

COMMUNICABLE DISEASE CENTER

INFLUENZA

SURVEILLANCE

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AND INFLUENZA DEATHS



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

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.

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I. SUMMARY

Since publication of the last Influenza Surveillance Report (No. 78, February 19, 1964), outbreaks first observed in northwestern Washington during late January have spread widely, involving communities throughout the states of Washington and Oregon. Serologic studies have indicated influenza A₂ virus as the etiologic agent in this epidemic. The state of California, while not experiencing extensive, community-wide outbreaks such as were seen in Washington and Oregon, has nevertheless had fairly wide dissemination of influenza A₂ virus among its residents -- as evidenced by changes in the following surveillance indices: 1) reports of respiratory illness, 2) school absenteeism, 3) weekly total of serologically confirmed A₂ influenza cases, 4) pneumonia-influenza mortality, and 5) the occurrence of two institutional outbreaks of laboratory confirmed influenza A₂ in widely separated parts of the state.

The recent influenza B epidemic in Japan (see Influenza Surveillance Report No. 78) which began in mid-January on the island of Kyushu, later spread to the main island of Honshu, and affected the Tokyo area in mid-February. Another influenza B epidemic was observed in the city of Singapore, Malaya, during January.

Outbreaks of influenza-like disease were observed in several parts of Europe during the months of February and March -- most of them attributable to Type A₂ virus. The affected areas included: Czechoslovakia, Greece, the United Kingdom, and Yugoslavia.

A report on the recent influenza A₂ epidemic in Taiwan is included in this issue. A discussion of antigenic relationships of recently isolated influenza viruses is given in the laboratory section of this issue.

II. EPIDEMIC REPORTS

Washington

The influenza epidemic which began during late January in northwestern Washington has spread in the ensuing weeks to involve communities throughout the state. (See Figure 1.)

By mid-February, outbreaks had already spread from their original northwestern focus and were being reported from many areas in central Washington -- notably from the adjacent counties of Chelan and Douglas, where a distinct increase in school absenteeism due to respiratory disease was observed. New outbreaks continued to be reported from the western part of the state at that time, including the cities of Seattle, Tacoma, and Port Angeles, as well as many communities in Grays Harbor and Kitsap Counties. While most epidemic activity was concentrated in the western and central regions at that time, isolated outbreaks were already being observed in some eastern communities. The town of Tekoa (population 1189) in Whitman County, situated just a few miles from the Idaho State line witnessed an explosive outbreak of influenza-like disease, with school absenteeism reaching 22-50% during the week ended February 15.

During late February, additional outbreaks were noted in the central part of the state, and clearcut evidence of significant eastward spread was observed. During the week ended February 29, six counties reported outbreaks for the first time. These were all situated in southern Washington and extended from the southwestern corner of the state, eastward to the Idaho border. By this time, earlier outbreaks noted in the northwestern corner of the state appeared to be subsiding.

By early and mid-March, almost all affected communities in western Washington were returning toward normal, and the epidemic had become concentrated in rural areas in the eastern half of the state. Among the areas most severely affected at that time was Spokane County, on the Idaho border, where increased school absenteeism caused the closing of 28 schools during the week ended March 14. Absenteeism returned toward normal relatively quickly however, and all county schools had reopened by the following week. Outbreaks in eastern Washington reached their peak in mid-March and were clearly waning by the end of the month.

Influenza A₂ virus was identified as the presumptive etiologic agent in the Washington epidemic in early February. (See Influenza Surveillance Report No. 78, February 19, 1964.) These preliminary findings were confirmed by means of conventional serologic studies performed at the Respirovirus Laboratory, C.D.C., in early March. Paired sera from eight typical cases were studied, and all eight showed significant (4-fold or greater) titer rises of H.I. antibody to influenza A₂ antigens. Additional serologic confirmations were obtained in the Seattle-King County area.

FIGURE I.

COUNTIES REPORTING OUTBREAKS OF INFLUENZA - LIKE DISEASE

Washington and Oregon, January 27 - March 28, 1964

I

Jan. 27 - Feb. 8



II

Jan. 27 - Feb. 22



III

Jan. 27 - March 14



IV

Jan. 27 - March 28



(Reported by Ernest A. Ager, M.D., Chief, Division of Epidemiology, State Department of Health, Olympia, Washington.)

Oregon

Outbreaks of influenza-like disease were first recognized in northwestern Oregon, near the Washington border, during mid-February. Columbia, Clackamas, Multnomah, Washington, and Yamhill Counties were among those from which earliest reports were received. The epidemic spread southward and somewhat eastward during late February and early March with notable outbreaks reported from Curry and Klamath Counties, among others. By mid-March, weekly case reports for the state were approaching 4,000, and the epidemic had clearly spread from its initial focus to involve extensive areas in the central and eastern parts of the state. The epidemic reached its peak later in the month, with case reports totalling 4,224 for the week ended March 21. This figure fell to 3,515 for the week ended March 28, and by early April the epidemic showed clear signs of waning in many affected areas. Serologic studies performed at the Oregon State Laboratories implicated influenza A₂ as the etiologic agent in these outbreaks. A total of 14 serologically confirmed cases have been identified to date.

(Reported by Dr. Grant Skinner, Director, Epidemiology Section, State Board of Health, Portland, Oregon.)

