Influenza Epidemic in Pennsylvania

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A NEW VARIANT of influenza A_2 virus was first reported from Hong Kong in July 1968. Laboratory studies demonstrated that the virus had undergone a major antigenic shift within the A_2 classification. In the 2 successive months, widespread outbreaks of illness in the Far East were ascribed to the A_2/Hong Kong/68 influenza virus. In a special session in September 1968, the Public Health Service Advisory Committee on Immunization Practices issued a statement that the change in the virus increased the probability of a significant outbreak of influenza in the United States during the winter of 1968–69 (*1*).

The introduction of the Hong Kong strain into the United States was documented first in early September and was followed by occasional small outbreaks at military establishments which received personnel from the Orient (*2*). In October and November increasing numbers of outbreaks were reported in civilian populations in the western States (*3*).

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In late November 1968, when isolated cases of influenza became more frequent in Pennsylvania, the division of communicable diseases, Pennsylvania Department of Health, organized a prospective study to determine the impact of the disease on that State's population. A statewide influenza surveillance system based on institutional absenteeism was organized to (*a*) delineate the extent of the outbreak in the State, (*b*) facilitate early laboratory confirmation of suspected cases, (*c*) elucidate the clinical and epidemiologic characteristics of influenza infections, and (*d*) provide current information on the course of the epidemic to concerned medical groups and the general population. After the outbreak, the resultant mortality was analyzed by reviewing death certificates issued throughout the State.

Observations on the epidemiology of the 1968–69 influenza epidemic in Pennsylvania are reported in this paper. Organization and operation of the influenza surveillance system and the laboratory studies are discussed in detail elsewhere (*4, 5*).

Methods of Study

Absenteeism. Monitoring of absenteeism was initiated with the selection of 13 sites for surveillance throughout the six geographic areas of the State (fig. 1). Several local institutions near each site agreed to furnish daily attendance fig-

ures. We attempted to enlist at least one industrial plant, one secondary school (grades seven through 12), and one elementary school (kindergarten through sixth grade) at each site (*4*). The total population under surveillance, excluding Pittsburgh and Philadelphia (which had separate surveillance systems), was 8,079,500. A sample of 101,613 people or about 1.2 percent was included in the daily monitoring of absenteeism.

Attendance figures were collected for 42 days from December 2, 1968, through January 31, 1969. The 19 days for which there were no reports were Saturdays, Sundays, or holidays. In addition, because of the Christmas recess, no data on absences were collected from the schools from December 23 through January 1. Moreover, not all of the participating institutions were successful in compiling and reporting daily attendance. On the average, for any given day, about 71 percent of the participating plants and 91 percent of the participating schools furnished attendance reports.

To facilitate interpretation of absences, we selected epidemic threshold levels sufficiently high to preclude their being exceeded by random daily or weekly fluctuations in absenteeism. Since it was impossible to obtain baseline absenteeism figures for each of the participating schools, the State average of about 5 percent per day recorded during the 1967–68 school year for elementary and secondary schools, exclusive of Pittsburgh and Philadelphia, was used.

The epidemic threshold was arbitrarily set at 9 percent, approximately double the baseline absenteeism for all participating schools. Based on very rough estimates of the usual absenteeism in industrial plants, the epidemic threshold for the plants was set twice as high as the expected level. This rationale was expected to lessen the possibility of sporadic variations being interpreted as unusual events.

Mortality. Death certificates issued throughout the State were reviewed and analyzed. With the aid of machine-tabulated punchcards prepared from death certificates, death rates were computed for age, sex, race, date of death, and cause of death. Cause of death was coded in accordance with recommendations of the National Center for Health Statistics (*6*), and adjusted for the changes in the World Health Organization's International Classification of Diseases adapted for use in the United States (*7*).

Mortality figures for January 1961 through May 1969 were reviewed. Age, sex, race, infant,

