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A study of acute respiratory disease in the community of Port Chalmers I. Illnesses within a group of selected families and the relative incidence of respiratory pathogens in the whole community

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SUMMARY

A study of respiratory diseases in the semi-isolated community of Port Chalmers, New Zealand, began in April 1973. The intensive surveillance of a selected group of 26 families involved the weekly reporting of illness, the collection of specimens for virus, Group A streptococci and *Mycoplasma pneumoniae* isolation and the collection of sera at 6-month intervals. A total of 956 illnesses were reported during 32 months. The median number of illnesses per year were: infants 4·4, children 2·5, female adults 2·4 and male adults 2·0. Of all these illnesses, 57% were upper respiratory, 31% were lower respiratory and 9% were enteric. The severity of these illnesses was not greater than would be expected in open communities. Surveillance by pathogen isolation only of the whole community through the patients in the general practice was carried out concurrently.

A total of 640 nasopharyngeal swab specimens were collected from which 161 viruses, 47 Group A streptococci and 2 *M. pneumoniae* were isolated. The overall isolation rate was 33%. The similarities between the epidemiological patterns of respiratory disease in the open community and the isolated community are discussed.

INTRODUCTION

The pattern of respiratory disease in the isolated community is one of explosive outbreaks following contact with the outside world, with all or nearly all the community being affected (Paul & Freese, 1933; Shibli *et al.* 1971). The degree of severity of these outbreaks is dependent on the 'antigenic virginity' or, indirectly, on the degree of isolation of the population (Paul & Freese, 1933). Between

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epidemics sporadic and mild infections may occur caused by endemic viruses, possibly latent within the community (Banker & Melnick, 1957; Miles *et al.* 1973). In contrast, respiratory pathogens in open communities may cause repeated waves of infections, often with more than one pathogen concurrently (Monto & Cavallaro, 1971; Hope-Simpson & Higgins, 1969).

Observations on the aetiology of acute respiratory disease in the small community of Port Chalmers indicated that, over a period, the epidemiological pattern was one of sequentially dominant viral pathogens. We have been investigating the pathogens involved, their periodicity, rate of spread and attack rates, by the intensive surveillance of a group of selected families, and concurrently, the surveillance of the whole community through patients in a general practice. This paper presents the results obtained during the 32 months of study between April 1973 and November 1975.

MATERIALS AND METHODS

Port Chalmers

Port Chalmers, with its surrounding rural area, has a total practice size of 3500 people served by two general practitioners. It lies on Otago Harbour, 14 km from the city of Dunedin.

Family recruitment

For the purpose of the study, a family was defined as a group of related people living in the same household. Families were chosen if they contained at least one school-aged child and were likely to be permanently resident in the area for the duration of the study.

Following recruitment, details about the family were recorded and an illness-reporting scheme initiated. The report sheet used is shown in Fig. 1 and is similar to that used by Monto & Cavallaro (1971). The sheet was to be completed on the Sunday ending each week for each member of the family in whom an illness had occurred. The mother was responsible for the reporting and was asked to include all illnesses for which symptoms were listed on the report sheet. She was also asked to notify all respiratory illness within 3 days of onset by telephoning the laboratory. A personal visit was made to each family during the first week of each month, at which time completed sheets were collected and any illness checked with the mother. All family information and illness data collected was coded and transferred onto punch cards for subsequent computer analysis.

Classification of illness

Illnesses were classified into the following categories: (i) respiratory below the larynx (LR); (ii) respiratory in the larynx and above (UR); (iii) enteric and (iv) 'other'. The differentiation between illnesses occurring close together in the same person was made by calling the illness 'new' if there was at least one day without symptoms before the onset of the next (Hope-Simpson, 1958). In addition, if there was a major change in symptoms, this was recorded as a new event.

Collection of specimens from the families

Specimens for pathogen isolation were collected within 3 days of onset when an illness was notified by telephone, discovered during a monthly visit or through contact with the general practitioner. Both throat and nasal swabs were taken, the cotton swab heads being broken off into virus transport medium: 0·1 % gelatine in Hanks' balanced salt solution (VTM). A second throat swab was also taken and the head broken off into Stuart's bacteriological transport medium. The specimens, if taken by the practitioner, were stored at 4 °C over-night. Otherwise they were transported directly to the laboratory and inoculated without prior freezing into culture media.

Blood specimens for serology were obtained routinely by venepuncture from all family members willing to be bled. The initial sample was obtained soon after recruitment, the majority being collected by May 1973, and at approximately 6-month intervals thereafter. Serum from each specimen was separated and stored at −20 °C until tested.

Collection of specimens from the community

Illness in the general community of Port Chalmers was investigated by collecting throat and nasal specimens from patients who visited the general practitioner or were visited by him. It was planned to investigate a minimum of six patients each week.