California

While the sharp, community-wide outbreaks which have characterized recent epidemics in Washington and Oregon have not been observed in California during the past winter, influenza A₂ virus was fairly widely disseminated in the state during the months of February and March, as evidenced by changes in the following surveillance indices: 1) reports of respiratory illness, 2) school absenteeism, 3) weekly total of serologically confirmed cases, 4) pneumonia-influenza mortality, and 5) occurrence of institutional outbreaks of laboratory confirmed influenza A₂.

Scattered reports of influenza-like disease were received from several areas including the cities of Los Angeles and San Francisco during late February and early March. Overall school absenteeism increased slightly in these two cities during that period. The disease was described as somewhat milder than classical influenza, and of slightly shorter duration. During early and mid-March, San Joaquin and San Mateo Counties reported a definite increase in the incidence of the flu-like illness, accompanied by school absenteeism reaching 20-25% in some schools.

In the latter county, high school students appeared to be more severely affected than those of elementary school age. Similar reports were received from Butte and Merced Counties, where industrial absenteeism was also somewhat elevated, as well as from Humboldt County.

Overall absenteeism in nine school jurisdictions in which continuous surveillance is maintained rose moderately in early March and reached a mean level of about 10 percent for the week ended March 14.

The number of serologically confirmed cases of influenza A₂ infection rose sharply in late February and by March 21, had reached a total of 34 cases, representing 10 counties.

Pneumonia-influenza deaths in eight reporting cities first exceeded the expected level in mid-February and reached a peak during the week ended March 21. Reported deaths during the past six weeks compared with the expected incidence (mean for the period 1958-63) are given in Table 1.

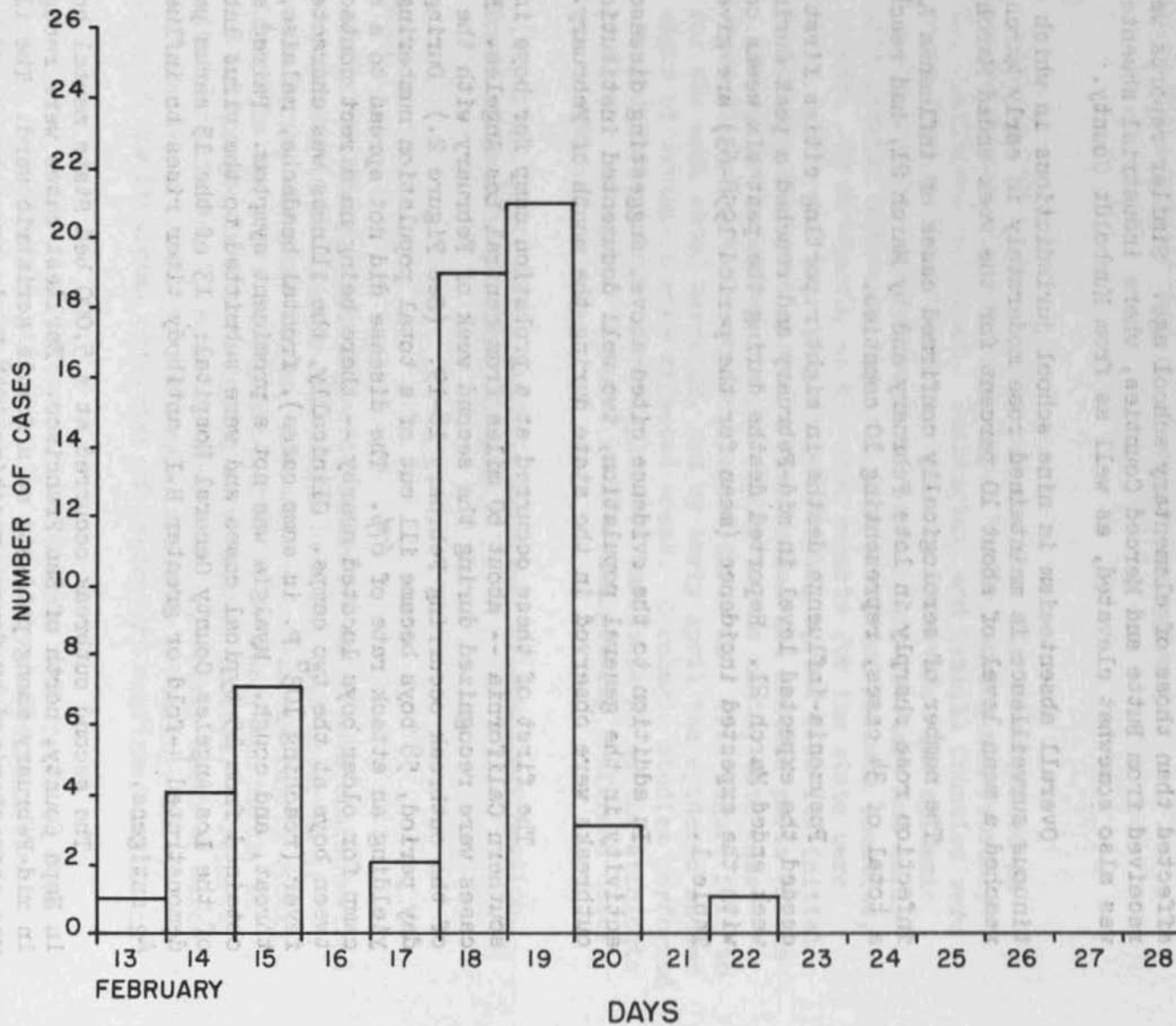
In addition to the evidence cited above, suggesting disease activity in the general population, two well documented institutional outbreaks were observed in the state during the month of February.