Figure 1. Influenza surveillance sites, Pennsylvania, December 1968–January 1969

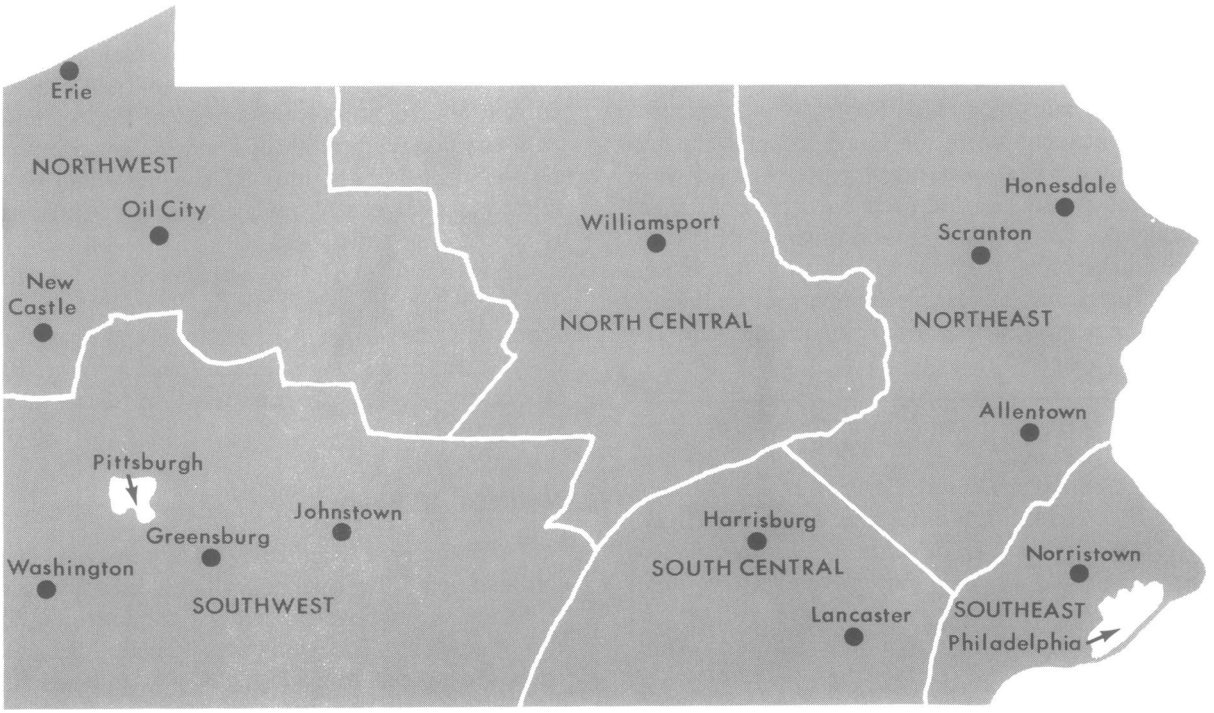
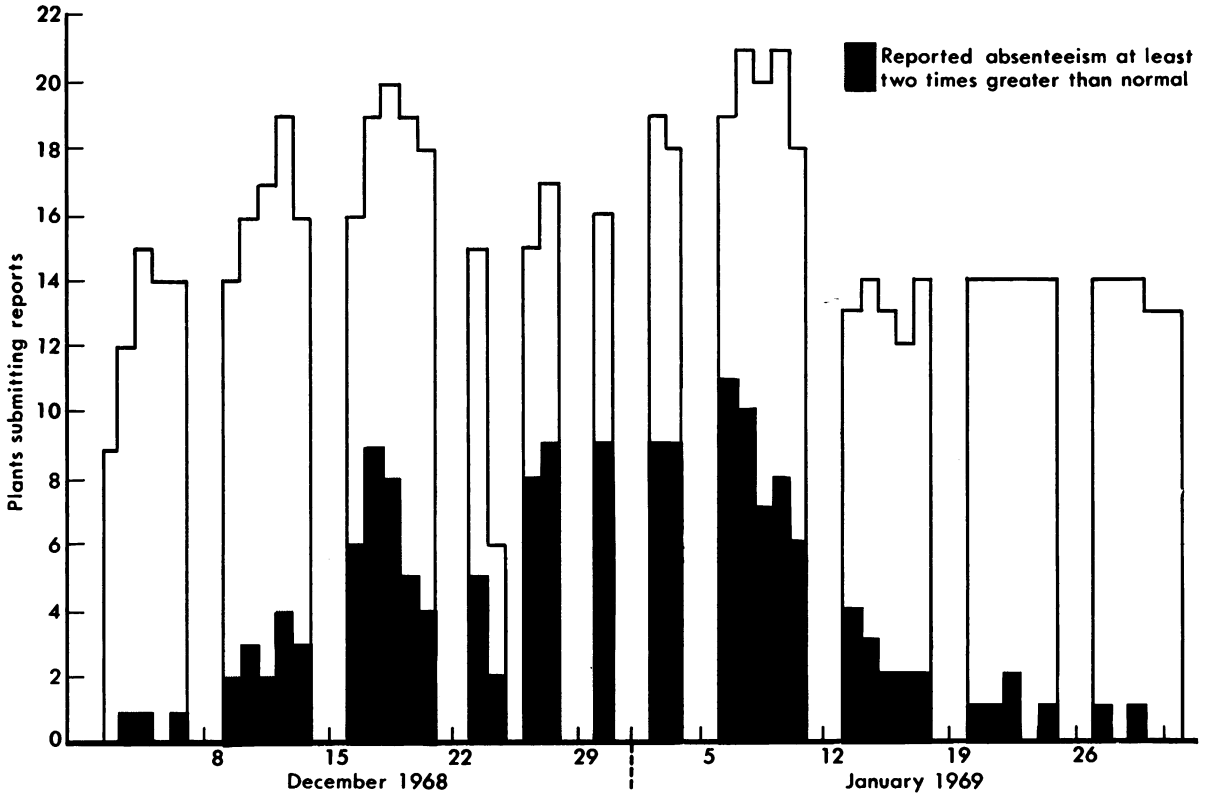