Collection of control specimens

Nose and throat swab specimens were obtained from individuals visiting the general practitioner for reasons other than an acute respiratory illness. These specimens were classed as 'normal'.

Virus isolation and identification

Primary cynomolgous monkey kidney (CMK) cell cultures were obtained from the Commonwealth Serum Laboratories, Melbourne, Australia. Human embryo lung (HEL-86, a semi-continuous strain), monkey embryo kidney (Mek-3) and HeLa cell lines were obtained from Dr I. D. Gust, Fairfield Infectious Diseases Hospital, Melbourne, Australia. CMK cell cultures were used routinely throughout the study. Vero and HEp-2 cell cultures were used in 1973 then replaced with Mek-3 and HeLa cells. HEL cell cultures were used when available in 1973, then routinely during the rest of the study.

The VTM specimens were inoculated in 0·2 ml amounts into one tube of each available cell culture. The tubes were incubated at 33 °C in a roller drum and observed regularly for 21 days. If degeneration occurred a blind passage was carried out by transferring 0·2 ml of dislodged cells and medium into a fresh cell culture tube. CMK cell cultures were tested for the presence of haemadsorbing viruses after 10 days and again before being discarded using 0·5 % freshly washed guinea-pig red blood cells (RBC's). Haemadsorbing viruses were identified by haemadsorption-inhibition (Grist *et al.* 1966) using reference antisera to

parainfluenza virus type 1, 2 and 3 and mumps virus supplied by the National Institute of Allergy and Infectious Diseases, U.S.A. and simian myxovirus (SV-5) prepared by Microbiological Associates Inc. Viruses also showing haemagglutination were identified by haemagglutination-inhibition using the microtitre technique, 0·5% freshly washed fowl RBC's and reference antisera to current influenza A and B virus strains supplied by the World Health Organization (Lennette & Schmidt, 1969). Reference sera were treated with receptor-destroying enzyme. Cytopathogenic viruses not showing haemadsorption were identified by neutralization or complement fixation tests (Grist *et al.* 1966), while the rhinoviruses were distinguished from other picornaviruses by their inactivation at pH3 (Lennette & Schmidt, 1969).

Specimens from influenza-like illnesses were inoculated amniotically and allantoically into 10-day-old embryonated hen's eggs and incubated at 37 °C for 48 h. Amniotic and allantoic fluids were then tested for the presence of haemagglutinating viruses. Pools of ten VTM specimens were prepared, each pool being inoculated intracranially and subcutaneously into a litter of unweaned mice. Mice were examined daily for 14 days. If paralysis or death occurred, each specimen within the pool was then inoculated into litters of unweaned mice. Virus identification was made by neutralization or complement fixation tests.

Each VTM specimen was also inoculated into diphasic medium for the isolation of *Mycoplasma pneumoniae* (Smith *et al.* 1967). Cultures were incubated at 37 °C for 4 weeks and if growth was suspected they were subcultured onto mycoplasma agar. Strains were identified by growth inhibition using filter paper disks saturated with antiserum (Microbiological Associates Inc.).

Bacteriological isolation and identification

Each specimen in Stuart's transport medium was streaked onto a human cell blood agar plate. The plates were incubated at 37 °C in 5% CO_2 in air. Colonies showing β-haemolysis were subcultured and identified as Lancefield Group A streptococci by the bacitracin disk test (Maxted, 1953).

Serology

Titration of serum samples for the presence of complement fixing (CF) antibody to common respiratory pathogens was carried out by the method of Bradstreet & Taylor (1962) adapted to the microtitre technique. Sera were heat inactivated (56 °C for 30 min) and tested at an initial 1/8 dilution. Antigens and reference sera were supplied by Microbiological Associates Inc.

Meteorological information

Air temperature and humidity recordings using a 'Casella' recording thermohydrograph were taken throughout the study at a central point in Port Chalmers.

Data analysis

A Burroughs 6700 computer was used to analyse the data.

Table 1. *Size of families under surveillance*

Size of family	No. of families	% of total
3	3	11·5
4	8	30·8
5	7	26·9
6	5	19·2
7	3	11·5

Table 2. *Distribution by age of family members under surveillance on indicated dates*

	No. of members		
Age group (years)	1 July 1973	1 July 1974	1 July 1975
< 1	1	—	1
1–4	18	14	7
5–9	33	26	29
10–14	15	23	22
15–20	6	9	13
Mothers	26	26	25
Fathers	23	23	23
Grandparents	5	4	4
Total	127	125	124

Table 3. *Socio-economic status of families as indicated by the occupation and educational attainment of the heads of the households*

Occupational classification	No.	New Zealand Educational level	No.
Professional	2	University graduate	1
Manager	3	Technical qualification achieved	14
Farmer	2	University Entrance exam. passed	2
Office worker	2	School Certificate exam. passed	1
Skilled worker	8	Less than School Certificate	8
Semi-skilled worker	7		
Unskilled worker	2		
Total	26		26