The first of these occurred at a probation camp for boys in southern California -- about 60 miles from central Los Angeles. Earliest cases were recognized during the second week of February with the peak of the outbreak occurring February 18-19. (See Figure 2.) During a 10-day period, 58 boys became ill out of a total population numbering 86, yielding an attack rate of 67%. The disease did not spread to a similar camp for older boys located nearby -- there being no direct contact between boys at the two camps. Clinically, the illness was characterized by fever (reaching 103° F. in some cases), frontal headache, malaise, sore throat, and cough. Myalgia was not a prominent symptom. Paired sera were obtained from 15 typical cases and were submitted to the virus laboratory of the Los Angeles County General Hospital: 13 of the 15 serum pairs demonstrated 4-fold or greater H.I. antibody titer rises to influenza A₂ antigens.

The second outbreak occurred at a 5,000 bed State mental hospital in Napa County, north of San Francisco. Earliest cases were recognized in mid-February among patients on a female geriatric ward. The illness was characterized by fever, reaching 104° F. in some cases, and non-productive cough. Headache, myalgia, and malaise were not part of the clinical picture. The disease spread fairly rapidly in the ensuing weeks,

Figure 2

INFLUENZA CASES BY DATE OF ONSET
 CAMP J.M.-LOS ANGELES COUNTY, CALIFORNIA



producing attack rates approaching 100% on some wards. Serologic studies performed at the State laboratories in Berkeley revealed 4-fold or greater titer rises to influenza A₂ antigen in 9 of 15 serum pairs submitted.

(Reported by Philip K. Condit, M.D., Chief, Bureau of Communicable Disease, State Department of Health, Berkeley, California.)

Table 1

Weekly Pneumonia and Influenza Deaths in Eight California Cities

<u>Week Ended</u>	<u>Observed</u>	<u>Expected</u>	<u>Percent Excess</u>
March 7	58	35	65
March 14	67	34	96
March 21	76	34	124
March 28	74	34	118
April 4	62	33	88
April 11	<u>57</u>	<u>30</u>	<u>90</u>
6 Week Total	394	200	97
6 Week Mean	66	33	100

III. INTERNATIONAL REPORT

Asia

Japan: The recent influenza B epidemic, first noted on the island of Kyushu in mid-January (see Influenza Surveillance Report No. 78) later spread to the main island of Honshu and affected the Tokyo area in mid-February. Attack rates were highest in school age children and school absenteeism was utilized for surveillance. Several hundred schools were closed in the course of the epidemic. The illness was relatively mild clinically, with fever lasting 2-3 days and upper respiratory symptoms the principal features.

Serologic studies have been performed in many prefectures and all implicated influenza B as the etiologic agent. Type B virus has been isolated in eggs from specimens obtained on both Kyushu and Honshu. The virus is not closely related to strains isolated from Japan in 1956 or in Taiwan in 1962, but is similar to Type B virus isolated in Japan in 1961. It is also closely related to the B/Great Lakes/54 strain.

(Reported by Dr. Hideo Fukumi, Chief, Japanese Influenza Center, Tokyo; Dr. F. Nishikawa, Japanese National Institutes of Health; and Dr. N. Yuzawa, Chief, Communicable Disease Control Section, Public Health Bureau, Ministry of Health and Welfare.)

Maylasia: Several influenza B isolates were obtained from cases seen during an outbreak of influenza-like disease which occurred in the city of Singapore during January. These isolates were closely related to the B/Japan/56 and B/Johannesburg/58 strains, and also, to a lesser extent, resembled the B/Taiwan/62 strain.

Europe

Czechoslovakia: An increase in the incidence of influenza-like disease was noted in late February and early March. School-age children and adolescents appeared to be most severely affected. Influenza A₂ virus was recovered from outbreaks in two adolescent populations and serologically confirmed cases were reported from several communities.

Greece: An epidemic of influenza-like disease was observed in the city of Athens and the neighboring port of Piraeus during February and early March. An overall attack rate of approximately 20% was estimated for the two cities.

United Kingdom: The Central Public Health Laboratory, London, has reported that influenza A₂ isolates have been recovered from several cases seen during late January and February in the Midlands and west of England. Eight strains have been characterized thus far, and all have been found similar to those isolated in London in 1963 and to the A₂/Netherlands/63 strain. In addition, two strains of influenza B virus have been isolated in Cambridge. One, recovered from a school outbreak, is identical to B/Johannesburg/58. The other, from an unrelated case, is being investigated further.

Yugoslavia: Morbidity due to influenza-like disease rose sharply in early March, reached a peak during the middle of that month, and has been declining slowly since then. In Belgrade, approximately 27,000 cases were being reported weekly at the height of the epidemic. It has been estimated that 70% of cases occurred in adults (over 20 years of age).

An influenza A₂ virus was implicated as the etiologic agent in this epidemic. The isolate appeared similar to 1957 strains.

(From the WHO Weekly Epidemiological Record, Geneva, 39, 1964.)

IV. LABORATORY REPORT

Roslyn Q. Robinson, Ph.D.
Chief, Respirovirus Unit and
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Antigenic Relationships of Recently Isolated Influenza Viruses

Influenza viruses isolated from epidemics in Taiwan (Type A₂) and Japan (Type B), have been used in hemagglutination inhibition tests for determination of antigenic relationships to viruses isolated in earlier years. Results of these tests are summarized in Tables 1 and 2.

