excellent opportunity to test the efficacy of unpaired acute and convalescent sera in rapidly delineating the cause of a respiratory disease epidemic.

On February 9 and 10 a list of the 117 student absentees was obtained from school officials. Each student was then contacted by phone to determine a more precise cause for his absence; all absent with respiratory symptoms compatible with influenza were visited in their homes. In an attempt to isolate the etiologic agent, throat swabs were taken for virus culture from 21 individuals. In addition, acute sera was drawn on 20 acutely ill persons selected for study as presumptive cases of acute influenza on the basis of reporting four of the following seven symptoms: fever, chills, headache, cough, sore throat, myalgia, or ocular pain. The 20 acute sera thus obtained would be paired in two weeks with convalescent sera from the same patients in a further attempt to definitively identify the responsible agent.

On February 14 a second list representing those 40 students who were absent from school with a respiratory illness during the preceding three weeks was obtained from the school. From this group of 40, seven were arbitrarily selected as being presumptive cases of convalescent influenza in the same manner as mentioned above; that is, on the basis of reporting four of the seven upper respiratory symptoms.

The most frequently mentioned and most troublesome complaint, noted in 90 percent of acutely ill individuals, was sore throat. Although cough symptoms were just as frequently present, they were typically mild and of a non-productive nature. The symptoms elicited from the 21 acute cases are shown in Table 1 listed in order of decreasing frequency:

Table 1

SYMPTOMS BY FREQUENCY OF OCCURRENCE
IN 21 ACUTELY ILL PATIENTS SELECTED FOR STUDY

Symptoms	<u>Cases Reporting Symptom</u>	
	Number	Percentage of All Cases
Sore throat	19	90
Cough	19	90
Fever	17	81
Rhinorrhea	15	71
Headache	14	67
Myalgia	13	62
Ocular pain	10	48
Chills	10	48
Dizziness	9	43
Abdominal cramps	6	29
Nausea	1	5
Vomiting	1	5
Diarrhea	0	0

Duration of illness among the 21 acute cases ranged from 1 to 8 days with a mean of 4.2.

A comparable listing of symptoms for the seven convalescent persons is represented in Table 2. Here it may be seen that a history of sore throat was less frequently elicited, while cough, fever, rhinorrhea and headache were uniformly present.

Table 2

SYMPTOMS BY FREQUENCY OF OCCURRENCE
IN 7 CONVALESCENT PATIENTS SELECTED FOR STUDY

Symptoms	Cases Reporting Symptom	
	Number	Percentage of All Cases
Cough	7	100
Fever	7	100
Rhinorrhea	7	100
Headache	7	100
Sore throat	5	71
Myalgia	5	71
Ocular pain	4	57
Chills	3	43
Dizziness	3	43
Diarrhea	2	29
Abdominal cramps	0	0
Nausea	0	0
Vomiting	0	0

Among the seven convalescent patients the duration of illness ranged from one to seven days with a mean of 4.4.

Average daily absenteeism in the junior high school had averaged 45 students (5.4%) per day over the preceding few months. As may be seen in Figure 7, an epidemic curve showing number of absentees by day, absenteeism rose to 10 percent during the first week of February and reached a peak of over 20 percent by the middle of the month. The dates of serum specimens obtained are depicted in Figure 7 by arrows.

Table 3 shows the laboratory results on the seven unpaired convalescent sera utilizing complement fixation antibody tests to influenza A, influenza B and adenovirus.

