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Age and secular distributions of virus-proven influenza patients in successive epidemics 1961–1976 in Cirencester: Epidemiological significance discussed

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SUMMARY

A general practice population of around 3900, under continuous clinical and laboratory surveillance, experienced 20 outbreaks of influenza between March 1960 and March 1976. Four epidemics were caused by subtype H2N2 type A viruses, seven by subtype H3N2 type A viruses and nine outbreaks by type B viruses. The age of every person proved virologically to have influenza is related to the age structure of the community and to the phase of the epidemic in which the virus-positive specimens were collected. Children 0–15 years old suffered a higher incidence rate than adults 16–90+. Pre-school children 0–4 suffered the highest rate of infection by viruses of both influenza A subtypes, whereas older school-children 10–15 suffered the highest rate of type B infections. Despite these high incidence rates neither pre-school nor schoolchildren appear to have been the major disseminators of any of these influenza viruses in the community.

Adults of all ages suffered a high rate of infection even into extreme old age, and the indiscriminate age distribution among adults was sustained in the successive epidemics. Such age-patterns are not those caused by a highly infectious immunizing virus surviving by means of direct transmissions from the sick, whose prompt development of the disease continues endless chains of transmissions. An alternative epidemic mechanism – whereby the virus does not spread from the sick but becomes latent in them, reactivating seasonally so that they later infect their companions – would produce age patterns similar to those recorded here for influenza patients. The suggested mechanism is illustrated by a simple conceptual model and the influenzal age patterns are discussed in relation to the recycling of influenza A subtypes.

INTRODUCTION

Infectious diseases impose an age-pattern on their victims which may convey important information concerning aspects of the association of the causal agent with its human host in the community attacked. Measles in a non-immune community attacks indiscriminately people of all ages but, when it returns, the pattern of the ages of persons attacked and spared bespeaks the number of years elapsing since its previous visit (Panum, 1940) and also the lifelong immunity usually conferred by an attack of measles.

The age-distributions may also convey information concerning the relative infectiousness of different agents in the same community. The more infectious agent will claim more victims and travel through the community more rapidly and its victims will, on average, be younger than those of the less infectious agent. Measles patients in a particular community are, on average, younger than varicella patients, and mumps patients are, on average, considerably older than either, indicating crudely the relative infectiousness of the three viruses (Hope-Simpson, 1952), though other factors such as the degree of urbanization of the community help to determine the actual ages at which persons are attacked.

The age-patterns of patients may also provide clues as to the source from which an agent has invaded a particular environment. The age of the primary household cases of variola minor in Brazil reveals the critical importance of the day-school child for introducing smallpox into the home and thence into the community at large (Smith *et al.* 1979). British practitioners are familiar with similar age-patterns in measles and chickenpox patients because the day school acts as a boosting mechanism for multiplying such infections and distributing them into the community via their households.

Several observers, noticing a higher incidence rate of influenza in children than in adults, have drawn similar inferences about the rôle of children in the spread of influenza (Banatvala *et al.* 1965; Hall, Cooney & Fox, 1973). Monto *et al.* 1969 (in Kilbourne, 1975) found that a community in which a high proportion of children had been vaccinated against influenza suffered a lower infection rate in an influenza epidemic than a neighbouring community in which the children had not been immunized. They suggested that children may be providing the most important source for disseminating influenza in the community, with the corollary that immunization of children should reduce spread of the virus. Schoolchildren in particular have been incriminated as introducers of influenza into the household (Philip *et al.* 1961; Dingle, Badger & Jordan, 1974) and Glezen *et al.* (1980) consider from their findings that schoolchildren are the major disseminators of influenza and that the impact of epidemics on the community could be significantly reduced by immunizing schoolchildren. Hall, Cooney & Fox (1973), finding the highest rate of infection in children below school age, suggested that they, rather than the schoolchildren, might be the main source of community spread.

Not all observers have corroborated such findings. Neither pre-school nor schoolchildren were found to have preferentially introduced the virus into households affected by the 1951 epidemic (Hope-Simpson, 1951; Hope-Simpson & Sutherland, 1954). Davis *et al.* (1970), having found that the first epidemic of type A H2N2 influenza in 1957 spread primarily within schools, and that a schoolchild was then five times as likely as an adult to have introduced the illness into the family, found that the behaviour of the first epidemic of type A H3N2 influenza in 1968 was different, an adult being as likely as a schoolchild to be the first family case – a finding confirmed by Hope-Simpson (1970). This dual mode of family introduction weakens the suggestion that vaccination of schoolchildren should abort an influenza epidemic.

The finding that children have sometimes comprised a high proportion of the early cases in an influenza epidemic has also been adduced as evidence of their key role in disseminating the disease (Sarateanu & Ehrengut, 1976; Glezen & Couch, 1978).

All such interpretations of the age-patterns are founded upon the generally held assumption that influenza virus, like measles virus, is being transmitted directly from the sick person to his non-immune companions who, if infected, promptly develop influenza. Should this hypothesis be incorrect, and influenza virus not be surviving by this simple mechanism of endless chains of direct transmissions, the significance of the age-patterns of the persons with influenza would need to be reconsidered because the information they were conveying would have a different epidemiological interpretation. The epidemic mechanisms of influenza are far from clear, and recently an alternative mechanism has been proposed in which type A influenza virus becomes so rapidly latent in the tissues of the patient that during his illness he infects no one. Next season or later the latent virus residues are reassembled as infectious viral particles and the erstwhile patient becomes briefly a symptomless but highly infectious carrier. Epidemics consist solely of persons infected from these carriers. There is no further horizontal spread (Hope-Simpson, 1979, 1981). The alternative hypothesis was evolved to meet a large number of features of epidemic influenza that are unexplained by direct horizontal spread, one of which, the age-pattern in influenza, was mentioned but not studied in detail.

This paper provides the ages of all the virus-proven influenza patients found in 16 years continuous surveillance of the population of a general practice. The population was accurately characterized by age, so that the incidence rates could be calculated. The timing of each case in its epidemic is obtained from the date on which the first virus-positive specimen was collected.

The question of children as the major disseminators of influenza is discussed. The age-patterns are also considered in relation to their concordance or otherwise with current concepts of influenzal epidemiology and with the suggested alternative hypothesis.

METHODS

The general practice population, some 3900 persons, was under continuous clinical and laboratory surveillance from 10 March 1960 until 31 March 1976. Specimens for virus and bacterial examination were collected from a high but variable proportion of persons attended in their home or in the practice premises by the two doctors. All sorts of medical conditions were thus examined and specimens were also taken from well persons and from those suffering from non-infective complaints. Details of the methods and laboratory techniques were given by Hope-Simpson & Higgins (1969).

The ages of virus-proven cases of influenza are grouped so as to distinguish children below school age (0–4 years), younger school-aged children (5–9 years), older school-aged children (10–15 years), young adults (16–19 years), and older adults (20–90+ years). Persons aged 15 years or less are also analysed in single-year age groups, persons of 20 years or more in 10-year age groups. For some purposes those over 70 years old are grouped together to obtain numbers comparable to those in the 10-year age groups.

