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Influenza surveillance and immunisation in New Zealand, 1990-1999

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Information provided by influenza surveillance is essential to guide both public health measures in the event of an influenza epidemic or pandemic, and decisions on vaccine composition. Influenza surveillance in New Zealand includes laboratory surveillance of influenza virus isolates, sentinel general practice (GP) surveillance of consultations for influenza-like illness (ILI), hospital discharge and mortality data, and vaccine coverage surveillance. During the 10 years, 1990-99, six influenza seasons have been dominated by influenza A(H3N2), three by influenza B, and one by influenza A(H1N1). In 1996, influenza A/Wuhan/359/95 (H3N2) virus caused a significant epidemic. On average, 2.7% of the population attended a GP because of an ILI during the influenza season. There were 278 hospital admissions and 34 fatalities a year directly attributed to influenza, although the true impact would have been much higher. High coverage with influenza vaccine is essential to reduce the impact of this highly preventable disease, particularly for those ≥ 65 years of age who experience the highest levels of hospitalisation and death.

Influenza continues to be a major threat to public health worldwide because of its ability to spread rapidly through populations. The disease is characterised by an abrupt onset of respiratory and generalised signs and symptoms, which typically resolve after several days in most people, although cough and malaise can persist for two or more weeks. In some people influenza can exacerbate underlying medical conditions, or lead to secondary bacterial or primary viral pneumonia. Severe illness, hospitalisation and death are most frequent in the very young, the elderly, and those affected by underlying chronic conditions.

Influenza virus types A, B and C cause clinically relevant disease in humans, although influenza C is not an important cause of epidemic disease. Influenza viruses are named after the places where they were first identified. For example, influenza A/Sydney/5/97 (H3N2) was first isolated in Sydney in 1997, and was influenza A isolate number 5 for that year. The code in brackets refers to the virus subtype.

Influenza viruses are successful human pathogens because of their ability to vary their two external proteins, the haemagglutinin (H) and neuraminidase (N). Mutations in the genomic RNA of both influenza A and B viruses produce a gradual change in the external proteins. This process is called 'antigenic drift' and results in changed viruses which are able to cause almost annual epidemics of influenza. The greater the change, the greater the capacity of the virus to

escape immune recognition and the greater the epidemic potential. At irregular intervals there are more dramatic changes, called 'antigenic shift', which result in the emergence of a new influenza A virus. These changes are the result of a genetic reassortment between animal and avian viruses, thought to occur in the domestic pig acting as an intermediate host or 'mixing vessel'. In the absence of immunity to these new subtypes, they rapidly spread worldwide causing pandemic influenza, with dramatically increased rates of morbidity and mortality. Both the new viruses responsible for the 1957 Asian (H2N2) and 1968 Hong Kong (H3N2) pandemics emerged by this mechanism. However, it is by no means certain that all antigenic shifts are caused by genetic reassortment. The 1918 Spanish (H1N1) pandemic virus may well have adapted from bird to man via the pig without reassortment, and the 1977 Russian

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(H1N1) virus (not a true pandemic virus) does not appear to be a reassortant. The direct transmission of an avian virus from bird to man has also occurred. In 1997, an influenza A(H5N1) avian virus caused an outbreak of influenza (chicken flu) in Hong Kong. Fortunately, this virus lacked the ability to be efficiently transmitted from person to person.¹

Currently influenza A(H1N1), A(H3N2) and B strains are co-circulating in the world. In New Zealand, their occurrence can vary both temporally and geographically during an influenza season or between seasons. The circulation of the A(H3N2) subtype since 1969, A(H1N1) since 1978, and B have followed a fairly uniform pattern, in which distinct antigenic variants have emerged and become predominant over a period of 2-5 years, only to be replaced by the next successful variant.

Influenza immunisation is the primary method for preventing influenza and its complications. New Zealand influenza immunisation recommendations target those at greatest risk of severe illness and death: people ≥ 65 years old and people < 65 years of age who have chronic medical conditions.² These groups are eligible for free influenza vaccination annually.