It may be seen from Table 1 that a currently prevalent A₂ virus (A₂/Taiwan/1/64) differs somewhat from the prototype strain, A₂/Japan/305/57 as well as the A₂/Japan/170/62 virus. A similar departure from the antigenic constitution of the prototype strain was observed among A₂ influenza viruses isolated during the epidemic in the United States in the early months of 1963. It appears that there has been a gradual antigenic change in A₂ viruses since the original isolation in 1957.

Type B influenza viruses, recently prevalent in Japan, appear to be quite similar to the B/Great Lakes/1739/54 prototype strain (Table 2). The B/Amakusa/1/64 virus is also related to the B/Maryland/1/59 virus, although the two are not identical. It is quite clear that B/Amakusa/1/64 is unrelated to the B/Taiwan/2/62 virus. This latter virus was isolated from a localized outbreak in Taiwan in 1962 and was subsequently found to be antigenically distinct from all type B influenza viruses isolated in earlier years. (See Influenza Surveillance Report No. 75, March 8, 1963, page 19.) As yet, there is no evidence that the variant strain, B/Taiwan/2/62, has spread from the initial localized outbreak.

Confirmation of Influenza Outbreak in Washington State

A rapid presumptive characterization of an influenza outbreak in Washington State using unpaired acute and convalescent sera was reported in Influenza Surveillance Report No. 78. The finding that influenza A, presumably subtype A₂, was responsible, has since been confirmed.

Influenza A₂ viruses have been isolated from throat swabs collected during the outbreak and paired serum specimens have yielded diagnostic antibody rises to A₂/Jap/305/57 as well as the influenza viruses isolated.

Table 1

Antigenic Relationships of A₂ Influenza Viruses

Chicken Antiserum	Antigens		
	Japan/305/57	Japan/170/62	Taiwan/1/64
Japan/305/57	<u>160</u>	160	80
Japan/170/62	40	<u>160</u>	40
Taiwan/1/64	20	40	<u>480</u>

Table 2

Antigenic Relationships of Type B Influenza Viruses

Chicken Antiserum	Antigens				
	B/Lee	B/GL	B/MD	B/Taiwan	B/Amakusa
B/Lee/40	<u>160</u>	20	10	10	10
B/GL/1739/54	10	<u>160</u>	40	10	80
B/MD/1/59	0	40	<u>640</u>	10	40
B/Taiwan/2/62	0	10	0	<u>80</u>	0
B/Amakusa/1/64	10	80	40	10	<u>160</u>

V. SPECIAL REPORT: Influenza in Taipei, Taiwan, 1964.

In late January the occurrence of an outbreak of upper respiratory disease was reported to the Communicable Disease Center by Dr. Howard Jenkin, virologist for the Naval Medical Research Unit (NAMRU) # 2 in Taipei, Taiwan (Formosa). The etiologic agent had not been identified, but influenza was suspected. Of particular concern was the possibility that this might represent an outbreak caused by the Taiwan strain of Type B influenza, an antigenically distinct Type B strain which was first isolated at NAMRU-2 in the spring of 1962.

With the concurrence of Captain Jack Millar, Chief, Preventive Medicine Division, U. S. Navy, Dr. Robert J. Warren, CDC Medical Epidemiologist assigned to Dr. John Enders' Laboratory, Boston, left for Taipei on January 31 to assist Dr. Jenkin in epidemiologic investigations of the outbreak. In addition to Drs. Jenkin and Warren, Captain Phillip V. Engler of the Fifth Epidemiological Flight (PACAF) in the Philippines participated in the study.

The abstract which follows is based on material kindly provided by these investigators.

Methods

Surveillance of Respiratory Disease on Taiwan: Available information regarding the occurrence and extent of outbreaks of illness in the Chinese civilian and military population, as well as the U. S. military and their dependents, was obtained through Dr. L. P. Chow, Chief, Technical Service, Taiwan Provincial Health Department; Col. Hsu, Deputy Surgeon General of Taiwan; and medical officers at each of the U. S. armed forces facilities on the island.

Pneumonia Deaths: Data for pneumonia deaths were tabulated from death certificates from the city of Taipei. Unfortunately, similar data for the entire island were not available at the time of this investigation.

Hospital Outpatient Visits: Clinicians at several hospitals were interviewed to determine whether or not the number of patients with a flu-like syndrome had increased. In general, information regarding the specific number of visits per day was not available.

Population Surveys Regarding Incidence of Influenza-like Disease: One hundred and seventy of approximately 750 American families living in the Tien Mou section of Taipei were randomly selected and interviewed by telephone. Questions asked included ages of all members of the household and the occurrence of "cold or flu-like illness" during the month

of January. Similar questions about illness between January 1 and February 7 were asked of Chinese residents of Su-Wun Lee, Ta Tung County, Taipei, in a door-to-door survey conducted by Dr. T. Y. Lee, Director of the Taipei Municipal Bureau of Health.

School Absenteeism: Information on daily absence was collected from the Taipei American School and the Dominican School. Dr. Lee provided rates on absenteeism in the Chinese schools in Taipei.

Employee Absenteeism: With the exception of absences among employees at NAMRU-2, data of this nature were not readily available.

Combined Epidemiologic and Serologic Studies: Since attack rates among employees of NAMRU-2 and high school students of the Taipei American School were particularly high, these groups were selected for more detailed epidemiologic questioning. Serum specimens were collected shortly after the peak of the epidemic and again 2-3 weeks later for determination of influenza antibody levels.

Viral and Serologic Studies: Throat and nasopharyngeal swabs for viral isolation attempts in eggs and tissue culture were obtained from more than 100 Chinese patients of hospital outpatient departments in Taipei. Those with most recent onset of clinical symptoms suggestive of influenza were selected for study. Acute phase sera were collected from all and convalescent sera from about one-fourth. Many patients refused to participate because of religious taboos against giving blood.