The statutory school leaving age in England and Wales was raised to the 16th year on 1 September 1972. Children born between 1 September and 31 January may leave at Easter after their 15th birthday. Those born between 1 February and 31 August may leave after the Spring Bank Holiday but permission depends

Table 1. *Number of virus-proven cases of influenza by type and subtype of virus, by age-group and rate/1000 of population/epidemic*

		Type A H2N2		Type A H3N2		Type B	
Age group	Population	Cases	Rate	Cases	Rate	Cases	Rate
0	50	5		11		1	
1	55	2		5		4	
2	58	6		8		9	
3	66	5		8		6	
4	68	4		8		6	
0–4	297	22	18·5	40	19·2	26	9·7
5	78	2		10		2	
6	68	1		12		6	
7	77	2		2		11	
8	72	2		5		3	
9	57	1		5		5	
5–9	352	8	5·7	34	13·8	27	8·5
10	51	3		5		5	
11	53	1		6		10	
12	64	2		6		7	
13	56	5		6		7	
14	58	1		3		7	
15	48	3		2		5	
10–15	330	15	11·4	28	12·1	41	13·8
(5–15)	(682)	(23)	(8·4)	(62)	(13·0)	(68)	(11·1)
16	190	1	1·3	18	13·5	12	7·0
20	538	13	6·1	43	11·4	11	2·3
30	433	14	8·1	31	10·2	11	2·8
40	448	6	3·4	41	13·1	16	4·0
50	481	15	7·8	39	11·6	6	1·4
60	429	11	6·4	37	12·3	8	2·1
70	274	6	5·5	16	8·3	5	2·0
80	106	2	4·7	6	8·1	4	4·2
90+	33	2	15·2	1	4·3	1	3·4
(70–90+)	(413)	(10)	(5·8)	(23)	(8·0)	(10)	(2·7)
16–90+	2932	70	6·0	232	11·3	74	2·8
Total	3911	115	7·4	334	12·2	168	4·8

on the examinations they expect to take. Thus after 1 September 1972 some children aged 15 would have been at school and some would have left. Before that date, many more 15-year-olds would have left school.

The general practice possesses a register of all patients by date of birth and keeps it up to date by a weekly correction for births, transfers and deaths. Each year a census is carried out recording the number of persons, male, female and total at each year of age. For this paper the census of 31 December 1972 was used to determine the rates. Comparison with the other relevant censuses showed this census to be reasonably representative of the population during the survey period. Rates are given as the number of cases per 1000 in the relevant age group of that population per epidemic. The dates on which each virus-positive specimen was

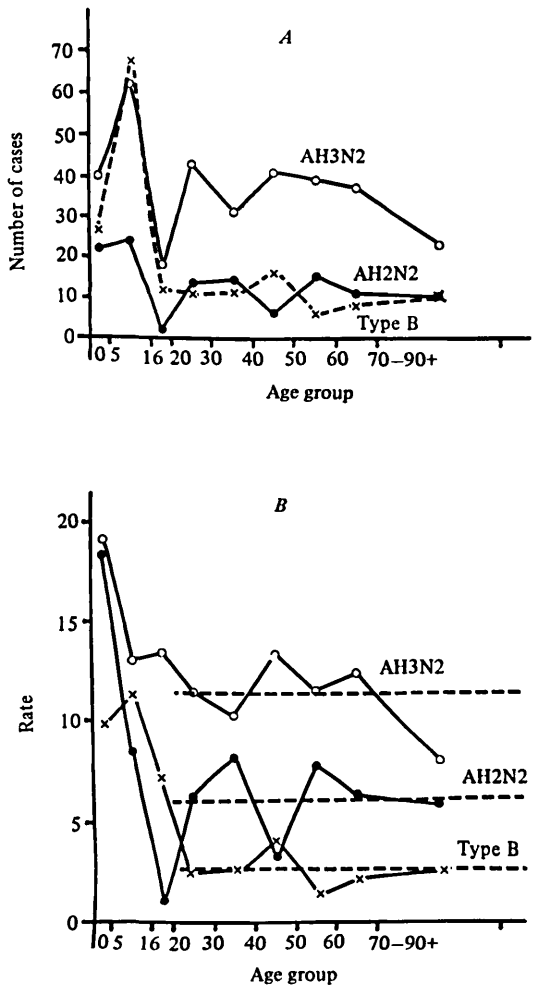

Fig. 1. Influenza virus infections by type and subtype by age group. Type A H2N2 ●, H3N2 ○, type B ×. A. Actual number of infections in age groups. B. Rate per thousand of population in age group per epidemic. Broken lines indicate the average incidence rate in persons aged 16–90+ years for each virus.

taken are related to their timing in the relevant epidemics ('day-in-epidemic') by reckoning as 'day one' the date of the first virus-positive specimen found in each epidemic.

RESULTS

The complete data have been summarized into 20 tables in the Appendix to this paper, each table providing: date the positive specimen was collected, day-in-epidemic and ages of infected persons for each outbreak of influenza. Only a few of the many analyses are used in this paper.

Text Table 1 demonstrates the following findings (see also Fig. 1, A, B).

1. None of the age groups in the general practice population escaped infection by either subtype of type A or by type B influenza viruses.

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Table 2. *Number and percentage of influenza cases in pre-school, school-aged and post-school-aged persons by phase of epidemic*

Age	Phase of epidemic: 1–10 days		11–20 days		21–30 days		31–39 days		40+ days		Total	
Type A H2N2 influenza cases by age and phase of epidemic, number and percentage												
0–4	1	8·3	1	9·1	4	17·4	4	21·1	12	24·0	22	19·1
5–15	0	0	1	9·1	5	21·7	3	15·8	14	28·0	23	20·0
16–90+	11	91·7	9	81·8	14	60·9	12	63·2	24	48·0	70	60·9
Total	12	100·0	11	100·0	23	100·0	19	100·1	50	100·0	115	100.0
Type A H3N2 influenza cases as above												
0–4	0	0	14	19·4	5	17·9	2	13·3	19	12·8	40	12·0
5–15	22	31·0	13	18·1	2	7·1	3	20·0	22	14·9	62	18·6
16–90+	49	69·0	45	62·5	21	75·0	10	66·7	107	72·3	232	69·5
Total	71	100·0	72	100·0	28	100·0	15	100·0	148	100·0	334	100·1
Type B influenza as above												
0–4	8	24·2	2	12·5	2	9·1	4	33·3	10	11·8	26	15·5
5–15	13	39·4	8	50·0	10	45·5	2	16·7	35	41·2	68	40·5
16–90+	12	36·4	6	37·5	10	45·5	6	50·0	40	47·1	74	44·0
Total	33	100·0	16	100·0	22	100·1	12	100·0	85	100·1	168	100·0

2. Children suffered a higher incidence rate of influenza than did adults, but the actual number of adult type A infections was much greater than the number in children (H2N2 70:45, H3N2 232:102). Type B infections in children, on the other hand, outnumbered those in adults (94:74).

3. Pre-school children suffered the highest rate of infection with both subtypes of type A virus, but they were outnumbered by the schoolchildren (H2N2, 23:22; H3N2, 62:40). Schoolchildren suffered a slightly higher rate of type B infections than pre-school children and greatly outnumbered them (60:26). The rate was highest in the older schoolchildren, who greatly outnumbered their school juniors (41:27).