Optimal vaccine efficacy is dependent on achieving a close antigenic match between the vaccine and circulating strains. To facilitate this, the World Health Organization (WHO) established an influenza surveillance network in 1947.³ New Zealand joined this network in 1953, and is now one of 80 participant countries. The goal of surveillance is the early detection of changes in the influenza virus to guide decisions on vaccine composition.

Since 1998 the WHO has issued recommendations on vaccine composition each September for the guidance of Southern Hemisphere countries, in addition to the recommendations each February for the Northern Hemisphere. At the end of each Southern Hemisphere influenza season, the Australian Influenza Vaccine Committee (AIVC), including observers from New Zealand and South Africa, review surveillance data and other laboratory and epidemiological data from both the Southern Hemisphere and the rest of the world. The influenza vaccine composition, in line with WHO recommendations, is then finalised for the following season.

This article reports on the burden of influenza in New Zealand, the circulating influenza virus strains, and vaccine coverage for the 10 years 1990-99. The various surveillance methods that generate these data are also described.

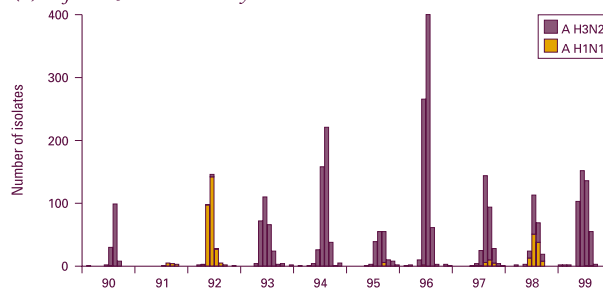
Methods

Laboratory surveillance: Ongoing surveillance of influenza is carried out by the four regional virus diagnostic laboratories at Auckland, Waikato, Christchurch and Dunedin Hospitals, and by ESR's virology laboratory. Both the ESR and Auckland Hospital laboratories are designated WHO National Influenza Centres. The regional virus diagnostic laboratories report all respiratory virus diagnoses, largely from hospital in-patients and outpatients, to ESR. These data are reported nationally in the *Virology weekly report*. The criteria for a laboratory identification of influenza are the isolation of the virus or the direct detection of viral antigen. Influenza isolates are typed and subtyped. From 1994, drug trials and associated surveillance programmes conducted by pharmaceutical companies contributed to these laboratory identification data.

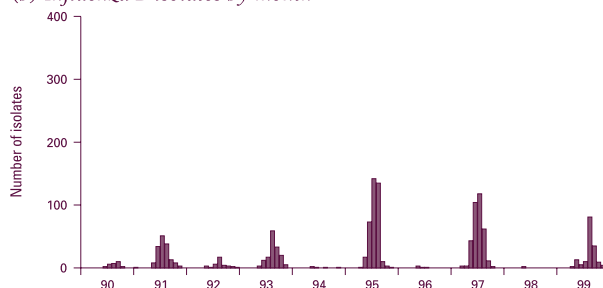
General practice (GP) sentinel surveillance: This surveillance system commenced in 1991 as part of the WHO Global Programme for Influenza Surveillance. It is operated nationally by ESR and locally by surveillance co-ordinators within the public health service in each health district. The system operates during the 'influenza season', from May through September each year. It is based on a network of voluntary sentinel GPs distributed on a population density basis of approximately 1 per 50 000. Each sentinel practice records the daily number of consultations that fit a case definition for an influenza-like illness (ILI), along with the patient's age group, on a standardised reporting form. The case definition used for ILI is an acute respiratory tract infection