RESULTS

Family characteristics

Recruitment of the families began during the first week of April 1973 and, after 5 months, 26 families comprising a total of 127 members had been placed under surveillance. Four subjects were lost during the study and one was added by a birth in late 1975. The sizes of the families under surveillance is shown in Table 1. The mean number of 4·9 members in each family is above the national average of 3·9 (New Zealand Year Book, 1973). The distribution of all members by age is shown in Table 2. The socio-economic status of the families was determined by means of the occupation and educational attainment of the head of each household (Table 3). Most of the families (18) lived in detached houses of 1000–1500 sq.ft,

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Table 4. *Distribution of families and family members under surveillance by reported morbidity*

No. of illnesses per person per year	No. of*				
	Families	Male adults	Female adults	Children 5–20 years	Infants 0–4 years
0–0·9	0	6	5	6	0
1–1·9	6	8	7	14	2
2–2·9	8	5	7	13	2
3–3·9	6	3	5	12	2
4–4·9	4	1	1	7	1
5–5·9	0	1	3	3	1
6–6·9	2	0	0	2	2
7–7·9	0	0	1	1	2
8–8·9	0	0	1	1	1
Totals	26	24	30	59	13
Mean no. in each year					
1973		2·4	3·0	3·2	4·6
1974		2·0	2·3	2·5	4·6
1975		1·7	1·9	1·9	4·0
Median no.	3·0	2·0	2·4	2·5	4·4

* The average number per year calculated over the whole study period.

Table 5. *Illness distribution by age in families under surveillance*

Age group	Total no. of illnesses	No. and % in each illness category				
		Respiratory				
		Lower	Upper	Total	Enteric	Other
0–4	173	51 (29)	103 (60)	(89)	12 (7)	7 (4)
5–20	439	95 (22)	285 (65)	(87)	44 (10)	15 (3)
Female adults	205	83 (40)	96 (47)	(87)	20 (10)	6 (3)
Male adults	139	63 (45)	61 (44)	(89)	13 (9)	2 (1)
Total	956	292 (31)	545 (57)	(88)	89 (9)	30 (3)

Figures in parentheses are percentages.

while 6 families were in large (> 1500 sq.ft.) and 2 families were in small (< 1000 sq.ft.) detached houses.

Morbidity

A total of 956 illnesses were reported by the families during 32 months under surveillance. Morbidity within the families and the average number of illnesses per year by age groups is given in Table 4.

The number of illnesses per person per year, calculated on a family basis, ranged from 1·0 to 6·0 with a median of 3·0. The largest variation was among children over 5 years (0–8 episodes per year) and was least among male adults (0–5 episodes per year). By comparing the mean number of illnesses reported by

Table 6. *The severity of illness in each category and age group as indicated by fever, malaise and restriction in usual activities*

	No. (%) in each illness category					No. (%) in each age group (years)			
	LR	UR	Enteric	Other	All Illness	0–4	5–20	Adults	All ages
Fever	70 (24)	98 (18)	11 (12)	15 (50)	194 (20)	50 (29)	92 (21)	52 (15)	194 (20)
Malaise	91 (31)	131 (24)	29 (33)	16 (52)	267 (28)	35 (20)	167 (38)	72 (21)	274 (29)
Restricted activity	128 (44)	174 (32)	45 (51)	19 (64)	366 (38)	54 (31)	224 (51)	96 (28)	374 (39)
Total no. of illnesses	292	545	89	30	956	173	439	344	956

each age group in 1973 with the mean number in 1975, there was a considerable decrease among children over 5 years and female adults, a smaller decrease among male adults and almost no decrease among infants.

The distribution of illnesses by age and clinical category is shown in Table 5 The majority of illnesses were respiratory (88%), and only 9% were enteric Infants had a slightly higher proportion of lower respiratory illness than school-aged children, but an unexpected result was that the adults had twice as many lower respiratory illnesses as infants and children. Analysis of the symptoms associated with the lower respiratory category of illness revealed that a productive cough occurred in 86% of adult illnesses, while associated with only 63% of infant and childhood illnesses.

Severity of symptoms

Because most reports were subjective assessments by the mother, accurate grading of the severity of the illness reported was not possible. Some analysis of severity has been attempted using the following criteria:

(1) fever (mother's assessment),

(2) malaise (an illness requiring bed rest),

(3) restriction of normal activity (an illness requiring time off work or school).

The results of this analysis by illness and category and age group are shown in Table 6. As expected, LR illnesses were more severe than UR illnesses. The duration of illness confirmed this; LR illnesses had a median duration of 8·2 days and UR illnesses, 5·8 days. Enteric illnesses were severe in terms of restricted activity and malaise, while their median duration was only 1·7 days. 'Other' illnesses, over one third of which were clinical measles or mumps, had the highest proportion of all criteria, while the median duration of illness was 5·0 days. There was an inverse relationship between age and the presence of fever.