Figure 2. Industrial absenteeism in 22 plants, Pennsylvania, December 1968–January 1969



maternal, and disease-specific mortality rates for 40 causes of death were calculated. Expected rates were calculated for each group by fitting a curve to the observed death rates during nonepidemic periods according to the methods of Serfling (*8*). The epidemic threshold, a curve 1.65 standard deviations above the expected mortality, was also calculated on the basis of previously described techniques (*9*).

Results

School absenteeism. Figure 2 shows the combined elementary and secondary school absenteeism. To equalize the weight of data from different surveillance sites, schools of the same category, in close geographic proximity, and in the same school district are presented as a single reporting unit (*4*). Thus, the 14 primary schools in the Harrisburg area are given the same statistical weight as the single elementary school in Erie.

Only the reporting units that had at least 9 percent absenteeism are plotted. The highest absenteeism was recorded during the third week of December and the first week of January, when about 55 percent of the reporting units registered more than 9 percent absenteeism. The curve suggests that had there been no Christmas recess, peak absenteeism would have occurred during the last week of December or the first week of January.

Absenteeism recorded the third week of December probably was influenced by the opening of the hunting season on December 16, as evidenced by the progressive decrease in absenteeism December 17–19, following the weekly peak on December 16. The greatest difference between secondary and primary school absenteeism was recorded on that day: secondary school absences ranged from 8 to 24.8 percent, for an average of 13.8 percent; primary school absences were generally about 3 percent lower, ranging from 4 to 14.3 percent, for an average of 10.7 percent.

Otherwise, absenteeism from secondary schools paralleled that in elementary schools by a slight, but a very consistent, margin. For example, on January 3, during a period of significant influenza activity, secondary schools reported absenteeism from 7.1 to 26.2 percent, for an average of 11.3 percent, while primary schools reported absen-

teeism ranging from 5.5 to 27.6 percent, for an average of 11.1 percent. On January 28, when most of the influenza activity had subsided, the absenteeism reported by secondary schools ranged from 2.2 to 8.9 percent, for an average of 6.4 percent; while primary school absenteeism ranged from 4 to 9 percent, for an average of 6.1 percent.

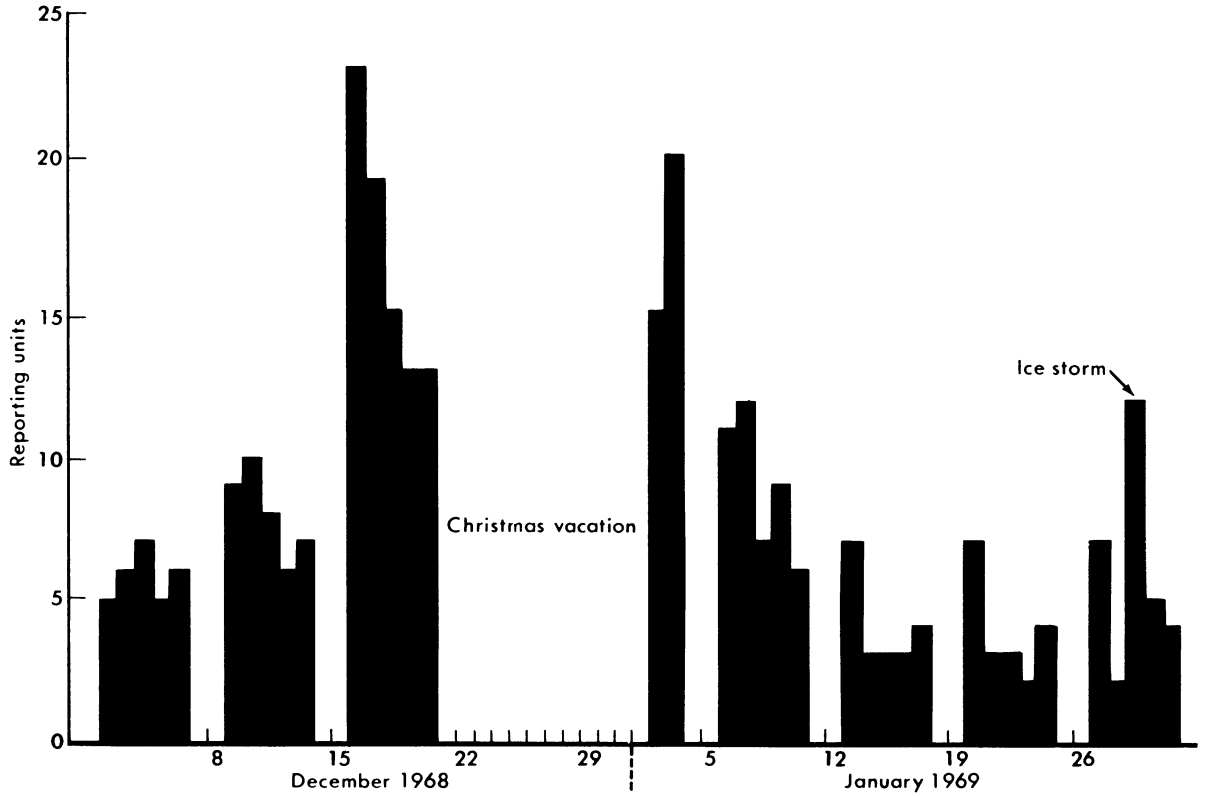
Industrial absenteeism. The expected daily absenteeism at the 22 industrial plants monitored ranged from 0.2 to 6.6 percent, for an average of 2.9 percent. Excluding days when bad weather or the opening of the hunting season precipitated brief increases, the highest daily absenteeism from individual plants during the period of surveillance ranged from 0.5 to 15.7 percent, for an average of 6.4 percent.