Figure 1: Influenza in New Zealand, 1990-99

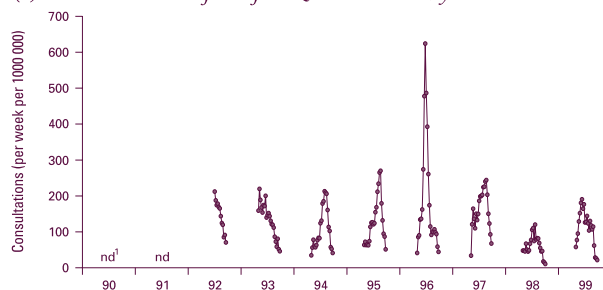
(a) Influenza A isolates by month



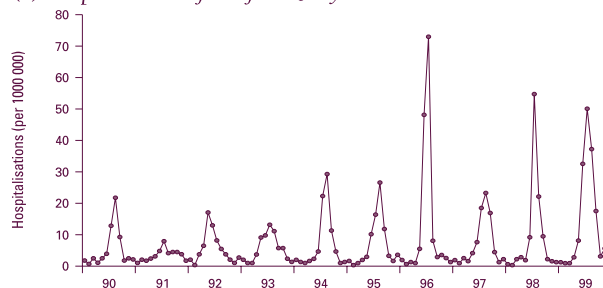
(b) Influenza B isolates by month



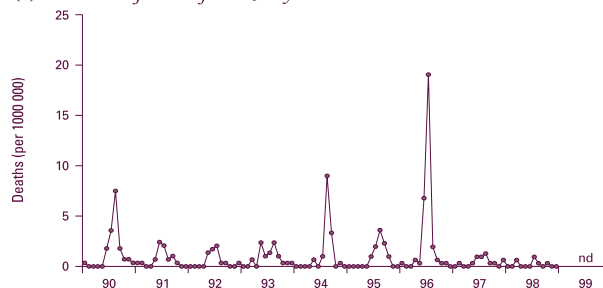
(c) GP consultations for influenza-like illness by week



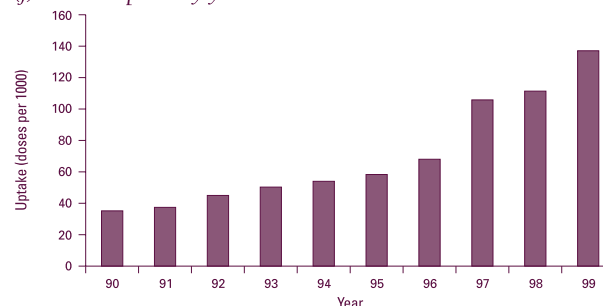
(d) Hospitalisations for influenza by month



(e) Fatalities from influenza by month



(f) Vaccine uptake by year



Note: 1 nd = no data available

Table 1. Influenza vaccine coverage rates for persons aged ≥65 years, 1997-99¹

Health region	1997		1998		1999	
	Number eligible	% cover	Number eligible	% cover	Number eligible	% cover
North	132 780	30	134 980	24	137 445	48
Midland	84 190	40	85 935	50	87 270	57
Central	97 670	37	98 530	48	99 170	52
Southern	122 160	48	123 380	58	124 390	64
Total	436 800	39	442 825	44	448 275	55
% increase over previous year		10 ²		6		11

Notes: 1 from reference 6

2 based on an estimate of coverage among ≥65 year olds in 1996

characterised by an abrupt onset of at least two of the following: fever, chills, headache and myalgia. These data are collected by the local co-ordinator by phone or fax each Friday. Each sentinel practice also provides respiratory samples from the first patient seen with an ILI on Monday, Tuesday and Wednesday of each week. These samples are forwarded to one of the regional laboratories or ESR for virus isolation and identification. Weekly consultation data, along with virus isolation data, are forwarded by local co-ordinators to ESR each Monday. Collated national data are presented in a weekly report, *Influenza surveillance update*. Consultation rates were calculated using the sum of the patient populations, reported by the participating practices, as the denominator. The national level of ILI activity is described using a set of threshold values: a weekly rate of 50-249 consultations per 100 000 patient population is considered indicative of normal seasonal influenza activity; 250-399 indicates higher than expected activity, and ≥400 indicates an epidemic level of disease.⁴

Hospitalisations: Hospitalisations for influenza (ICD-9CM code 487) were extracted from the New Zealand Health Information Service's National Minimum Dataset (NMDS) for the years 1990-99. Influenza-related hospitalisations were conservatively taken to include only those where influenza was the principal diagnosis. Repeat admissions were included, as repeat infections with another influenza A subtype or B virus are possible.