Pathogen isolations

A total of 210 pathogens were isolated from 640 nasopharyngeal swab specimens from all illness categories. Of these, 161 (77%) were viruses, 47 (22%) Group A streptococci, and 2 (1%) *Mycoplasma pneumoniae*. The total isolation rate was

Table 7. *Association of isolates with illness category, age distribution of subjects, and the total number of specimens collected*

Pathogen	Total no. of isolates	Number (%) of isolates: Per illness category				Per age group			
		LR	UR	Other	Normal	0–4	5–20	21–49	50+
Influenza A	53	35 (66)	18 (34)	—	—	4 (7)	23 (43)	23 (43)	3 (6)
Influenza B	17	1 (6)	16 (94)	—	—	1 (6)	10 (59)	5 (29)	1 (6)
Parainfluenza 1	3	1 (33)	2 (67)	—	—	—	3 (100)	—	—
Parainfluenza 2	11	1 (9)	10 (91)	—	—	2 (18)	4 (36)	4 (36)	1 (9)
Parainfluenza 3	10	6 (60)	4 (40)	—	—	3 (30)	5 (50)	1 (10)	1 (10)
RSV	2	2 (100)	—	—	—	1 (50)	1 (50)	—	—
Rhinovirus	54	10 (18)	43 (80)	—	1 (2)	9 (17)	21 (39)	20 (37)	4 (7)
Enterovirus	5	1 (20)	4 (80)	—	—	1 (20)	4 (80)	—	—
Mumps virus	2	—	—	2 (100)	—	—	2 (100)	—	—
Herpes virus	4	2 (50)	2 (50)	—	—	1 (25)	1 (25)	1 (25)	1 (25)
GpA streptococcus	47	7 (15)	40 (85)	—	—	8 (17)	19 (40)	16 (34)	4 (9)
M. pneumoniae	2	—	2 (100)	—	—	—	—	2 (100)	—
Total	210	66 (31)	141 (67)	2 (1)	1 (0·5)	30 (14)	93 (44)	72 (34)	15 (7)
Total no. of specimens	640	95 (15)	509 (80)	13 (2)	23 (3)	70 (11)	243 (38)	262 (41)	64 (10)

Table 8. *Months in which respiratory pathogens were isolated*

	A	M	J	J	A	S	O	N	D	J	F	M	A	M	J	J	A	S	O	N	D	J	F	M	A	M	J	J	A	S	O	N
Influenza A	—	—	—	—	—	6	34	1	—	—	—	—	—	—	—	3	1	1	—	—	—	—	—	—	—	—	—	3	5	—	1
Influenza B	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2	10	5
Parainfluenza type 1	—	—	1	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—
Parainfluenza type 2	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2	7	2	—	—	—	—	—	—	—	—	—	—	—	—	—
Parainfluenza type 3	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	3	5	2	—	—
RSV	—	—	—	—	—	—	1	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Rhinovirus	—	—	—	—	—	—	—	—	—	—	2	10	15	13	1	—	—	—	—	—	—	1	1	—	1	3	3	2	2	2
Enterovirus	—	—	—	—	—	—	—	—	1	3	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1
Mumps	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	1	—	—	—
Herpes virus	—	1	—	—	—	—	—	—	—	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	1	—
GPA streptococcus	3	—	1	5	3	2	7	7	—	—	1	4	3	1	1	—	—	—	2	4	—	—	—	—	—	—	1	—	1	—
M. pneumoniae	—	—	—	—	—	—	—	—	—	—	—	—	1	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	1973									1974												1975										

REPORT OF ACUTE RESPIRATORY DISEASE

Please answer by writing Y for YES in the boxes.

Leave BLANK for NO

Name : 1–5

Report for week-ending 6–11

A. Since last week has had a cold, a sore throat, the 'flu', or any other respiratory illness ? ☐ 12

B. Since last week has he/she had an upset stomach or diarrhoea ? ☐ 13

If YES to either of above, please answer the following questions :

(1) On what date did the illness start ? ☐☐☐☐☐☐ 14–19

(2) Were any of the following symptoms present :–

(a) Any fever ? (Temperature if known...°) ☐ 20
(b) Any chills ? ☐ 21
(c) A headache ? ☐ 22
(d) An earache ? ☐ 23
(e) Any general aches or pains ? ☐ 24
(f) A stuffy or runny nose ? ☐ 25
(g) A sore throat ? ☐ 26
(h) Swollen or tender glands ? ☐ 27
(i) Any hoarseness ? ☐ 28
(j) A cough ? ☐ 29
(k) Any phlegm from the chest ? ☐ 30
(l) Any wheezy breathing ? ☐ 31
(m) Any chest pain or discomfort on breathing ? ☐ 32
(n) Any nausea or an upset stomach ? ☐ 33
(o) Any vomiting ☐ 34
(p) Any diarrhoea ? ☐ 35
(q) Any aching or redness of eyes ? ☐ 36
(r) Any stiffness of neck ? ☐ 37
(s) Any rash ? ☐ 38
(t) Any other symptoms ? ☐ 39
(Please state) ..... (Code) ☐☐☐ 40–42