4. The rate of influenza among the adults, although less than that of the children, was high and remained so throughout the adult age groups (Fig. 1). Some variations must be ascribed to the small numbers, for example the very high rate of type A H2N2 infections in nonagenarians and of type B infections in persons over 80 years old. For both these viruses the rate in the aggregate of all persons over 70 years old is near to the adult mean incidence rate. The youngest adults, 16–19 years, suffered little recorded influenza from type A H2N2 virus, but were heavily attacked by influenza A viruses of H3N2 subtype and by type B viruses.

Table 2 shows how widely all three viruses were distributed throughout the three main age groupings in all stages of the epidemics. Adults comprised the major proportion of early cases in all type A H2N2 epidemics.

The schoolchildren with type A H3N2 infections took their highest proportion of cases in the earliest 10-day phase of the combined epidemics but even so were heavily outnumbered by adults.

Children with type B infections outnumbered adults at almost all stages of the

Table 3. *The numbers and percentage of adults, schoolchildren and pre-school children in each epidemic caused by each of the three viruses. Also the adults are compared with the totals of the persons under* 16 *years old*

Distribution of three influenza viruses by age group by epidemic

	Adults (16+ yrs)		School (5–15)		Pre-school (0–5)		Total		Adults (16+ yrs)		Children (0–15)	
Epidemic	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
						A H2N2						
1963	12	85·7	1	7·1	1	7·1	14	99·9	12	85·7	2	14·3
1964	10	62·5	4	25·0	2	12·5	16	100·0	10	62·5	6	37·5
1966	21	56·8	8	21·6	8	21·6	37	100·0	21	56·8	16	43·2
1967–8	27	56·3	10	20·8	11	22·9	48	100·0	27	56·3	21	43·7
Total	70	60·9	23	20·0	22	19·1	115	100·0	70	60·9	45	39·1
						A H3N2						
1968–9	62	79·5	12	15·4	4	5·1	78	100·0	62	79·5	16	20·5
1969–70	75	65·8	26	22·8	13	11·4	114	100·0	75	65·8	39	34·2
1971–2	43	79·6	5	9·2	6	11·1	54	99·9	43	79·6	11	20·4
1972–3	16	59·3	6	22·2	5	18·5	27	100·0	16	59·3	11	40·7
1973–4	9	100·0	0	—	0	—	9	100·0	9	100·0	0	—
1974–5	17	51·5	8	24·2	8	24·2	33	99·9	17	51·5	16	48·4
1976	10	52·6	5	26·3	4	21·1	19	100·0	10	52·6	9	47·4
Total	232	69·5	62	18·6	40	12·0	334	100·1	232	69·5	102	30·5
						Type B						
1961–2	13	46·4	11	39·3	4	14·3	28	100·0	13	46·4	15	53·6
1965	8	53·3	6	40·0	1	6·7	15	100·0	8	53·3	7	46·7
1966	4	36·4	2	18·2	5	45·5	11	100·1	4	36·4	7	63·6
1968	7	36·8	7	36·8	5	26·3	19	99·9	7	36·8	12	63·2
1970	5	41·7	4	33·3	3	25·0	12	100·0	5	41·7	7	58·3
1971	15	55·6	10	37·0	2	7·4	27	100·0	15	55·6	12	44·4
1973 (spring)	2	100·0	0	—	0	—	2	100·0	2	100·0	0	—
1973–4	16	39·0	19	46·3	6	14·6	41	99·9	16	39·0	25	61·0
1976	4	30·8	9	69·2	0	—	13	100·0	4	30·8	9	69·2
Total	74	44·0	68	40·5	26	15·5	168	100·0	74	44·0	94	56·0

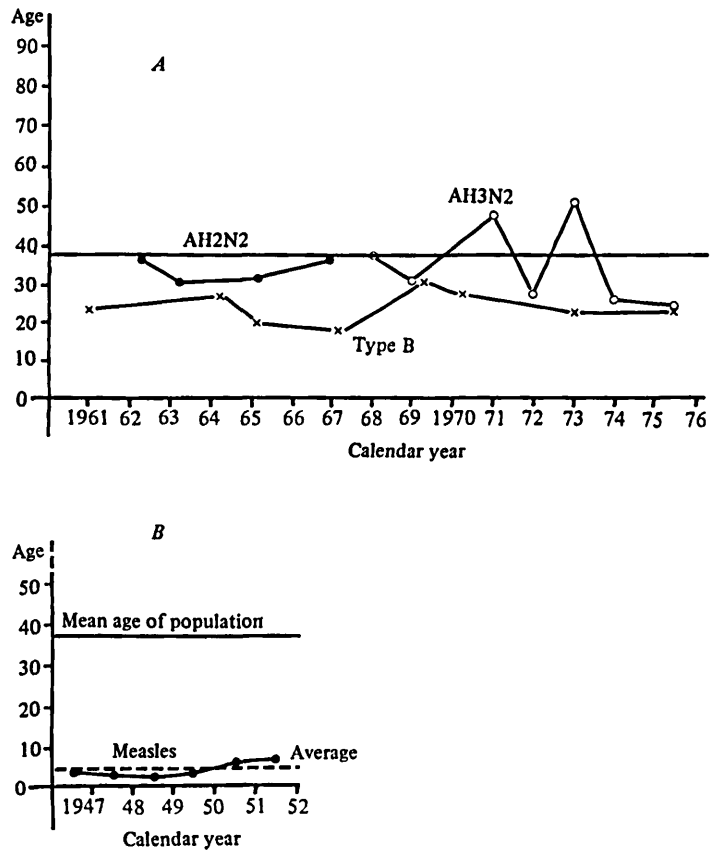

Fig. 2. A. The average age of the influenza patients in each successive epidemic: type A H2N2 ●, H3N2 ○, type B ×. The average age of the general practice population is shown. B. The average age of measles patients in successive epidemics in the same population 1947–52.

epidemics, but in the first ten days of epidemics schoolchildren and adults were almost equal in number and proportion.

Table 3 and Fig. 2 show the extent to which these wide age distributions were maintained in the successive epidemics caused by all three viruses, and how that of type B infections differed persistently from those of the type A infections. The details can be followed by reference to the Appendix tables. Table 4 and Fig. 2 also show how closely the average age of the persons attacked in the eleven succesive type A epidemics approximated to the average age of the whole general practice population, 37·56 years. The average age of all persons attacked by type A H2N2 influenza viruses was 32·8 years, that of those attacked by the H3N2 subtype was 33·4 years, whereas that of all persons attacked by influenza B virus was 23·7 years. The average age of measles patients in successive epidemics in the same population is shown in Table 4D and Fig. 2B.

Successive epidemics showed no consistent trend of change in the age patterns of persons infected by any of the three influenza viruses.

Table 4. *The average age of the persons attacked in each epidemic caused by the three influenza viruses, compared with that caused by measles in the same population*

(Average age of population 37·56 years.)