Figure 3 shows the proportion of daily reports that indicated at least twice the baseline for a given industrial plant. It appears that more than 10 percent of the plants had excessive absenteeism from December 9 to January 17. The peak occurred January 6, when 57.9 percent of the plants reported absenteeism ranging from 0.4 to 11.5 percent, for an average of 4.7 percent. A slightly smaller peak of 56.3 percent was reported December 30. The first 3 days of the hunting season, December 16–18, produced a separate peak (47.4 percent on December 17) distinct from the absenteeism curve as a whole.

Mortality

The observed number of pneumonia-influenza deaths exceeded the epidemic threshold during the winters of 1962–63, 1964–65, 1967–68, and 1968–69, reflecting periods of known influenza activity. The 1968–69 epidemic was the largest since 1962–63. Weekly pneumonia and influenza mortality and the total mortality for the 1968–69 epidemic peaked during the first week of January 1969. The excess deaths, determined by subtracting the expected from the observed number, during the last five influenza outbreaks in Pennsylvania is shown in the following table.

Figure 3. School absenteeism in 32 reporting units[1] with 9 percent or more absenteeism, Pennsylvania, December 1968–January 1969



[1] A reporting unit consists of one or more schools in a single school district.

Table 1. Pneumonia and influenza deaths, by age, sex, and race, Pennsylvania, December 1968–January 1969

Age	White males			White females			Nonwhite males			Nonwhite females		
	Population	Number of deaths	Rate per 100,000	Population	Number of deaths	Rate per 100,000	Population	Number of deaths	Rate per 100,000	Population	Number of deaths	Rate per 100,000
Less than 1	81,700	26	31.8	77,800	13	16.8	11,000	6	54.6	11,000	4	36.4
1–4	360,100	8	2.2	345,400	6	1.8	45,500	3	6.6	45,800	2	4.4
5–9	521,300	3	.6	503,500	4	.8	55,700	0	.....	55,500	0	.....
10–14	519,400	4	.8	501,000	1	.2	50,500	1	2.0	50,900	0	.....
15–19	476,300	3	.6	458,800	2	.4	41,600	2	4.8	45,200	0	.....
20–29	659,300	7	1.0	711,100	10	1.4	52,000	2	3.8	64,700	3	4.6
30–39	569,000	12	2.2	613,500	9	1.4	51,700	8	15.4	59,500	5	8.4
40–49	685,000	34	5.0	748,100	31	4.2	55,200	10	18.2	62,100	6	9.6
50–59	597,900	99	16.6	649,000	58	9.0	41,300	14	33.8	43,400	8	18.4
60–69	406,000	192	47.2	481,200	108	22.4	29,100	22	75.6	18,900	10	53.0
70–74	143,900	126	87.6	185,100	61	33.0	8,700	13	149.4	9,400	8	85.2
75–84	142,800	188	131.6	204,700	159	77.6	7,200	5	69.4	9,300	4	43.0
85 and over	24,300	83	341.6	41,300	110	266.4	1,300	6	461.6	2,000	2	100.0
Total	5,187,000	785	15.2	5,520,500	572	10.4	450,800	92	20.4	477,700	52	10.8

Epidemic period	*Excess deaths*
February–March 1962	1,335
February–March 1963	4,118
January–March 1965	970
January–February 1968	2,099
December 1968–January 1969	2,804

Comparison of the pneumonia-influenza mortality with total mortality shows a significant increase in deaths ascribed to causes other than pneumonia-influenza during periods of influenza activity. Data on mortality attributed to 40 disease categories were compared to determine how the number of deaths per month attributed to each category had varied from January 1963 to April 1969. Heart disease, cerebrovascular disease, diabetes, bronchitis, and asthma, in addition to pneumonia-influenza, exceeded their expected mortality level by more than 1.65 standard deviations during periods of influenza activity. Distribution of the excess mortality during December 1968 and January 1969 by stated cause of death is shown in the following table.