(3) If the illness was given a name by a doctor, please state name : ..... (Code) ☐☐☐ 43–45

(4) Were antibiotic drugs prescribed by a doctor ? ☐ 46
If so, state name ..... (Code) ☐☐☐ 47–49
and date course started ..... ☐☐☐☐☐☐ 50–55

(5) Was any time spent in bed because of the illness ?
(a) at home ? ☐ 56
(b) in hospital ? ☐ 57
(c) and number of days (days) ☐☐☐ 58–60

(6) Other than days in bed, was any time taken off work/school or were there any restrictions on usual activities ? ☐ 61
Number of days (days) ☐☐☐ 62–64

(7) Is the illness still present ? ☐ 65
If NO, on what date was it last present ? ☐☐☐☐☐☐ 66–71
(NOTE. If the illness is still present, it is also reported on the following weeks questionnaire).

(8) Were nose and throat swab specimens taken ? ☐ 72
If so, date taken ? ☐☐☐☐☐☐ 73–78
Was blood taken ? ☐ 79
3 80

Fig. 1. Weekly illness reporting sheet used for family surveillance.

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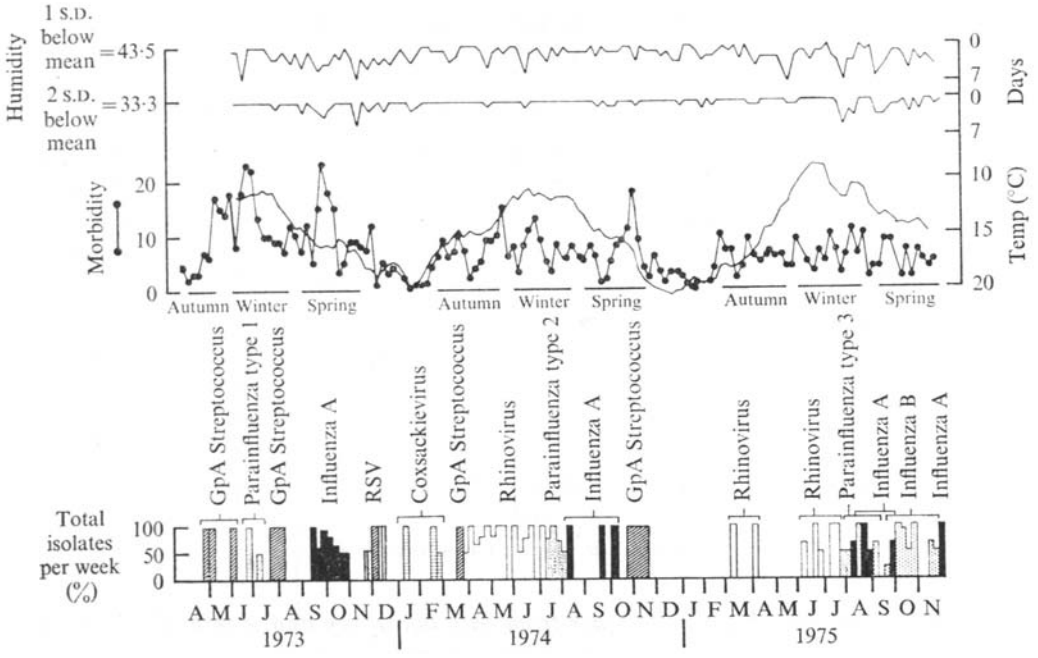

Fig. 2. (A) Total weekly morbidity and its association with meteorological observations, namely the maximum weekly air temperature (inverted) and the minimum weekly humidity shown as the number of days the humidity fell below 1 and 2 standard deviations below the mean of 53·6%. (B) The predominant pathogen isolated in each week during the study.

33%. Correlation with illness category and the age distribution of subjects (Table 7) shows that isolations were made more frequently from LR illness 66/95 (70%) than from UR 141/509 (28%) and symptom-free individuals 1/23 (4%), while isolations from infants 30/70 (43%) were more frequent than from school-aged children 93/243 (38%), adults up to 49 years 72/262 (27%) or adults over 50 years 15/64 (23%).