Results: The first evidence of an influenza epidemic on Taiwan was noted at NAMRU-2. Many of the American employees there were absent with a febrile upper respiratory illness in early January. Individual questioning revealed that 15 of 37 (40.5%) had been ill. Absences among Chinese workers were much less common than among Americans, but further investigation revealed that the Chinese preferred, if possible, to go to work when they were ill rather than to remain in unheated homes. However, in individual interviews, only 41 of 180 Chinese (22.8%) reported any respiratory illness during January. The peak occurrence in each group was during the second week of January. A secondary peak in the Chinese occurred near the end of January, but no additional cases were noted in Americans after the middle of the month.

In an attempt to determine the extent and severity of the outbreak, both civilian and military health authorities throughout Taiwan were contacted. There was no evidence of spread of the epidemic outside of the Taipei area. However, an increase in the number of visits to the out-patient departments of hospitals in Taipei occurred during the second week of January. This increase persisted until late in the month and was due to an influx of patients with relatively mild upper

respiratory illness. Complications were rare, and the number of recorded pneumonia deaths per month showed no significant increase over the previous year's experience. (See Table 1.)

More concise data on the severity of the epidemic were obtained from surveys carried out in American and Chinese populations. Age specific attack rates are presented in Tables 2 and 3. Although there is little difference in the incidence of illness for children of pre-school age in the two surveys, attack rates for older children and adults are considerably higher in Americans than in Chinese. It should be emphasized that the two surveys were conducted at different times, in different people, and in different cultural groups. However, the results seem to substantiate the trends noted among workers at NAMRU-2.

An increased incidence of upper respiratory illness during the last week of January was noted in the American Survey, but perhaps a tendency for better recall for recent events could contribute to this trend. Similar information was not collected for the Chinese.

Further information on the effects of age and national background were obtained from school absenteeism records. Daily absence from 875 elementary, 575 intermediate, and 530 high school students of the Taipei American School are illustrated in Figures 1-3. Only slight increases in absenteeism are apparent except for the high school where a sharply defined epidemic occurred.

A questionnaire survey answered by 449 of the high school students revealed that approximately 60 percent of the students had been ill between Christmas vacation and February 10, the date of the questionnaire. The peak of the epidemic, by date of onset, was January 25, with a secondary peak on January 28 (Figure 4).

Absences at the Dominican School, a Roman Catholic School for both American and Chinese, failed to reveal excessive absenteeism except in the kindergarten. This could be accounted for by the occurrence of mumps in 38 of 68 (56 percent) of these children during January.

The rates of absenteeism for the first six grades of all Chinese schools in Taipei were 0.4 percent for November and 0.5 percent for December. In January rates remained low (0.9 percent) but were approximately twice those for either of the preceding two months.

International vaccination certificates of all Americans at NAMRU-2 and 268 students of the Taipei American High School were reviewed in an attempt to ascertain the effectiveness of influenza

immunization. Unfortunately, only a few had received vaccine since April 1963 and a meaningful evaluation was impossible.

Laboratory: The results of hemagglutination inhibition tests on paired acute and convalescent sera from patients of the Taipei Provincial Children's Hospital clearly indicated that Type A influenza (presumably A₂) was the predominant etiologic agent. A fourfold or greater increase in titer was found in 15 of 23 tested. The convalescent titer was 10²₄ or greater in nine. A significant change in Type B (Taiwan 2/62) titer did not occur in any of these children.

Sera were also tested from 68 employees of NAMRU-2 who were bled in mid-January and early February without respect to a history of illness. Seventeen (25 percent) had a fourfold increase and 7 (10 percent) a fourfold decrease in titer to A₂ (Japan/170/62) virus. In addition, four patients manifested a significant increase in titers to Type B (Taiwan/2/62) influenza and seven a decrease. Five of the 11 patients with changing titers for Type B also were among those with fourfold differences for Type A₂.

Aliquots of sera and two isolates were submitted from NAMRU-2 to Dr. Roslyn Q. Robinson, Chief, Respiratory Virus Unit, Laboratory Branch, CDC. Type A₂ was implicated by the results from both serologic and viral studies. (See Laboratory Report.)

CONCLUSIONS AND SUMMARY

An outbreak of influenza occurred in Taipei in January and February 1964. Type A₂ appears to have been the predominant etiologic agent. The epidemic was not explosive and appeared to be confined to Taipei. Data from surveys of housing areas, NAMRU-2 employees and school absenteeism all indicated that attack rates were higher in Americans than Chinese except in preschool children. Sharp outbreaks were experienced at NAMRU-2 and the Taipei American High School. Neither group had received influenza immunizations during the preceding six months.