Cause of death	*Number*	*Percent*
Total excess	2,804	100.0
Pneumonia-influenza	782	27.9
Heart disease	851	30.3
Cerebrovascular	179	6.4
Diabetes	101	3.6
Bronchitis	90	3.2
Asthma	21	.8
Other	780	27.8

The age, sex, and race of persons who died from pneumonia-influenza in Pennsylvania during December 1968 and January 1969 are shown in table 1. In almost all age groups, influenza mortality was greater for males than females. White persons in general had a lower pneumonia-influenza mortality than nonwhite persons. The pneumonia-influenza mortality per 100,000 adjusted to a standard population in order to remove the difference caused by a varying age distribution, was 16.1 for white males, 25.8 for nonwhite males, 9.2 for white females, and 14.6 for nonwhite females.

Table 2 shows the age, sex, and race of persons whose deaths were recorded during the December 1968 and January 1969 influenza epidemic and compares them with similar data recorded during December 1965 and January 1966, when there was no influenza epidemic.

Table 3 compares the expected mortality rates for December 1968–January 1969 with the observed rates for eight age groups. The percentage of excess is a measure of the relative increase in mortality derived by dividing the excess mortality rate by the expected rate and multiplying the result by 100. Persons 5 through 39 years had higher relative increases in mortality than all other age groups.

No excess mortality was observed for children under 5 years of age during the 1968–69 epidemic. To examine the effects of influenza epidemics on infant mortality more closely, the observed monthly mortality of children under 1 year was plotted for January 1961 through May 1969. Compared on a histogram, observed infant mortality did not deviate significantly from ex-

pected mortality during periods of influenza activity. Similarly plotted maternal mortality also did not show any significant deviation from expected levels during influenza epidemics.

Geographic Spread of Influenza

Geographic progression of the epidemic in Pennsylvania was reviewed by analyzing the absenteeism and mortality recorded in each of the State's six geographic areas and Philadelphia. It took 5 weeks for peak pneumonia-influenza mortality to cross the State, beginning in the densely populated southeast corner and spreading west and north (fig. 4). On the average, peak recorded absenteeism preceded peak mortality by 8 or 9 days, but in one region peak absenteeism preceded peak mortality by as much as 4 weeks, and in another, peak absenteeism followed peak mortality by 1 week.

Discussion

The observed course of influenza activity in Pennsylvania during the winter of 1968–69 can best be characterized by the theoretic mechanism of influenza spread described by Jordan (*10*). The pattern he perceived consisted of slow, insidious seeding of virus in the community; followed first by sporadic, self-limited outbreaks and then by widespread infections, exceeding the epidemic threshold; and resulting in a brief, full-scale outbreak terminated by a rapid decrease of influenza activity.

The first documented seeding of the influenza virus in Pennsylvania was as early as September 1968, when a laboratory technician working with the A_2/Hong Kong/68 virus in Philadelphia became ill and transmitted the infection to her husband. Sporadic cases of influenza, some documented by isolations of A_2/Hong Kong/68 influenza virus, were reported to the State division of communicable diseases in October and early November 1968. The majority of these cases were in the southeastern part of the State, most of them in Philadelphia.

Isolated cases, however, were also reported from other parts of the State. In particular, several well-documented cases of influenza at Pennsylvania State University, which is located in the central part of the State, were confirmed by viral studies several weeks before peak influenza activity occurred in that region (*5*).

Sporadic outbreaks involving institutionalized populations began in Philadelphia during the second half of November. A State hospital for the

Table 3. Percent excess mortality (deaths per 100,000 persons), influenza epidemic, Pennsylvania, 1968–69

Age (years)	Expected rate	Observed rate	Excess rate	Percent excess
Less than 1	423.9	376.9	−47.0	−11.1
1–4	17.3	14.6	−2.7	−15.6
5–9	6.8	8.6	1.8	26.5
10–19	9.3	11.4	2.1	22.6
20–39	23.9	30.5	6.6	27.6
40–59	140.9	165.5	24.6	17.5
60–74	604.9	709.0	104.1	17.2
75 and over	2,004.7	2,148.5	143.8	7.2

Table 2. Mortality rates (deaths per 100,000 persons) for an epidemic a nonepidemic period (December