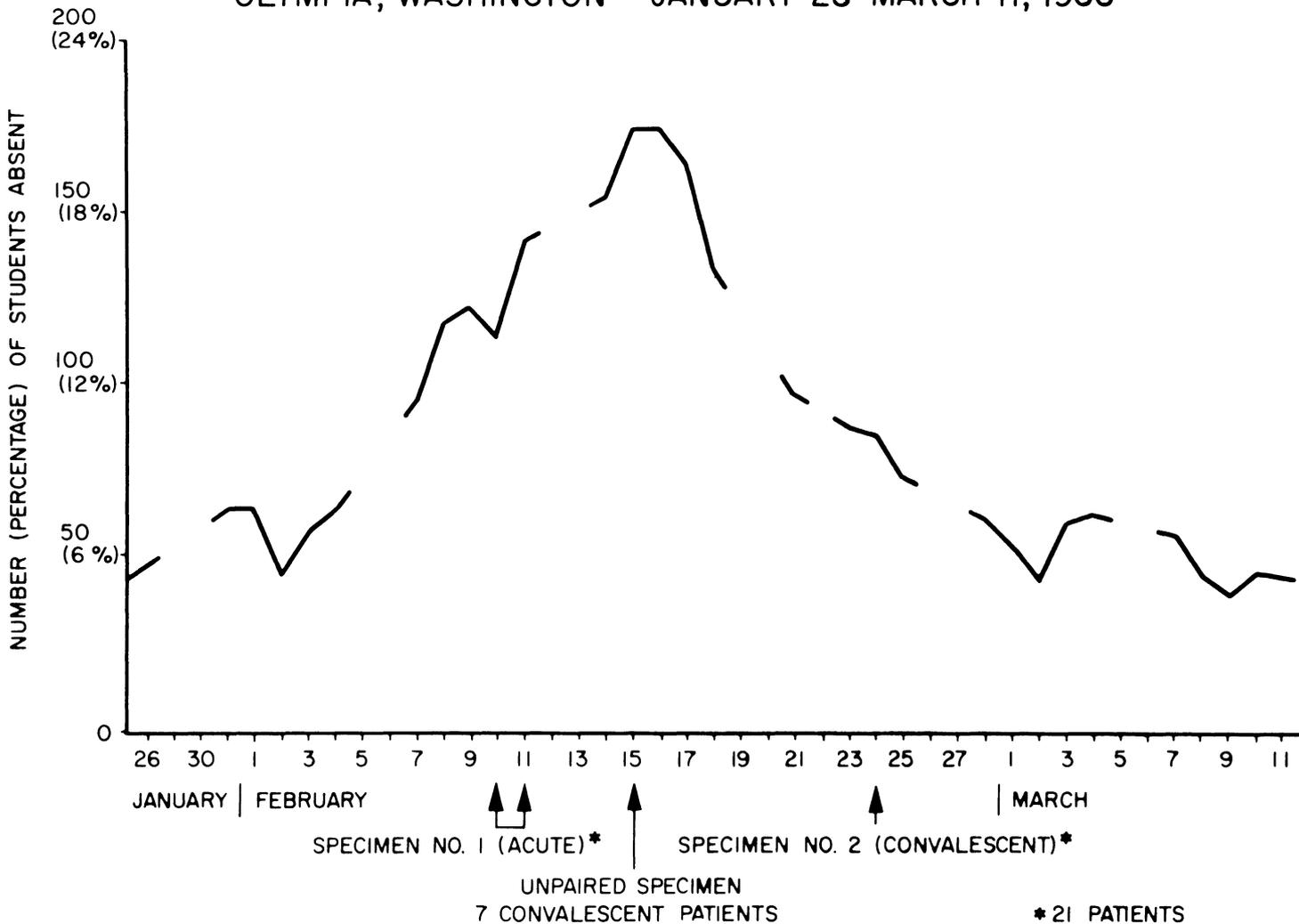
Table 3

SINGLE SPECIMEN ANTIBODY TITERS, IMMUNIZATION STATUS AND
INTERVAL BETWEEN ONSET OF ILLNESS AND DATE OF SPECIMEN
IN 7 CONVALESCENT PATIENTS SELECTED FOR STUDY

Student	C.F. Antibody Titer			Influenza Immunization	Interval Between Date of Symptom Onset and Date of Specimen
	Influ B	Influ A	Adeno		
KS	1:1024	0	0	Never	15 days
MM	1:1024	1:8	1:32	Never	14 "
CD	1:256	0	0	3 years ago	14 "
JK	1:256	1:16	1:32	3 years ago	21 "
GM	1:64	0	1:32	Never	15 "
CF	0	0	1:8	Unknown	19 "
DW	0	0	1:32	Unknown	22 "

Complement fixation tests done on the 20 sera drawn on acutely ill persons selected for study revealed low titers for the three respiratory antigens studied. (Table 4.)

Figure 7
INFLUENZA B
ABSENTEEISM BY DAY* - WASHINGTON JUNIOR HIGH SCHOOL
OLYMPIA, WASHINGTON - JANUARY 28-MARCH 11, 1966



* BREAKS IN GRAPH REPRESENTS WEEKENDS OR HOLIDAYS

Table 4

SINGLE SPECIMEN C.F. ANTIBODY TITERS AGAINST THREE ANTIGENS
IN 20 ACUTELY ILL PATIENTS SELECTED FOR STUDY

	<u>C.F. Antibody Titers</u>			
	1:128	1:64	1:32	_ 1:16
Influenza B	0	0	3	17
Influenza A	1	0	1	18
Adenovirus	0	1	6	13

Comparing the 20 single specimen acute sera with the seven single specimen unpaired convalescent sera, it may be seen that statistically significant differences exist between the titers to influenza B. (Table 5)

Table 5

COMPARISON OF ACUTELY ILL AND CONVALESCENT PATIENTS
BY C.F. ANTIBODY TITERS AGAINST THREE ANTIGENS

Antigen and Antibody Titer	Acutely Ill Patients		Convalescent Patients	
	Number	Percentage	Number	Percentage
Influenza B				
≤ 1:16	17	85	2	29
≥ 1:32	3	15	5	71
Influenza A				
≤ 1:16	18	90	7	100
≥ 1:32	2	10	0	0
Adenovirus				
≤ 1:16	13	65	3	43
≥ 1:32	7	35	4	57

The differences in the unpaired acute and convalescent sera titers to influenza A and adenovirus may be chance alone. These laboratory results indicated that the epidemic could be tentatively attributed to influenza B.