(A) Influenza A H2N2 virus

Epidemic	No.	Total age	Average age
1963	14	518	37·00
1964	16	482	30·13
1966	37	1151	31·11
1967–8	48	1620	33·75
Total	115	3711	32·79

(B) Influenza A H3N2 virus

Epidemic	No.	Total age	Average age
1968–9	78	2770	35·51
1969–70	114	3582	31·42
1971–2	54	2292	42·44
1972–3	27	724	26·81
1973–4	9	455	50·56
1974–5	33	855	25·91
1976	19	472	24·84
Total	334	11150	33·38

(C) Influenza B virus

Epidemic	No.	Total age	Average age
1961–2	28	638	22·78
1965	15	412	27·47
1966	11	219	19·91
1968	19	310	16·32
1970	12	367	30·58
1971	27	723	26·78
1973 spring	2	150	75·00
1973–4	41	870	21·22
1976	13	293	22·54
Total	168	3982	23·70

(D) Measles virus

Epidemic	No.	Total age	Average age
1947	12	73	6·1
1948	25	101	4·0
1949	16	78	4·9
1950	18	136	7·5
1951	13	89	6·8
1952	16	121	7·6
Total	100	598	6·0

DISCUSSION

When considering the interpretation of the age patterns of the persons found in this survey to be infected with influenza virus one must remember that the population studied was small and that not all cases of influenza were identified. Nevertheless the findings are thought to provide a reasonably accurate account of the distribution of influenza in that community throughout the fairly long period.

The question of the special role suggested for children as 'the major spreaders' of influenza in the community will first be considered before turning to the epidemiological implications of the patterns of infection throughout all the age groups.

Children as disseminators of type A H2N2 influenza viruses

No evidence was found that schoolchildren had been acting as major disseminators of type A H2N2 influenza virus in the general practice population. Schoolchildren were far outnumbered by adults (23:70) despite the higher incidence rate they suffered (Table 1), and they did not predominate at an early stage of any of the four epidemics (Appendix, Tables 1–4). Children too young to go to school suffered

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an incidence rate more than double that of school-aged children, and more than treble that suffered by the adult population (Table 1), yet they too were greatly outnumbered by adult cases of influenza (22:70) and they featured mainly in the middle or towards the end of the epidemics (Table 2). The ages of persons recorded as the first case(s) in each type A H2N2 influenza epidemic were: 35, 69, 50 and 60, and 64 years.

Children as disseminators of type A H3N2 influenza viruses (Appendix Tables 5–11)

Schoolchildren constituted a high proportion of the early cases in only two of the seven epidemics caused by H3N2 viruses, and were the first recorded cases in two of the other five epidemics. In 1969–70 eleven of the 30 cases recorded in the first epidemic week were in children of school age, and in the little epidemic of 1972–3 all of the six cases in schoolchildren occurred during the first week. None of the five other H3N2 influenza epidemics provided any evidence that schoolchildren were the major disseminators, school-aged patients being distributed at random throughout their course. In the two of these epidemics in which a schoolchild was the first case recorded, the evidence is against such a special role. A 15-year-old schoolboy, sent home a few days earlier to escape the influenza then attacking his residential school many miles away, developed influenza on 12 December 1968. Despite the lack of immunity to the novel H3N2 subtype in the local Cirencester population, no further case occurred in the general practice population until, more than five weeks later, a woman aged 35 years, unconnected with the schoolboy, seemed to have initiated our epidemic. The first recorded case in 1976 was in a 13-year-old schoolchild, but only four other schoolchildren were attacked and they were sparsely distributed throughout this desultory epidemic.

Adults with type A H3N2 influenza greatly outnumbered both school- and pre-school children (232:60:40). Although pre-school children suffered the highest rate of incidence their pattern of secular distribution throughout the epidemics indicated no special propensity to spread the infection. No child featured in the epidemic of 1973–4. The ages of the persons recorded as having the first case of type A H3N2 influenza in each epidemic were: 15, 19, 69, 14, 70, 50 and 13 years.

Children as disseminators of type B influenza viruses

Children suffered a much higher proportion of the type B infections than they did of the type A infections. Children with type B influenza outnumbered adults (94:74) and schoolchildren outnumbered pre-school children (68:26), although the incidence rate in the population under five years old was higher than that in the younger school-aged children and almost equal to that in all school-aged children. The highest rate was suffered by the older schoolchildren (Table 1).

Despite the relative abundance of cases in schoolchildren there is little evidence that they were acting as major disseminators of type B influenza virus in the community. They were widely dispersed throughout the duration of all the epidemics except for that of 1965, in which they all appeared in the first half of the epidemic (Table 2 and Appendix Tables 8–20).

There was no evidence that pre-school children were playing a special role in spreading the type B viruses.

The ages of persons recorded as the first case(s) in each type B influenza epidemic were: 9, 12, 58, 12, 90, 6 and 15, 70, 11 and 62 years.

The findings in this study confirm that children had suffered a higher incidence rate than adults of infections by both subtypes of type A and by type B influenza viruses, but the evidence does not support the hypothesis that either school-aged or pre-school children had been acting as 'the major disseminators' of any of these viruses in the community.

The age patterns of the adults with influenza

The two subtypes of influenza A virus and type B influenza virus all imposed rather similar age distributions in their attacks on the adults of the general practice population (Table 1). The incidence rates in adult age groupings, although lower than those in children, were nevertheless surprisingly high, and these rates were sustained even into extreme old age except for some reduction in the rate of type A H3N2 infections in persons over 70 years old, (Fig. 1).

These high incidence rates in all adult age groups were maintained in the successive epidemics (Table 3). The later epidemics of type A H3N2 influenza showed a tendency towards a lower average age not seen in successive epidemics caused by the H2N2 subtype or by type B influenza virus (Table 4 and Fig. 2). The H2N2 epidemics studied were the last four of the eight caused by H2N2 type A influenza viruses in this community.

The recycling of subtypes of type A influenza virus

The age patterns of influenza patients cannot be considered apart from the phenomenon of the reappearances of long-vanished subtypes of influenza A virus.

Sera collected from some elderly persons in 1956 and 1957 were found already to have contained antibodies to the H2N2 subtype before H1N1 viruses had been displaced worldwide by H2N2 viruses in the 'Asian' influenza pandemic of 1957. A similar phenomenon occurred in 1968, when sera from some elderly persons were found to have possessed antibody to H3N2 viruses before that subtype had appeared and displaced H2N2 viruses in 1968–9 epidemics of 'Hong Kong' influenza. It has been generally agreed that these findings indicate that both H2N2 and H3N2 subtypes of influenza A virus had had periods of world dominance in the same temporal order during the last quarter of the nineteenth century (Masurel & Marine, 1973; Davenport, 1977). Francis (1953) had suggested that influenza virus might possess so limited an antigenic repertoire that, over the years, vanished influenza viruses might be expected to reappear in cyclical fashion. Two occurrences have recently fortified Francis' hypothesis. Firstly a virus, thought to be antigenically similar to that which caused the 1918 influenza pandemic, caused an outbreak of influenza at Fort Dix, USA, in 1976 (Kendal *et al.* 1977). Secondly, in 1977 an H1N1 virus identical with the variant circulating in 1953 reappeared throughout the world, at first largely confining its attacks to persons born since 1953 (Nakajima, Desselberger & Palese, 1978).

Masurel & Hejtink (1983) have advanced serological evidence that H1N1 and H3N2 viruses circulated contemporaneously in the early years of the twentieth century as they are doing now. The age-patterns of persons attacked by the viruses of such recycled influenza A subtypes have resembled those imposed by, for

Table 5. *The age patterns of influenza attacking an ideal non-immune population of* 400 *in two successive epidemics*

(The numbers indicate each person by year of age; 0 = under one year old.)