Patterns of pathogen incidence

The epidemiology of a pathogen in a community may vary from short severe epidemics to an endemic presence over many months. To identify these patterns, pathogen isolations were grouped in monthly intervals (Table 8), while the predominant pathogen isolated in each week has been plotted as a percentage of the total isolations in each week in Fig. 2. Where equivalent numbers of viruses and Group A streptococci have been isolated, the viruses have been shown preferentially. Also shown in Fig. 2 is the total family morbidity reported in each week, the maximum weekly air temperature shown as the inverse of the moving average taken over 4-week intervals, and the minimum weekly humidity shown as the number of days in each week during which the minimum humidity fell below 1 and 2 standard deviations below the mean of 53·6% for the whole study period.

Morbidity increased in the autumn and decreased in the late spring of each year. Pathogen isolation rates were highest in the spring of 1973, late autumn and

Table 9. *Fourfold or greater CF antibody rises in the sera from family members collected at 6-month intervals*

Pathogens tested for	No. with CF antibody rises* during indicated time intervals				
	May–Nov. 1973	Nov.–May 1973–4	May–Nov. 1974	Nov.–May 1974–5	May–Nov. 1975
Influenza A	23 (27%)	—	3 (3%)	—	3 (4%)
Influenza B	—	—	—	—	4 (5%)
Influenza C	1	—	—	1	1
Parainfluenza 1	—	—	—	—	2
Parainfluenza 2	—	—	2	—	—
Parainfluenza 3	—	—	2	—	4 (5%)
RSV	2	—	—	1	—
Adenovirus	—	2	—	1	—
Mumps S Antigen	—	—	1	—	4 (5%)
Measles virus	—	—	—	4 (5%)	—
M. pneumoniae	—	5 (5%)	4 (4%)	4 (5%)	2
Psittacosis	1	—	1	—	—

* Fourfold or greater CF antibody rise.

Table 10. *Number of serum samples from each age group and month of collection*

Year and month	Age groups sampled				No. of paired sera in each 6-month interval
	0–4	5–20	Adults	All ages	
1973					
May	5	35	51	91	86
Nov.	6	37	52	95	92
1974					
May	4	43	50	97	92
Nov.	3	39	51	93	86
1975					
May	0	40	47	87	85
Nov.	0	39	50	89	
Total	18	233	301	552	

early winter of 1974, and the late winter and spring of 1975. These high rates also correlate with periods of low humidity.

A distinct epidemic of clinical influenza due to the variant Influenza A/Port Chalmers/1/73(H3N2) occurred in each year of the study. The first and largest epidemic began in the early spring of 1973, continued for 7 weeks and was associated with a sudden increase in morbidity among families. Fourfold or greater rises in CF antibody titre occurred in 23 (27%) of paired sera (Table 9). The number of paired sera in each 6-month interval and the total number of sera collected are shown in Table 10.

In 1974 and 1975, isolations of the same influenza virus variant were made over a 7- to 8-week period in the late winter and early spring months. Only a small change in family morbidity was recorded with a fourfold or greater rise in CF

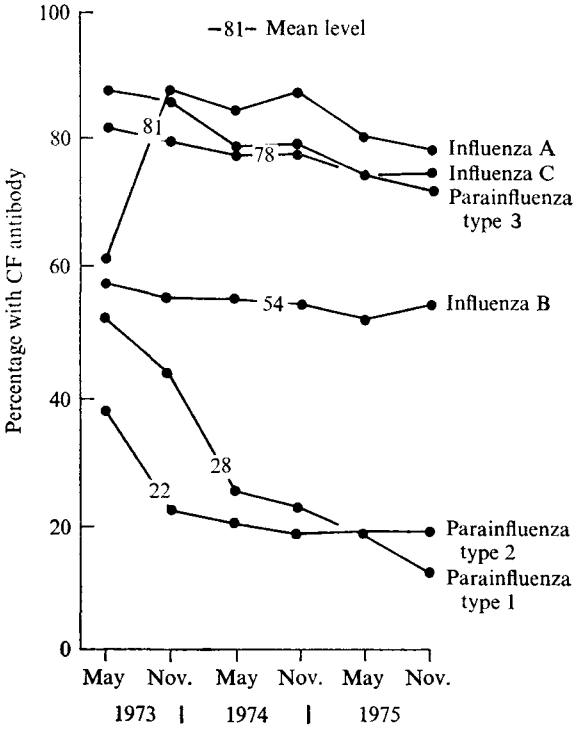

Fig. 3. Percentage of family members with CF antibody titres 1/8 or greater in sera collected at 6-month intervals between May 1973 and November 1975.

antibody being detected in 3 (3%) in 1974 and 3 (4%) in 1975. Late in November of 1975, one isolation of an influenza A virus was made from a 58-year-old male with an upper respiratory tract illness. This was shown to be similar to influenza A/Victoria/3/75(H3N2). Over these three influenza seasons, influenza A virus attacked all age groups and in two thirds of the cases was associated with lower respiratory symptoms.