Table 1

Pneumonia Mortality Rate Versus Total Mortality
 During the Winter Months
 From November 1962 - January 1964 in Taipei, Taiwan

<u>Date</u>	<u>Pneumonia Mortality</u>				<u>Total Mortality</u>	<u>Pneumonia / Total Mortality / Mortality</u>
	<u>Broncho</u>	<u>Lobar</u>	<u>Atypical</u>	<u>Total</u>		
November 1962	7	4	2	13	378	0.034
December 1962	13	2	7	22	339	0.064
January 1963	7	16	7	30	414	0.074
February 1963	16	6	8	30	425	0.070
November 1963	11	2	5	18	319	0.056
December 1963	11	1	14	26	336	0.077
January 1964	16	8	8	32	368	0.086

Table 2

Incidence of "Cold or Flu-like Illness"
Tien Mou Section, Taipei, January, 1964
By Age Group
(American Families)

<u>Age (Years)</u>	<u>No. Questioned</u>	<u>No. Ill</u>	<u>Percent Ill</u>
0-4	101	36	36
5-9	112	52	46
10-14	91	32	35
15-19	55	27	49
20 and over	343	107	31
Unknown	<u>21</u>	<u>8</u>	<u>38</u>
Total	686	262	38

Table 3

Incidence of "Cold or Flu-like" Illness in Su-Wun Lee,
Ta Tung County, Taipei, From Jan. 1 to Feb. 7, 1964
(Chinese Families)

<u>Age Group</u>	<u>No. Questioned</u>	<u>No. Ill</u>	<u>Percent Ill</u>
Preschool children	290	145	50.0
Older Children and Adults	<u>1099</u>	<u>73</u>	<u>6.6</u>
Total	1389	218	15.7