Age	White male			White female		
	Epidemic	Nonepidemic	Excess	Epidemic	Nonepidemic	Excess
Less than 1	357.5	440.6	−83.1	295.0	344.5	−49.5
1–4	13.6	17.5	−3.9	12.3	15.1	−2.8
5–9	7.7	6.8	.9	8.3	5.4	2.2
10–14	9.6	6.7	2.9	4.2	4.0	.6
15–19	21.7	18.3	3.4	8.3	7.6	.7
20–29	31.8	21.8	10.0	12.5	10.0	2.5
30–39	42.1	36.6	5.5	21.1	22.5	−1.4
40–49	111.4	95.9	15.5	67.0	56.9	10.1
50–59	314.8	292.2	22.6	154.4	130.8	23.6
60–69	782.7	670.9	111.8	376.5	340.6	35.9
70–74	1,329.2	1,179.3	149.9	767.4	675.3	92.1
75–84	2,183.8	2,040.6	143.2	1,513.8	1,490.0	23.8
85 and over	4,420.4	4,436.2	−15.8	3,927.1	3,845.0	82.1
Total	248.9	226.5	22.4	181.9	170.5	11.4

mentally ill, a large, city-owned nursing home, and a home for the chronically ill were among the first to report outbreaks. Early in December the University of Pennsylvania and Temple University reported increased visits to their health clinics for treatment of influenza-like illnesses. Apparently a full-scale outbreak was beginning. This pattern was repeated across the State. Sporadic outbreaks identified foci of influenza activity, and as more persons and communities were affected, the foci coalesced, cases exceeded the epidemic threshold, and a full-scale epidemic occurred.

Absenteeism. During this epidemic, reports of absenteeism from school were useful in identifying areas with possible influenza activity and helping us decide where to direct collection of specimens for viral studies to confirm the presence of influenza. The susceptibility of school children in the spread of influenza, though heavily emphasized by Langmuir and others (*11–13*), was difficult to confirm.

Schools did, however, have a higher rate of absenteeism than the industrial plants and usually were the first institutions in the community to be affected by the epidemic. Nevertheless, the highest average daily absenteeism recorded by any school during the epidemic was only 12.3 percent. The State's division of communicable diseases did not receive any report of elementary or secondary school closings in Pennsylvania as a result of influenza-induced student or teacher absenteeism during December or January. Perhaps the intervention of the Christmas recess reduced the influenza attack rate in the schools.

In general, the increase in industrial absenteeism followed increased school absenteeism by about 1 week. More than 10 percent of the reporting industrial plants reported absenteeism of at least twice the expected level for 6 weeks from December 9 through January 17. Peak industrial absenteeism recorded on January 6, 1969, followed the estimated school absenteeism peak by 1–2 weeks. This observation corresponds to those made during the 1957–58 influenza epidemic, which showed that the adult population is stricken about 1–2 weeks after the school age population (*14*).

Analyses of the age- and sex-specific attack rates for the 1957–58 epidemic revealed that working age men had lower attack rates than school children and their immediate household contacts. In a study of 60 Cleveland families during the fall of 1957, Jordan and co-workers found an attack rate of 72.9 percent for school children, 32.3 percent for mothers, 30 percent for preschool children, and only 16.1 percent for fathers (*15*).

During the 1968–69 epidemic in Pennsylvania, the highest daily absenteeism in the individual industrial plants ranged from 0.5 percent to 15.7 percent, for an average of 6.4 percent, about half the average of the highest daily absenteeism of 12.3 percent in schools. If it is assumed that absenteeism reflects the influenza attack rate, then it appears that the attack rate among industrial workers was significantly lower than among school children.

Mortality. Collins' studies have demonstrated that in the United States the most sensitive meas-

period (December 1968–January 1969) compared with mortality rates for 1965–January 1966), Pennsylvania

All other males			All other females			Total		
Epidemic	Non-epidemic	Excess	Epidemic	Non-epidemic	Excess	Epidemic	Non-epidemic	Excess
834.4	709.1	125.3	644.8	781.8	−137.0	376.9	436.4	−59.5
27.5	37.4	−9.9	25.0	21.8	3.2	14.6	17.8	−3.2
18.8	23.3	−4.5	7.6	9.0	−1.4	8.6	7.0	1.6
20.7	7.9	12.8	4.2	3.9	.3	7.5	5.4	2.1
40.1	33.7	6.4	4.6	19.9	−15.3	15.6	14.2	1.4
82.2	50.0	32.2	33.8	32.5	1.3	24.4	17.6	6.8
129.1	100.6	28.5	78.9	42.0	36.9	37.3	32.7	4.6
275.8	210.1	65.7	194.8	144.9	49.9	99.1	83.1	16.0
522.8	435.8	87.0	312.5	265.0	47.5	242.8	217.1	25.7
1,078.9	958.8	120.1	1,109.1	1,074.1	35.0	589.5	518.1	71.4
1,630.4	1,218.4	412.0	1,087.4	914.9	172.5	1,030.7	904.4	126.3
1,912.2	1,569.4	342.8	1,457.9	1,204.3	253.6	1,783.1	1,700.3	82.8
3,770.9	2,307.7	1,463.2	3,233.3	2,400.0	833.3	4,078.1	3,982.6	95.5
279.9	228.0	51.9	194.4	170.0	24.4	216.1	197.7	18.4

ure of an influenza epidemic is the number of excess deaths ascribed to pneumonia and influenza (*16*). He also demonstrated that during influenza epidemics excess mortality occurred as a result of other causes of death (*17*).