Immediately following the presence of influenza A in the community in 1975, an influenza-B virus strain similar to B/Hong Kong/5/72 was isolated over a period of 10 weeks. Little change in family morbidity was recorded and only 4 (5%) fourfold or greater CF antibody rises were detected. As with influenza A all age groups were affected, with a larger proportion of school-aged children (59%). In contrast, the illnesses were nearly all upper respiratory (94%).

Influenza C virus was not isolated although fourfold or greater rises in CF antibody were detected in 2 (6%) school-aged children and one adult. The majority of family members tested possessed CF antibody at a titre of $\geqslant 1/8$, the mean level over the study period being 78% (Fig. 3).

Parainfluenza viruses were present in each winter. Type 2 virus caused a distinct 7-week epidemic in 1974, while Type 3 virus was present for 7 weeks in 1975-though it was the dominant virus for only 1 week. Infection with type 1 virus occurred in 1973 and 1975 (one only). The number of family members with detectable CF antibody to type 1 virus decreased between May 1973 and November

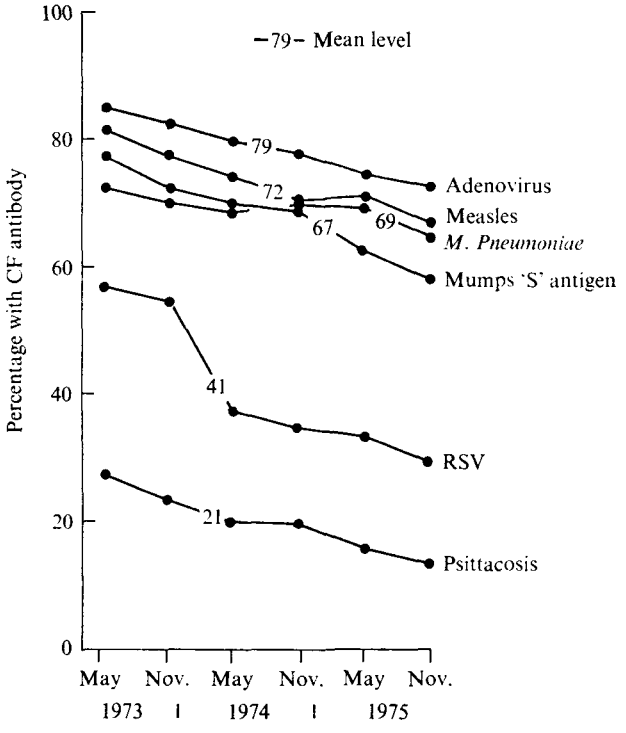

Fig. 4. Percentage of family members with CF antibody titres 1/8 or greater in sera collected at 6-month intervals between May 1973 and November 1975.

1975, the decrease being significant both for school-aged children (0·2 % level by Student's *t*-test) and adults (0·1 % level). Types 1 and 2 viruses were associated with upper respiratory illness, while type 3 virus was more frequently associated with lower respiratory illness. All age groups were affected, although the proportion of infants and children affected (75 %) was greater than with influenza B virus (59 %).

Respiratory syncytial virus was isolated twice in the late spring of 1973, immediately after the influenza A epidemic. Both were from illnesses with lower respiratory symptoms in children aged 1 and 5 years old. The number of family members with detectable CF antibody decreased between May 1973 and November 1975, the decrease being significant for both school-aged children (5 % level by Student's *t* test) and adults (1 % level).

Measles virus was not isolated, though four cases of clinical measles in school-aged children occurred in the summer of 1974–5 and these were confirmed by serology. The number of family members with detectable CF antibody decreased between May 1973 and November 1975 (Fig. 4), the decrease being significant for both school-aged children (1 % level by Student's *t* test) and adults (5 % level).

Mumps virus was isolated in the winter of 1975 from two young children, and during the same period 4 (5 %) fourfold or greater CF antibody rises to Mumps 'S' antigen were detected. The number of family members with detectable CF

antibody decreased between May 1973 and November 1975, the decrease being significant only among adults (5 % level by Student's *t* test).

No adenoviruses were isolated, although fourfold or greater rises in CF antibody occurred during the summers of 1973–4 in 2 adults and 1974–5 in one child. The majority of family members tested possessed detectable titres of CF antibody, the mean number over the study period being 79 %.

Rhinoviruses were isolated over a 14-week period during the autumn and early winter of 1974 and caused an increase in family morbidity during this period. In 1975 the pattern differed, with sporadic isolations made over 8 months from early autumn to late spring. Subsequent re-inoculation of specimens collected in 1973 did not alter the isolation rate for that year. Isolations were most frequent from upper respiratory illnesses, though nearly one fifth were from illnesses with lower respiratory symptoms. The age distribution was similar to that for influenza A virus, but with a slightly higher proportion of the younger age groups affected.

Coxsackie B3 virus was isolated during the summer months of 1974. Isolations were made at other centres in New Zealand during the same period. A single Coxsackie A virus was isolated late in 1975.