Figure 1

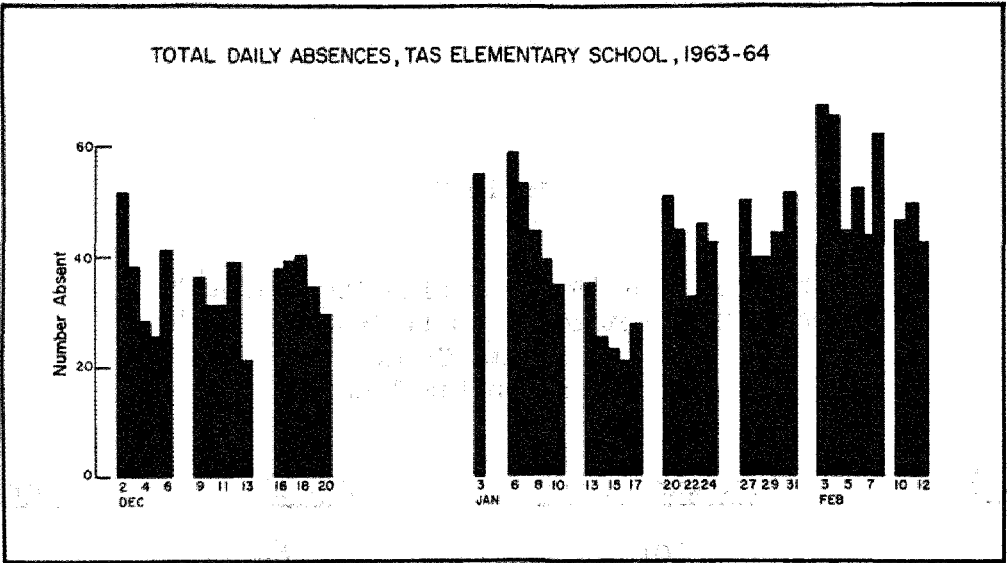


Figure 2

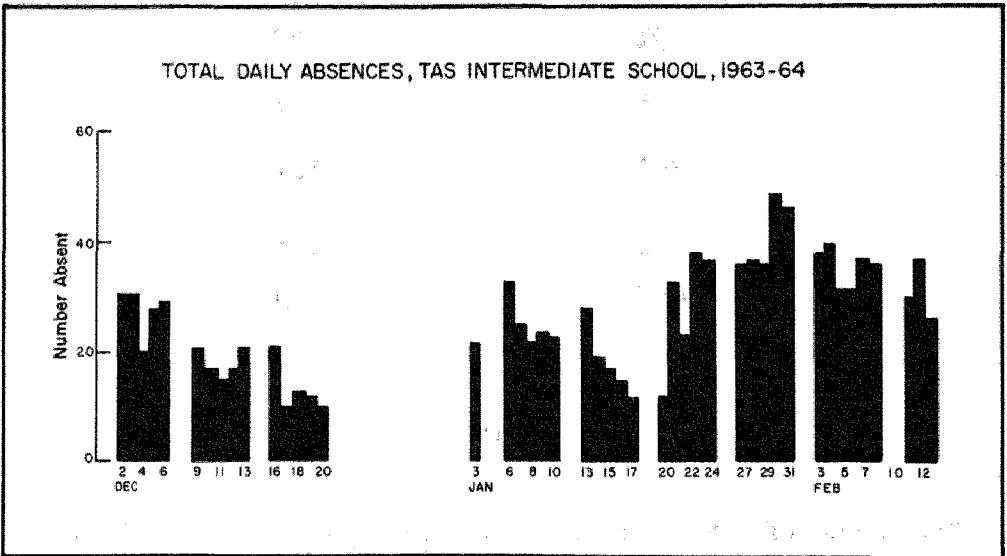


Figure 3

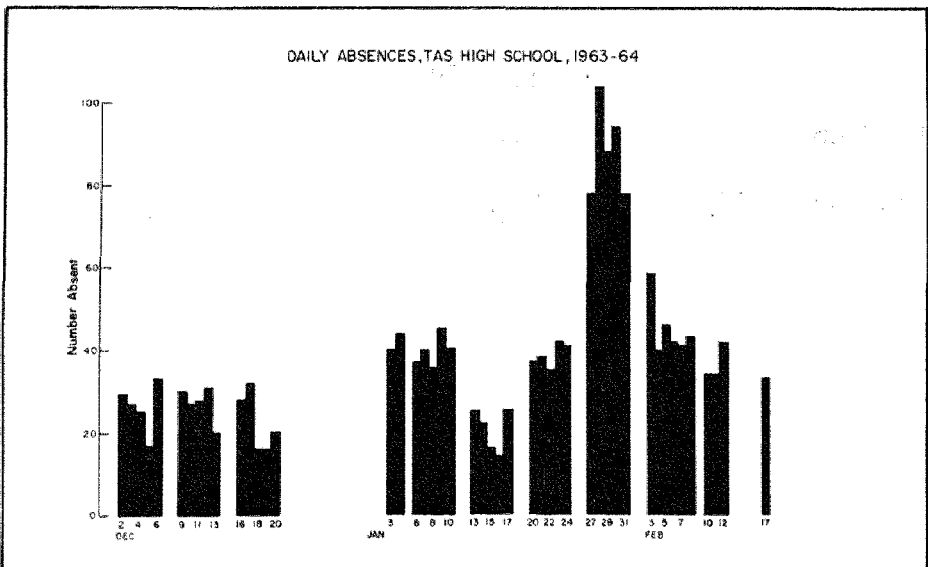
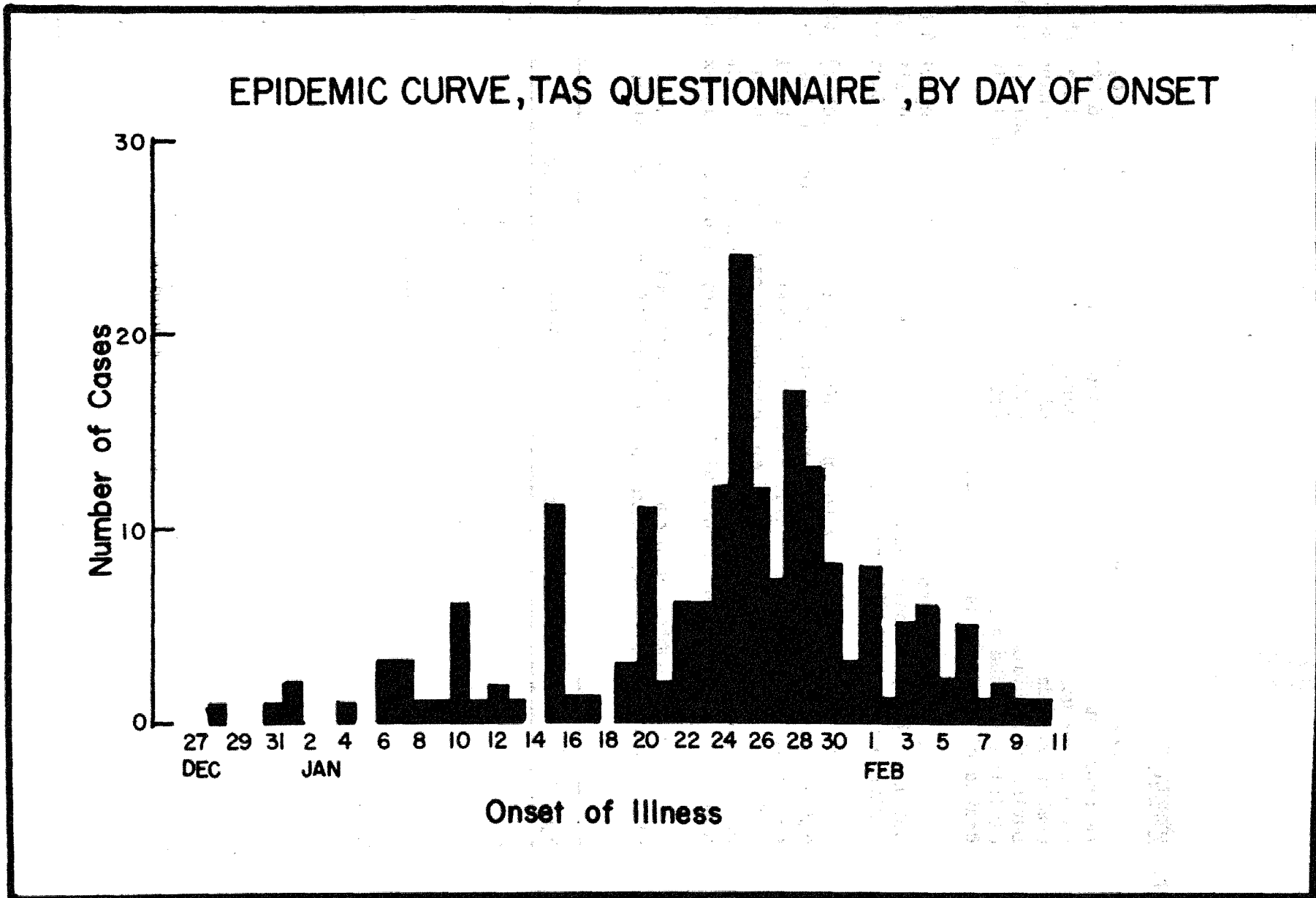


Figure 4



VI. WEEKLY PNEUMONIA AND INFLUENZA DEATHS

For the past 10-week period, February 8 through April 18, the reported numbers of pneumonia-influenza deaths for the United States as a whole were within expected ranges, but in the Pacific States the number exceeded the epidemic threshold in each of the latter nine weeks. During the nationwide Influenza A₂ outbreak of 1962-1963, this geographic division was the only one that did not experience pneumonia-influenza deaths of epidemic proportions.