Calculating the total excess mortality during an influenza epidemic is the most direct method of assessing the extent and severity of the epidemic and its impact on a given community. However, total excess mortality is a less sensitive index of influenza activity than the excess number of deaths from pneumonia and influenza. The 2,804 excess deaths during the 1968–69 influenza epidemic in Pennsylvania suggested the most severe influenza epidemic since February–March 1963, when 4,118 excess deaths occurred.

Of the disease categories represented on death certificates, six showed significant correlation with influenza. During the 1968–69 epidemic, heart disease accounted for the greatest proportion, 30.3 percent, of the excess mortality. Smaller percentages were attributed to pneumonia-influenza, cerebrovascular disease, diabetes, bronchitis, and asthma.

The remaining 27.8 percent excess mortality may have been caused by diseases such as emphysema, occupational pneumoconiosis, and tuberculosis. However, the number of deaths ascribed to some of these disease categories was small, and the changes in the recording and classification of diseases precluded comparing figures from consecutive years. Consequently, data on deaths caused by diseases in these categories did not show statistically significant correlations with periods of influenza activity.

The greater prevalence of chronic cardiovascular and pulmonary diseases among older persons undoubtedly contributed to their high mortality during the epidemic. Physiological studies substantiate the observation of increased risk of death for patients with cardiovascular diseases, chronic pulmonary disorders, or diabetes when influenza infection is superimposed (*18, 19*).

Comparison of the observed and expected monthly infant mortality from January 1961 through May 1969 did not show any statistically significant increases during the influenza epidemic. Conversely, the observed infant mortality during the 1968–69 epidemic was lower than expected: infant mortality continued to fluctuate around the usual monthly level during the influenza epidemic. February 1963 was the only month during a known influenza epidemic in which the observed infant mortality exceeded that expected by more than 1.65 standard deviations. Thus, on the basis of the data obtained in this study, we concluded that the last five influenza epidemics in Pennsylvania have had a negligible impact on infant mortality.

The relative age-specific excess mortality figures are characterized by high levels in the school age and young adult groups and lower levels in the

Figure 4. Geographic progression of absenteeism, percentage of reports showing abnormal absenteeism, Pennsylvania, December 1968–January 1969

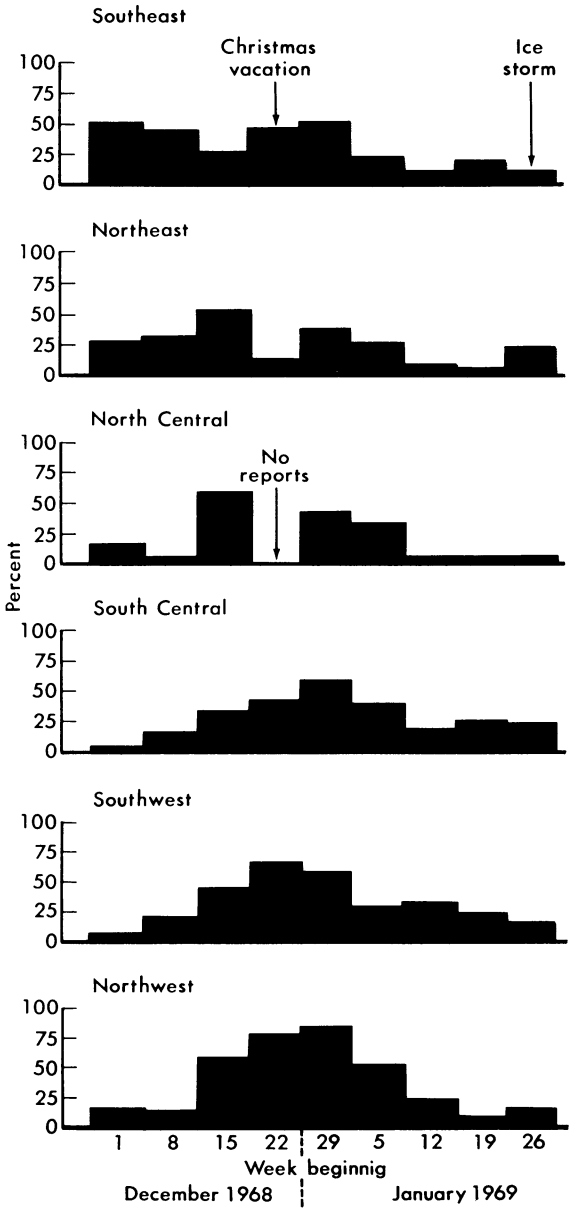
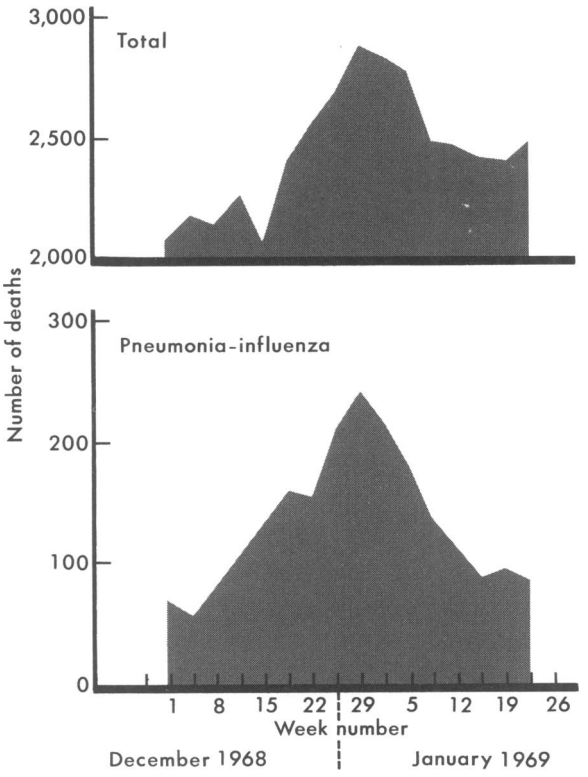