There was no distinct distribution of Herpes virus isolations.

Group A steptococci were isolated almost continuously during 1973 and 1974 with periods of increased incidence in late July 1973 and during the latter half of the influenza A epidemic in October and November 1973. They were virtually absent in 1975. The age distribution was very similar to that for rhinovirus infections, being particularly common in school-age children. Isolations were mainly from upper respiratory illnesses and on seven occasions a virus was isolated at the same time.

Mycoplasma pneumoniae was isolated in the late autumn and winter of 1974. In the period from November 1973 until November 1975, 15 fourfold or greater CF antibody rises were detected, 2 being in infants, 9 in school-aged children and 4 in adults. The number of family members with detectable CF antibody remained constant (mean number 69 %) over the study period.

DISCUSSION

Regular personal contact by one of us, keeping each family informed of surveillance results, maintained a high level of participation. The median number of illnesses, 3·0 per person per year, is lower than that reported in New York (4·8) and Seattle (4·1) (Fox *et al.* 1972). The New York and Seattle Virus Watch studies used families with new-born infants, and it is possible that the lower number of illnesses reported in Port Chalmers is due to the progressive decline in the proportion of infants in the study. Although 1973 was an influenza A epidemic year, the number of illnesses per infant decreased little between 1973 and 1975 as compared with school-aged children and female adults.

Respiratory illness in Port Chalmers accounted for 87 % and enteric illness for 9 % of all illness reported and this compares with 78 % and 8 % in New York, and

86% and 15% in Seattle. One striking difference was that lower respiratory illness constituted 31% of all respiratory illness which was a much higher proportion than that reported in New York (2%) (Fox *et al.* 1966); Seattle (6%) (Fox *et al.* 1972) and Tecumseh (25%) (Monto, Napier & Metzner, 1971). This result can be explained partly by the criteria for classification used. However, the high level of influenza A virus activity in the community may have contributed.

Although the assessment of fever was made by the mother, the proportion of all illness with fever (20%) is similar to that reported in Seattle (23%) and to that reported for all respiratory illness in Tecumseh (25%). Fever in relation to age groups in Seattle was 0–5 years (30%); 6–19 years (21%); parents (13%). Overall the analysis of severity criteria indicates that the illnesses reported in Port Chalmers were not more severe than would be expected in an open community.

The seasonal pattern of family morbidity, mostly respiratory illness, is similar to that observed in other studies (Hope-Simpson, 1958; Sutton, 1965). The association of a period of low humidity with high pathogen isolation rates is paradoxical. Although it has been demonstrated that the infectivity of influenza virus declines more slowly in dry air with a low relative humidity (Hemmes, Winkler & Kool, 1960), other respiratory viruses, also common in winter, are less stable in dry than in moist air (Buckland & Tyrrell, 1962). This observation cannot be readily explained.

The differing epidemiological behaviour of pathogens from the periodic epidemic presence of some virus types and probable endemic presence of other virus and pathogen types is similar to that reported in open community studies in larger populations. The following pathogens however deserve further discussion. In 1973, influenza A/Port Chalmers/1/73 caused the largest epidemic observed during the study. The epidemiological pattern was characteristic of a new variant entering a highly susceptible community and causing a relatively large, well-defined epidemic, followed by epidemics in succeeding years with reduced severity. There was an epidemic due to the variant influenza A/Victoria/3/75 in Port Chalmers during February 1976 (Jennings, personal observation), and this makes the single isolation of a similar strain during November 1975 very interesting. After introduction of this new variant at this time, it is possible that it then circulated subclinically and undetected (Hall, Cooney & Fox, 1973) until some unknown factor initiated the epidemic in 1976. We still know very little about how influenza circulates in the community, particularly in the inter-epidemic periods.

The presence of parainfluenza viruses was also characterized by epidemics of infection. Type 2 virus usually occurs in limited outbreaks, while type 3 virus occurs endemically with periodic increases in incidence (Chanock, Bell & Parrott, 1961; Monto, 1973; Medical Research Council, 1965). In this study, type 3 virus was isolated after an apparent absence for 2 years, and its epidemiological behaviour may be related to the number of susceptible individuals in the community.

At the other end of the range of epidemiological behaviour, the rhinoviruses, Group A streptococci and *M. pneumoniae* were primarily endemic in the community. An interesting aspect of rhinovirus infections was that 18% had predominantly lower respiratory symptoms. It is possible that the virus type responsible

for these infections had been absent from the community for a long period. In the absence of antigenic stimuli from repeated infections, which in the open community reinforce immunological memory (Holmes *et al.* 1976), they may have been unusually severe.

The epidemiological pattern of successive waves of respiratory patterns through the community of Port Chalmers was not as complex as the pattern in large open communities. The reasons for the replacement of the major respiratory virus types are obscure, although some interference phenomenon may be involved.

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