(i) The population previous to attack contains five symptomless carriers thus **65**

31	27	2	18	53	57	81	76	52	48	25	29	6	8	49	52	76	78	53	48
7	5	92	23	32	30	8	55	29	27	8	56	63	85	54	60	74	56	55	49
60	58	24	17	12	13	9	47	7	5	0	59	63	86	28	31	51	60	31	26
42	40	20	22	36	37	11	12	81	49	22	82	41	61	30	33	57	39	34	29
18	16	1	2	44	46	47	17	58	47	27	53	45	65	7	11	30	37	10	4
72	69	82	51	45	23	20	15	56	27	1	57	20	40	5	9	32	18	8	3
67	41	38	26	23	4	93	54	33	26	63	31	19	43	2	8	9	16	6	1
43	18	16	3	1	0	71	51	32	16	62	31	15	20	0	83	7	15	3	78
46	22	12	2	28	6	69	26	12	14	40	11	36	18	69	63	4	74	1	56
20	1	3	8	31	9	65	23	10	13	43	9	30	16	67	60	85	51	92	60
17	29	7	49	53	24	39	2	7	9	19	8	9	76	45	39	65	56	70	35
0	35	9	26	5	2	37	1	78	91	17	17	6	74	48	37	67	30	74	34
2	55	31	30	37	8	15	85	55	89	31	55	4	42	25	15	43	33	48	13
25	65	33	56	16	7	14	59	51	67	29	61	3	46	24	14	44	9	52	12
27	92	69	52	14	4	9	57	27	65	8	32	58	21	29	12	23	7	27	11
49	86	67	18	44	46	78	33	24	43	6	34	57	19	4	67	25	5	27	87
54	62	43	21	26	68	75	31	3	41	4	11	33	16	3	68	3	4	6	79
75	60	43	19	37	72	43	29	0	20	28	9	37	14	1	43	3	82	5	56
81	56	31	10	17	46	40	27	77	26	49	79	12	53	68	46	2	83	3	58
61	59	33	11	15	43	18	6	83	10	73	78	9	56	59	23	0	61	1	35

(ii) Reactivation of virus latent in carriers transmits influenza to 40 contacts: *epidemic I*

31	27	2	18	53	57	81	76	52	48	25	29	6	8	49	52	76	78	53	48
7	5	92				8	55	29	27	8	56	63	85	54	60	74	56	55	49
60	58	24				9	47	7	5	0	59	63	86	28	31	51	60	31	26
42	40	20				11	12	81	49	22	82				33	57	39	34	29
18	16	1	2	44	46	47	17	58	47	27	53				11	30	37	10	4
72	69	82	51	45	23	20	15	56	27	1	57				9	32	18	8	3
67	41	38	26	23	4	93	54	33	26	63	31	19	43	2	8	9	16	6	1
43	18	16	3	1	0	71	51	32	16	62	31	15	20	0	83	7	15	3	78
46	22	12	2	28	6	69	26	12	14	40	11	36	18	69	63	4	74	1	56
20	1	3	8	31	9	65	23	10	13	43	9	30	16	67	60	85	51	92	60
17	29	7	49	53				7	9	19	8	9	76	45	39	65	56	70	35
0	35	9	26	5				78	91	17	77	6	74	48	37	67	30	14	34
2	55	31	30	37				55	89	31	55	4	42	25	15	43	33	48	13
25	65	33	56	16	7	14	59	51	67	29	61	3	46	24	14	44	9	52	12
27	92	69	52	14	4	9	57	27	65	8	32	58	21	29				27	11
49	86	67				78	33	24	43	6	34	57	19	4				27	87
54	62	43				75	31	3	41	4	11	33	16	3				6	79
75	60	43				43	29	0	20	28	9	37	14	1	43	3	82	5	56
81	56	31	10	17	46	40	27	77	26	49	79	12	53	68	46	2	83	3	58
61	59	33	11	15	43	18	6	83	10	73	78	9	56	59	23	0	61	1	35

Table 5 (*cont.*)

(iii) Next season reactivation of virus latent in 40 patients of last season's epidemic transmits influenza to 80 of their contacts: *epidemic II*

31	27						76	82	48	25	29	6	8	49	52	76	78	53	48
7	5						55	29	27	8	56	63	85	54	60	74	56	55	49
60	58						47	7	5	0						51	60	31	26
41	40						12	81	49	22						57	39	34	29
18	16						17	58	47	27						30	37	10	4
72	69	82	51	45	23	20	15	56	27	1						32	18	8	3
67	41	38	26	23	4	93	54	33	26	63						9	16	6	1
43	18	16	3	1	0	71	51	32	16	62	31	15	20	0	83	7	15	3	78
46	22	12	2	28	6	69	26	12	14	40	11	36	18	69	63	4	74	1	56
20	1	3	8						13	43	9	30	16	67	60	85	51	92	60
17	29	7	49						9	19	8	9	76	45	39	65	56	70	35
0	35	9	26						91	17	17	6	74	48	37	67	30	74	34
2	55	31	30						89	31	55	4	42	25	15	43	33	48	13
25	65	33	56						67	29	61	3	46						12
27	92						57	27	65	8	32	58	21						11
49	86						33	24	43	6	34	57	19						87
54	62						31	3	41	4	11	33	16						79
75	60						29	0	20	28	9	37	14						56
81	56						27	77	26	49	79	12	53	68	46	2	83	3	58
61	59	33	11	15	43	18	6	83	10	73	78	9	56	59	23	0	61	1	35

(iv) Age distributions of population, cases and escapes in epidemic I and cases and escapes in epidemic II to show that this proposed mechanism preserves widely age-distributed attack rates in successive epidemics and limits the spread of the influenza virus

	Population		Epidemic I				Epidemic II			
Age group*	No.	%	Cases	%	Escapes	%	Cases	%	Escapes	%
0–9	83	20·8	10	25·0	73	20·6	21	26·3	52	18·9
10–19	54	13·5	6	15·0	47	13·2	11	13·8	36	13·1
20–29	51	12·7	6	15·0	43	12·1	8	10·0	35	12·7
30–39	46	11·5	7	17·5	38	10·7	6	7·5	32	11·6
40–49	44	11·0	5	12·5	39	11·0	11	13·7	28	10·2
50–59	44	11·0	0	—	44	12·4	11	13·7	33	12·0
60–69	33	8·3	4	10·0	28	7·9	4	5·0	24	8·7
70–79	24	6·0	1	2·5	23	6·5	3	3·8	20	7·2
80–89	16	4·0	1	2·5	15	4·2	4	5·0	11	4·0
90+	5	1·2	0	—	5	1·4	1	1·2	4	1·5
Total	400	100·0	40	100·0	355+	100·0	80	100·0	275	99·9
	(5 persons are carriers)				(+5 carriers)		(+40 carriers and 5 immune)			

* Epidemic I, average age 27·8 years; epidemic II: average age 33·8 years.

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example, measles virus returning to a community after many years absence. It may therefore be instructive for some purposes to consider the whole period of world dominance of an influenza A virus subtype as if it were a single epidemic caused by an immunizing virus.