Figure 5. Pneumonia-influenza mortality compared with mortality from all causes, by week, Pennsylvania, December 1968–January 1969



elderly and the preschool age groups. This pattern may become characteristic of influenza epidemics caused by new strains of influenza A viruses. Serfling and co-workers have shown that a similar pattern occurred during the 1957–58 epidemic, when a new influenza A_2 virus appeared and was antigenically modified in 1960 and 1963 to produce more even distribution of relative age-specific excess mortality (*20*). Other observations showed that the age-specific attack rates during 1960 and 1963 had changed correspondingly, resulting in much lower attack rates in school age children and young adults, while remaining approximately the same in the older groups (*13, 14*).

An immunologic explanation of this phenomenon was offered by Widelock and associates, who determined influenza A_2 titers in different age groups (*21*) by serologic assays. They showed that the persistence of antibodies in a high percentage of younger persons corresponded to the reduced attack rate and lower excess mortality in subsequent epidemics caused by influenza A_2 virus. In older persons, because of lower initial attack rates in the 1957–58 epidemic and possible waning of antibody levels, the attack rates and excess mortality in 1960 and 1963 were approximately the same magnitude as in 1957–58. Consequently, if the same pattern of immunity prevails for viruses like A_2/Hong Kong/68, subsequent epidemics caused by these viruses may be expected to produce significantly lower attack rates and mortality in school age children and young adults but nearly identical attack rates and mortality in the older persons.

Age-specific mortality rates were generally lower for white than nonwhite persons. A possible explanation for this may be the higher socioeconomic status of the white population. Life insurance statistics show that influenza-pneumonia mortality is significantly higher for persons insured under an "industrial policy." An industrial policy generally reflects a lower socioeconomic status of the insured; the "ordinary life policy" generally reflects higher socioeconomic status (*22*). Other studies have also noted a similar relationship between the socioeconomic level and influenza mortality, although a causal relationship is hard to establish (*23*).

No correlation between maternal mortality and influenza epidemics in Pennsylvania from 1961 to 1969 was observed. In the influenza epidemics before 1932, Collins demonstrated that death rates caused by acute complications of pregnancy and childbirth showed influenza-associated excesses (*16*). In subsequent epidemics, it has been impossible to demonstrate this correlation by analyzing mortality statistics. Under the present system of registering, coding, and tabulating death certificates, when a pregnant woman dies from pneumonia or influenza, the pregnancy is not noted. Consequently, these deaths are excluded when maternal mortality data are analyzed.

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The epidemiologic features of the 1968–69 influenza epidemic in Pennsylvania were studied by concurrent statewide absenteeism and retrospective review of death certificates. Peak industrial absenteeism occurred on January 6, 1968, following the estimated peak school absenteeism by a little more than 1 week. The highest average daily absenteeism was 12.3 percent for schools and 6.4 percent for industrial plants. School children and young adults had significantly higher attack rates than older adults and preschool children.

In addition to pneumonia-influenza deaths, excess mortality was also registered for persons with heart disease, cerebrovascular disease, diabetes, bronchitis, and asthma. Mortality increased almost geometrically with age. White persons had lower mortality rates than nonwhites; and females, in general, had a lower mortality rate than males. No significant increase in infant or maternal mortality could be demonstrated.