Confirmatory evidence that the outbreak was caused by influenza B virus was subsequently obtained. Four of the throat swab specimens obtained have demonstrated the presence of type B influenza by complement fixation studies. Hemagglutination inhibition tests showed reaction with B/Taiwan antisera at a titer of 1:64 and B/Great Lakes at 1:32. Paired sera were obtained on the 20 acutely ill patients two weeks later. Diagnostic four-fold or greater rises in antibody titer against influenza B was documented in 14 of the 20 (70%); over half of the patients demonstrated absolute titer values of 1:1024 in the convalescent specimens. No four-fold titer rises were observed to either type A influenza or adenovirus.

A re-examination of the most prominent symptoms in the 14 serologically proven cases of influenza B revealed that the symptoms they presented were essentially the same as those mentioned by the total group of 21 acutely ill patients selected for

study. However the duration of illness of the 14 serologically confirmed cases was somewhat greater than that reported for all 21 acutely ill cases.

In summary, an outbreak of type B influenza at a junior high school in Olympia, Washington in early February 1966 was utilized to study the value of acute convalescent unpaired sera in the early diagnosis of influenza outbreaks. The results of the study tend to indicate that carefully selected unpaired convalescent sera can be of use in demonstrating the etiology of an outbreak of acute febrile respiratory disease.

(Reported by Dr. Ernest A. Ager, Chief, Division of Epidemiology, State Department of Health, Olympia, Washington; and an EIS Officer assigned to the State Department of Health, Washington.)



Key to all disease surveillance activities are those in each State who serve the function as State epidemiologists. Responsible for the collection, interpretation and transmission of data and epidemiological information from their individual States, the State epidemiologists perform a most vital role. Their major contributions to the evolution of this report are gratefully acknowledged.

STATE	NAME
Alabama	Dr. W. H. Y. Smith
Alaska	Dr. Thomas R. McGowan
Arizona	Dr. Philip M. Hotchkiss
Arkansas	Dr. Wm. L. Bunch, Jr.
California	Dr. Philip K. Condit
Colorado	Dr. C. S. Mollohan
Connecticut	Dr. James C. Hart
Delaware	Dr. Floyd I. Hudson
D. C.	Dr. William E. Long
Florida	Dr. E. Charlton Prather
Georgia	Dr. W. J. Murphy
Hawaii	Dr. Ralph B. Berry
Idaho	Dr. John A. Mather
Illinois	Dr. Norman J. Rose
Indiana	Dr. A. L. Marshall, Jr.
Iowa	Dr. Ralph H. Heeren
Kansas	Dr. Don E. Wilcox
Kentucky	Dr. Calixto Hernandez
Louisiana	Dr. John A. Trautman
Maine	Dr. Dean Fisher
Maryland	Dr. John H. Janney
Massachusetts	Dr. Nicholas J. Fiumara
Michigan	Dr. George H. Agate
Minnesota	Dr. D. S. Fleming
Mississippi	Dr. Durward L. Blakey
Missouri	Dr. E. A. Belden
Montana	Dr. Mary E. Soules
Nebraska	Dr. E. A. Rogers
Nevada	Dr. B. A. Winne
New Hampshire	Dr. William Prince
New Jersey	Dr. W. J. Dougherty
New Mexico	Dr. Kathleen Hawkins (Acting)
New York State	Dr. Julia L. Freitag
New York City	Dr. Harold T. Fuerst
North Carolina	Dr. Martin P. Hines
North Dakota	Mr. Kenneth Mosser
Ohio	Dr. Calvin B. Spencer
Oklahoma	Dr. Robert Leroy Carpenter
Oregon	Dr. Edward L. Goldblatt
Pennsylvania	Dr. W. D. Schrack, Jr.
Puerto Rico	Dr. Rafael A. Timothee
Rhode Island	Dr. James E. Bowes
South Carolina	Dr. G. E. McDaniel
South Dakota	Dr. G. J. Van Heuvelen
Tennessee	Dr. C. B. Tucker
Texas	Dr. Van C. Tipton
Utah	Dr. Robert Sherwood
Vermont	Dr. Linus J. Leavens
Virginia	Dr. James B. Kenley
Washington	Dr. E. A. Ager
West Virginia	
Wisconsin	Dr. Josef Preizler
Wyoming	Dr. Robert Alberts