The epidemiological interpretation of the age-patterns of influenza found in this survey

The strategy whereby influenza virus secures its survival appears to differ from that of, for example, measles virus. Highly infectious immunizing viruses that, like measles virus, travel horizontally through the community by direct spread from the sick, at their first invasion attack such a large proportion of a non-immune community that the virus cannot continue to be supported by chains of transmission and becomes extinct in most communities, and recurrence must await the re-introduction of the virus from elsewhere. Only in urban populations exceeding some 250,000 persons does recruitment by births provide sufficient non-immune subjects to maintain such viruses in continuous circulation (Bartlett, 1957).

The behaviour of the first epidemic caused by influenza A H3N2 virus in 1968–9 resembled that of measles virus attacking a non-immune community, in that it attacked persons of all ages, so that the average age of those attacked approached that of the general practice population (Table 4B). The population was not, however, totally non-immune to H3N2 virus, because persons who had been attacked by H2N2 virus during the preceding 11 years of its dominance suffered a preferentially lower rate of infection during the first three H3N2 influenza A epidemics (Hope-Simpson, 1972). But the difference from the behaviour of measles virus which appears to be of great epidemiological importance lies in the much smaller numbers of the non-immune portion of the community attacked at each successive visit by influenza virus. Measles attacks such a large proportion that the community must await replenishment of susceptible subjects by births in numbers sufficient to support another measles epidemic, and subsequent measles epidemics at frequent intervals must consist almost entirely of the young. Influenza usually attacks a much smaller proportion even at the first epidemic caused by a new subtype. The low attack-rate is not due to low infectiousness, as witnessed by the high rate in institutions. Yet the first H3N2 epidemic of type A influenza attacked less than five per cent of the community studied, and the great epidemics of 1957 and of 1969–70 attacked only some 12–15 per cent. Consequently a much larger pool of susceptibles awaits second and subsequent influenza epidemics than is the case for measles. The age patterns of influenza are difficult to explain by any modification of the hypothesis of direct spread, and they suggest the existence of some strategy whereby this intensely infectious virus avoids immediate horizontal spread and ensures a pool of susceptible subjects of all ages available for epidemic after epidemic until the whole community has become immunized against the current subtype. Simultaneously a large proportion will again be ripe for infection by the return of an earlier subtype.

Any satisfactory epidemiology of influenza must answer the questions raised by these findings. Why does influenza attack so relatively few susceptible persons when its great infective capability is witnessed by the virus attacking one-sixth of this community in six weeks in 1957 and again in 1969? How in the early months

Table 6. *Model of measles attacking the same ideal population after an absence of 60 years and then returning after a further absence of nine years. Bold figures indicate immune persons*

Age groups* (10-year)	(i) Population no.	(ii) Epidemic I		(iii) Epidemic II		(iv) Epidemic II Single years of age			
		Cases	Escapes	Cases	Escapes	Age	Cases	Escapes	Population
0–9	83	83	0	46	25+12	0	2	4	6
10–19	54	54	0	0	**54**	1	2	7	9
20–29	51	51	0	0	**51**	2	4	4	8
30–39	46	46	0	0	**46**	3	5	5	10
40–49	44	44	0	0	**44**	4	5	3	8
50–59	44	43	1	0	1+**43**	5	6	0	6
60–69	**33**	0	**33**	0	**34**	6	7	0	7
70–79	**24**	0	**24**	0	**23**	7	8	0	8
80–89	**16**	0	**16**	0	**16**	8	7	2	9
90+	**5**	0	**5**	0	**5**	9–10	0	**12**	**12**
Total	400	321	1	46	26		46	25	83
Immunes:	78		78		328			12	12

(i) Distribution of population in 10-year age groups.
(ii) Cases and escapes in first epidemic. Almost everybody under 60 years old is attacked.
(iii) Cases and escapes in second epidemic. The cases are confined to persons in the youngest age group.
(iv) Analysis of the youngest age group by single years of age, to show that even in this group some susceptible children are escaping.
* Average age: epidemic I, 26·3 years; epidemic II, 5·7 years.

Table 7. *The average age of persons attacked in the model epidemics*

(A) Influenza model

Epidemic	No.	Total age	Average age
I	40	1180	29·5
II	80	2700	33·8

(B) Measles model

Epidemic	No.	Total age	Average age
I	321	8455	26·3
II	46	260	5·7

of 1968 did H2N2 influenza A virus contrive to mount a large eighth epidemic only eleven years after its first epidemic in 1957, and in that last epidemic how did it succeed in attacking persons in all age groups so that the average age of those attacked was over 35 years (Table 4)? Second attacks by the virus numbered less than two per cent, and no second attack was encountered in older persons.

The questions are related, and the answer must be that the epidemic mechanisms by which influenza viruses survive as species differ from those of measles in a manner which prohibits 'horizontal' invasion of a non-immune community in successive epidemics and largely preserves the age structure of those who escape attacks.

The alternative hypothesis to that of direct spread proposes that epidemics can only arise in populations already widely seeded with carriers of latent residues of influenza virus. Seasonal reactivation would produce epidemics consisting mostly of small foci of cases of influenza grouped around the symptomless carriers shedding reactivated virus. These cases would constitute the whole epidemic because, by definition, there could be no horizontal spread from them.

Would such a 'vertical' epidemic mechanism produce the findings reported in this paper, in which epidemic after epidemic at short intervals each attacked a slice of the whole age range of the community, thus securing that the whole community ultimately became immunized, each age group having participated in each step of the stepwise procedure?

Models of epidemic mechanisms

Models may assist in the difficult task of understanding the effects of different epidemic processes upon the age patterns of those attacked in a community invaded by an infectious disease. Table 5(i–iv), is designed to demonstrate the behaviour of the epidemic mechanism proposed as an alternative to that of direct spread. Table 5(i), is a diagram in which the numbers represent years of age of an ideal community of 400 persons so arranged that households of various common compositions and relationships stand beside one another. Five of these persons are symptomless carriers of influenza virus. The age structure is summarized in Table 5(iv). In Table 5(ii), the latent influenza virus residues have reactivated to infectiousness in the five symptomless carriers in this otherwise non-immune community, causing 40 persons to catch influenza from them. The ages of all those

Table 8. *The age distribution of cases of influenza and measles in the general practice population (A) compared with proposed epidemic mechanism of influenza in model population and direct spread of measles in model population (B)*

(The table illustrates how the proposed epidemic mechanism allows a wide distribution of influenza throughout all age groups in successive epidemics, as found in the general practice observations, in contrast to the distribution of measles cases which depend on direct spread of the virus.)

(A) *Percentage distribution by 10-year age group of general practice population of virus-positive influenza cases and of measles cases*

Age group	General practice population	Influenza cases			Measles cases
		A H2N2	A H3N2	B	
0–9	16·6	26·1	22·2	31·6	97·2
10–19	13·3	13·9	13·8	31·5	2·0
20–29	13·7	11·3	12·9	6·6	0·2
30–39	11·1	12·2	9·3	6·5	0·2
40–49	11·5	5·2	12·3	9·5	0·2
50–59	12·3	13·0	11·7	3·6	0
60–69	11·0	9·6	11·1	4·8	0
70+	10·6	8·7	6·9	6·0	0
Total	100·1	100·0	100·2	100·1	99·8

(B) *Percentage distribution by 10-year age-group of model population (see Table 5) of two successive epidemics of influenza by proposed epidemic mechanism (Table 5), contrasted with those of two epidemics of measles spreading directly from the sick (Table 6): Epidemic I after 60 years absence, epidemic II returning after nine years*

Age group	Model population	Influenza		Measles	
		Epidemic I	Epidemic II	Epidemic I	Epidemic II
0–9	20·8	25·0	26·3	25·8	100·0
10–19	13·5	15·0	13·8	16·8	0
20–29	12·7	15·0	10·0	15·8	0
30–39	11·5	17·5	7·5	14·3	0
40–49	11·0	12·5	13·7	13·7	0
50–59	11·0	0	13·7	13·7	0
60–69	8·3	10·0	5·0	0	0
70+	11·2	5·0	10·0	0	0
Total	100·0	100·0	100·0	100·1	100·0

attacked and spared in this first epidemic are shown in Table 5 (iv). In Table 5 (iii), a second epidemic in the subsequent season is seen developing around last season's cases, now become carriers of reactivating virus. Again the analyses of the ages of those attacked and spared in this second epidemic are given in Table 5 (iv).