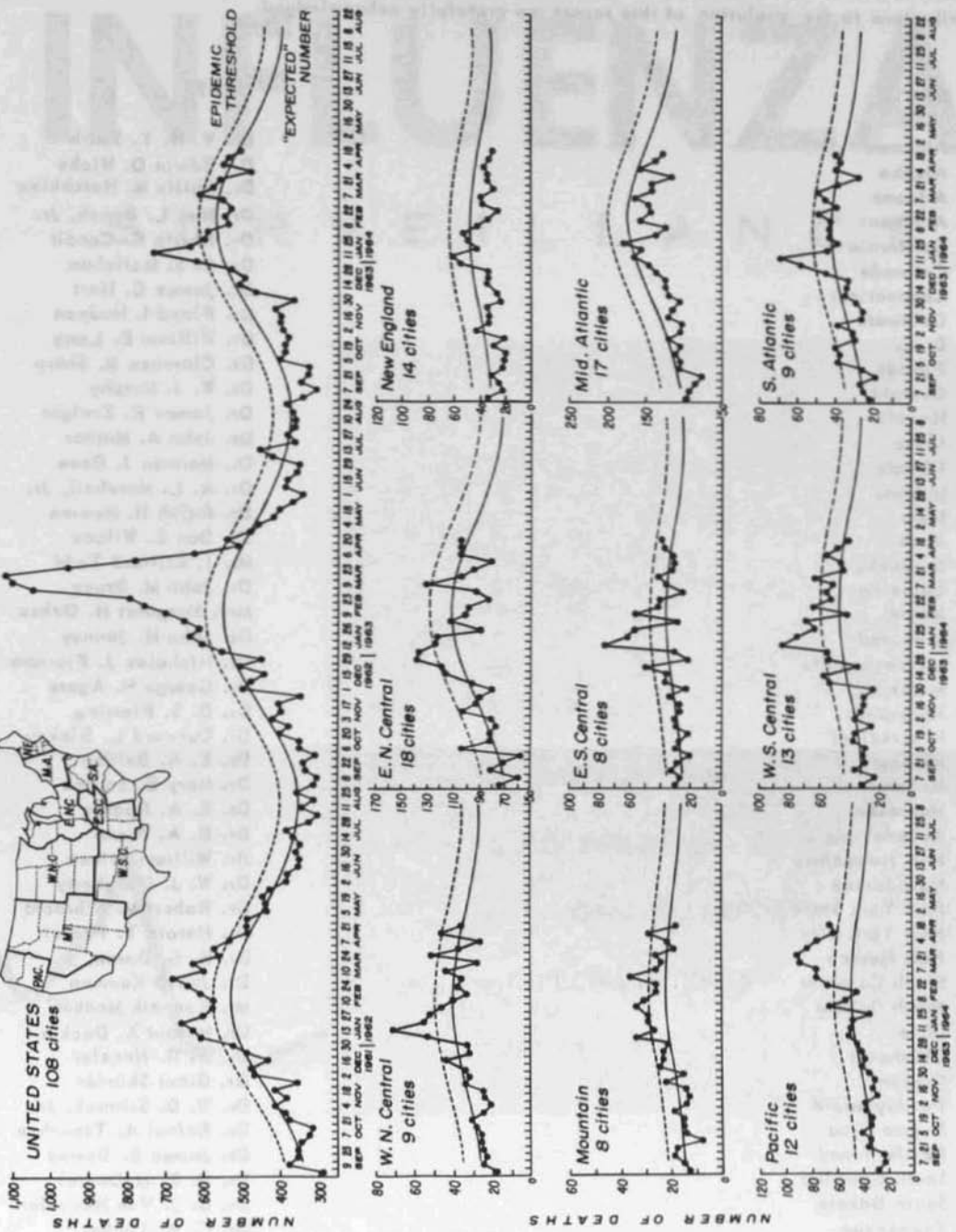
The twelve reporting cities in the Pacific Division reported a total of 724 pneumonia-influenza deaths this year as compared with 518 during a comparable 10-week period last year, - a 40 percent increase.

For one week during the past 10 weeks (week ended March 7), the total number of pneumonia-influenza deaths for the United States exceeded the epidemic threshold. This was a result of the continuing outbreak on the west coast and an increase to expected levels in the numbers reported by the Middle Atlantic and East North Central States after two weeks in which both divisions had reported less than expected.

Weekly number of pneumonia-influenza deaths for the Pacific States for 10 comparable weeks in 1963 and 1964 are shown below:

1964 Week Ended	Number	1963 Week Ended	Number
Feb. 15	43	Feb. 16	54
22	66	23	41
29	75	Mar. 2	52
Mar. 7	75	9	40
14	88	16	42
21	90	23	49
28	84	30	58
Apr. 4	76	Apr. 6	53
11	62	13	66
18	<u>65</u>	20	<u>63</u>
	724		518

WEEKLY PNEUMONIA AND INFLUENZA DEATHS



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.

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New York City	Dr. Harold T. Fuerst
New Mexico	Dr. H. G. Doran, Jr.
North Carolina	Dr. Jacob Koomen
North Dakota	Mr. Kenneth Mosser
Ohio	Dr. Harold A. Decker
Oklahoma	Dr. F. R. Hassler
Oregon	Dr. Grant Skinner
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. Elton Newman
Vermont	Dr. Linus J. Leavens
Virginia	Dr. James B. Kenley
Washington	Dr. E. A. Ager
West Virginia	Dr. L. A. Dickerson
Wisconsin	Dr. Josef Preizler
Wyoming	Dr. Helen A. Moore