The proposed epidemic mechanism is evidently performing the two functions of limiting the immediate wide distribution of the virus in a community highly favourable for direct spread, and of preserving the age structure of those unattacked so that epidemic after epidemic may affect persons in all age groups. The second epidemic outnumbered the first, and in a more authentic three-dimensional model the expansion would have been much greater. This expanding tendency has been noted in field studies of influenza (Glezen, Couch & Six, 1982).

It does not, however, continue in successive epidemics because the populations in the neighbourhood of the reactivating carriers become relatively heavily immunized and so less favourable to the virus. Mobility of the population, as in 1918, would allow the virus to elude this inbuilt safeguard. The rate of the initial reactivating carriers (1·25%) chosen for the model in Table 5(ii) is unrealistically high.

For contrast Table 6 shows the same ideal community attacked by a single introduction of measles after 60 years absence of the disease during which nobody has been vaccinated against measles. The direct transmissions secure that almost everybody under 60 years old is attacked in a great wave of horizontal spread (Table 6(ii)). Nine years later (Table 6(iii)) sufficient children have been born for the community to support a second outbreak, but now the community is so full of immune persons that direct spread is not so easy for the virus and a proportion of pre-school children escapes (see Table 6(iv)). Thereafter measles will return every few years attacking almost entirely schoolchildren and some of their younger siblings, the day school now being the most favourable environment for direct transmissions.

Table 7 contrasts the high average age of the second model epidemic of influenza, spreading by the alternative epidemic mechanism, with the low average age of the second model measles epidemic, spreading directly from the sick.

The models illustrate how the age pattern of persons attacked by the mechanism of horizontal spread must differ from that caused by a mechanism of latency and seasonal reactivation of the virus, and how the model of the latter accords with the age patterns found in the successive epidemics recorded in this paper (Table 8).

The evidence provided in this paper is independent of that provided from other aspects of influenzal behaviour in earlier papers.

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APPENDIX

Appendix tables A1–A20 giving dates, day-in-epidemic and age of patients in each epidemic caused by each virus.

Table A1. *Influenza A H2N2 virus*, 1963 *epidemic*

Date March	Day	Age
1	1	35
4	4	32 38
5	5	3
6	6	29 30 78
11	11	39
13	13	22 25
16	16	63
25	25	9
27	27	60
April		
2	33	55
School-aged		1
Pre-school		1
Adults		12
Total		14

Bold figures indicate schoolchild.

Table A2. *Influenza A H2N2 virus*, 1964 *epidemic*

Date January	Day	Age
26	1	69
February		
9	15	35
19	25	13 47
20	26	8 months
24	30	13
25	31	16 42 48
28	34	58
March		
3	37	3 33
9	44	10 15
16	51	59
20	55	21
School-aged		4
Pre-school		2
Adults		10
Total		16

Bold figures indicate schoolchild.

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Table A3. *Influenza A H2N2 virus*, 1966 *epidemic*

Date		
January	Day	Age
3	1	50 60
13	11	**15**
15	13	80
17	15	2
21	19	26 28 33
24	22	63
26	24	4
27	25	38
February		
1	30	60
3	32	2 2
4	33	2
5	34	**13**
7	36	**13** 33
9	38	59
10	39	63
11	40	5 months
16	45	2 **10** 20
18	47	**5 12** 37 54
21	50	1 **13** 25 47
23	52	**11** 40 70
25	54	50
March		
7	64	63
School-aged		8
Pre-school		8
Adults		21
Total		37

Bold figures indicate schoolchild.

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Table A4. *Influenza A H2N2 virus*, 1967–8 *epidemic*

Date		
December	Day	Age
8	1	64
11	4	73
January		
1	25	51 **53**
2	26	4 38 75
3	27	20 24 66 90 95
4	28	3 **8**
5	29	**5**
8	32	24 82
10	34	52
15	39	**15**
16	40	59
19	43	24
24	48	4 42 52
25	49	10 months 8
26	50	3 **7** 72
29	53	**6 10 14**
30	54	61
31	55	2 55
February		
2	57	**12**
6	61	**7** 36
7	62	9 months 74
8	63	50
12	67	11 months
13	68	4
20	75	1 33
March		
1	85	3 24
5	89	58

School-aged	10
Pre-school	11
Adults	27
Total	48

Bold figures indicate schoolchild.

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Table A5. *Influenza A H3N2 virus*, 1968–9 *epidemic*

Date			Date		
December	Day	Age	March	Day	Age
12	**1**	**15**	3	82	16 20 24 47 57 59
January			10	89	53
17	37	35	12	91	35
20	40	20	15	94	18 25
23	43	40 41	17	96	62
24	44	57	18	97	32
27	47	21 42 46	19	98	51 54
28	48	31	20	99	40 55
29	49	63	21	100	1 60
February			28	107	44
1	**52**	**6**	April		
5	56	18 43	3	113	96
6	57	45	8	118	62
10	61	66	9	119	33 37 63 71
11	62	45	10	120	10 months
12	63	**6** 51	11	121	**11**
13	64	20 39 52	15	125	**1 8**
15	66	60		School-aged	12
17	68	**12** 56		Pre-school	4
18	69	**5** 17 18 48		Adults	62
19	70	29		Total	78
21	72	**13** 29 45 64			
22	73	39 39 42			
24	75	**8** 12 28			
25	76	21 46 46			
26	77	**11** 22			
27	78	**3** 9 49			
28	79	61			

Bold figures indicate schoolchild.

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Table A6. *Influenza A H3N2 virus*, 1969–70 *epidemic*

Date December	Day	Age
5	1	19
8	4	**5** **6** 18 24 32 41 47 51 52
9	5	**10** **10** 39 73
10	6	**5** **5** **6** 24 24 26 42 47
11	7	**5** **8** **9** **12** 35 72
12	8	**8** **10** 30 39
13	9	31 59 63 69
15	11	**5** **14** 17 39 52 52 53
16	12	4 months 2 2 **6** **6** **6** **7** 29 34 39 49 76
17	13	**10** 19 20 33 58 67
19	15	3 **6** 19 28 29 32 44 47 56 58 60 65
20	16	1 44
22	18	2 4 4 **5** **6** **8** **9** 24 25 58
23	19	3 42 52 52 60 69
24	20	51
27	23	61 64
29	25	1 2 22 22 49 60 68
30	26	43 64 64 67
31	27	22 23
January		
1	28	2 51 61
2	29	62
5	32	**13**
6	33	43
7	34	10 months

School-aged	26
Pre-school	13
Adults	75
Total	114

Bold figures indicate schoolchild.

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Table A7. *Influenza A H3N2 virus*, 1971–2 *epidemic*

Date		
December	Day	Age
6	1	69
7	2	**9**
10	5	50
11	6	43
13	8	54 58 80
14	9	32 58 65 68
17	12	**6**
18	13	81
20	15	16
21	16	4 40 77 87
23	18	62 48
24	19	2
27	22	**11**
29	24	47
30	25	5 months 20 81
31	26	4
January		
4	30	**5** 68
6	32	65 67
7	33	**6** 30 33 74 77
10	36	4 months 53
15	41	51
18	44	57
20	46	46 50
24	50	16 18 63
28	54	58
February		
4	61	35 36 56
7	64	63
10	67	44
16	73	19
18	75	27
21	78	3

School-aged	5
Pre-school	6
Adults	43
Total	54

Bold figures indicate schoolchild.

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Table A8. *Influenza A H3N2 virus*, 1972–3 *epidemic*

Date		
December	Day	Age
18	1	**14**
19	2	32
20	3	**11**
21	4	**12**
22	5	**14** 17
23	6	**11**
24	7	**13** 20 30
26	9	49
27	10	18 20 22 46 68 72 72
28	11	8 months 2
January		
3	17	67
5	19	49
7	21	31
22	36	29
February		
12	57	5 months 3
13	58	8 months

School-aged	6
Pre-school	5
Adults	16
Total	27

Bold figures indicate schoolchild.

Table A9. *Influenza A H3N2 virus*, 1973–4 *epidemic*

Date		
December	Day	Age
28	1	77
January		
12	16	30
March		
13	76	82
20	83	29
April		
3	97	16
4	98	21
5	99	71 74
9	103	55

School-aged	0
Pre-school	0
Adults	9
Total	9

Table A10. *Influenza A H3N2 virus*, 1974–5 *epidemic*

Date		
December	Day	Age
2	1	50
January		
20	52	**12**
21	53	32
27	59	3 **9 11** 51
28	60	**5** 53 70
30	62	21 42
February		
4	67	24
7	70	**5** 28
8	71	75
10	73	3 **13**
11	74	4
14	77	23
15	78	10 months
17	80	80
18	81	48
22	85	4
24	87	1
March		
4	98	19 59
10	101	**13**
11	102	62
18	109	22
28	119	3
April		
2	124	2 7
School-aged		8
Pre-school		8
Adults		17
Total		33

Bold figures indicate schoolchild.

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Table A11. *Influenza A H3N2 virus*, 1976 *epidemic*

Date		
January	Day	Age
1	1	**13**
2	2	22
5	5	78
14	14	4
February		
3	34	**10**
11	42	71
13	44	38 42
17	48	**12**
23	54	4 **6**
25	56	4 months 24
March		
2	62	29
9	69	**15**
10	70	47
12	72	28
15	75	29
25	85	2 months
School-aged		5
Pre-school		4
Adults		10
Total		19

Bold figures indicate schoolchild.

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Table A 12. *Influenza B virus*, 1961–2 *epidemic*

Date		
December	Day	Age
15	1	**9**
23	9	**11**
24	10	**15**
27	13	47
29	15	**9** 18 20
January		
1	18	**5**
3	20	1
6	23	24
9	26	**12** 23
10	27	**13**
11	28	84
12	29	3
13	30	**14** 42
15	32	27
16	33	73
18	35	3 60
27	44	**10**
February		
5	53	54
7	55	**15**
26	64	19
27	65	3 16
April		
10	76	**8**

School-aged	11
Pre-school	4
Adults	13
Total	28

Bold figures indicate schoolchild.

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Table A 13. *Influenza B virus*, 1965 *epidemic*

Date		
February	Day	Age
2	1	**12**
8	7	**10**
22	21	**11**
27	26	**39**
March		
1	28	**7** 38
3	30	**13** 48
4	31	**17**
12	37	**14**
15	40	**1**
April		
22	78	28
26	82	22
29	85	41
May		
17	103	48

School-aged	6
Pre-school	1
Adults	8
Total	15

Bold figures indicate schoolchild.

Table A 14. *Influenza B virus*, 1966 *epidemic*

Date		
April	Day	Age
12	1	58
13	2	2 73
14	3	14 30
15	4	4 months 2 2
18	7	1 7 30

School-aged	2
Pre-school	5
Adults	4
Total	11

Bold figures indicate schoolchild.

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Table A 15. *Influenza B virus*, 1968 *epidemic*

Date		
February	Day	Age
27	1	**12**
29	3	3 21
March		
1	4	3
5	8	**11** 39
6	9	48
11	14	**12 13 13**
13	16	4 **6** 17
19	22	29
25	28	**6**
28	31	2 44
April		
8	42	25
9	43	2

School-aged	7
Pre-school	5
Adults	7
Total	19

Bold figures indicate schoolchild.

Table A 16. *Influenza B virus*, 1970 *epidemic*

Date		
April	Day	Age
14	1	90
21	8	**6**
24	11	**13**
May		
4	21	4 43
6	23	**11 14**
11	28	30 65
15	32	2
21	38	3
22	39	86

School-aged	4
Pre-school	3
Adults	5
Total	12

Bold figures indicate schoolchild.

Table A 17. *Influenza B virus*, 1971 *epidemic*

Date			
January	Day		Age
22	1	**6 15**	
25	4	4	
February			
5	15	66	
8	18	**12** 47	
25	35	**7**	
March			
8	46	41 **41**	
9	47	66	
16	54	17	
22	60	**12 13**	
23	61	11 **12**	
24	62	**5**	
26	64	2 16 17 31 53 58	
29	67	48	
31	69	17 **76**	
May			
10	119	16	
12	121	**14**	

School-aged	10
Pre-school	2
Adults	15
Total	27

Bold figures indicate schoolchild.

Table A 18. *Influenza B virus* 1973 *epidemic*

Date		
May	Day	Age
1	1	70
7	7	80

School-aged	0
Pre-school	0
Adults	2
Total	2

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Table A 19. *Influenza B virus*, 1973–4 *epidemic*

Date		
October	Day	Age
12	1	**11**
December		
21	71	16
23	73	16 77
28	78	**9 9 10 14** 34
January		
1	82	**7**
4	85	**6** 32 42 49
10	91	53
14	95	**11**
15	96	**15**
18	99	56
20	101	1
21	102	**6**
25	106	4 **7 7 8**
February		
4	116	**7** 26 26
5	117	4 36 42
6	118	4
13	125	**9**
15	127	**7**
21	133	2
26	138	**8**
March		
2	142	**13**
4	144	64
7	147	2
12	152	84
18	158	21
19	159	**15**

School-aged	19
Pre-school	6
Adults	16
Total	41

Bold figures indicate schoolchild.

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Table A20. *Influenza B virus*, 1976 *epidemic*

Date		
January	Day	Age
1	1	62
23	23	**7**
February		
9	40	68
11	42	**11**
13	44	**14**
17	48	**10**
24	55	**10 11** 30
25	56	45
March		
1	60	**11**
8	68	**7 7**

School-aged	9
Pre-school	0
Adults	4
Total	13

Bold figures indicate schoolchild.

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