Hospitalisation, mortality and coverage rates were calculated using population data from the 1991 and 1996 censuses, with interpolated numbers for non-census years, as the denominators.

Mortality: Deaths from influenza (ICD-9CM 487) were also extracted from the NMDS. These mortality data were only available up to 1998.

Immunisation coverage: Prior to 1997, the number of doses of vaccine distributed in New Zealand was reported by the vaccine distributors, and coverage rates were estimated from these reports. In 1997 influenza vaccination was made available free to those ≥65 years of age, and in 1999 free vaccination was extended to risk groups <65 years.^{5,6} The data that medical practitioners provide to Health Benefits Limited to claim reimbursement are used to estimate coverage among these two groups entitled to free vaccination.

Comparison of surveillance data: Influenza disease rates detected by each surveillance system (sentinel, hospitalisation, mortality) were compared. Because the GP sentinel system only operates during the influenza season from May to September, this comparison used cumulative disease rates from each surveillance system for this period. The comparison was also restricted to the years (1993-98) for which data were available for all three systems.

Results

Laboratory surveillance: Influenza virus isolates obtained each year from 1990 to 1999 are shown in *Figures 1a* and *1b*. These isolates include those submitted year round to virus laboratories and those collected through the GP sentinel system. There were a total of 4325 isolates, of which the majority (70.2%, 3083) were influenza A and the remainder were influenza B. Influenza A(H3N2) was the predominant virus subtype isolated in six years and influenza A(H1N1) in one year (1992). In 1996, influenza A/

Wuhan/359/95 (H3N2) virus caused a significant epidemic. Influenza B activity was relatively high every second year, and was the predominant virus in three years (1991, 1995 and 1997).

GP sentinel surveillance: The national weekly consultation rates for influenza are shown in *Figure 1c*. Over the seven years from 1993 to 1999, the average annual cumulative incidence for the five-month influenza season was 2725.8 episodes of ILI per 100 000, or 2.7% of the patient population in the participating practices (range from 1.4-4.1% during 1993-99). Influenza in 1996 was considered to have reached epidemic levels, while seasonal activity in 1995 was also higher than normal. In all other years, normal seasonal influenza activity was recorded (ie, peak activity was in the range of 50-249 episodes of ILI per 100 000 per week). Rates were highest in infants <1 year old (775.9 per 100 000) and lowest in those ≥60 years (206.9).

Hospitalisations: A total of 2776 influenza hospitalisations were recorded in the 10 years from 1990 to 1999 (*Figure 1d*), giving an average annual hospitalisation rate of 7.5 per 100 000. There was an apparent increase in hospitalisations during the 10 years. The number of admissions peaked at 464 in 1996 and 518 in 1999 - both years dominated by influenza A(H3N2). Hospitalisation rates were high for infants under 5 years of age (24.0 per 100 000), and increased from 55 years of age with a rate of 33.7 per 100 000 for those ≥65 years. During the 1990-99 period, 21.5% of hospitalisations were in those ≥65 years of age. Age-standardised hospitalisation rates (standardised to the 1996 census population) were generally higher for Maori (9.5 per 100 000) and Pacific Islands people (8.0) than for European (7.3).

Mortality: Influenza mortality data for 1990 to 1998 are shown in *Figure 1e*. There were 307 influenza fatalities recorded during this nine year period, an average annual rate of 0.9 per 100 000. Deaths from influenza peaked at 94 in 1996 during the influenza A(H3N2) epidemic. The death rate is markedly higher in those ≥65 years (10.5 per 100 000) and this age group accounts for the majority of deaths (94.1%). The age-standardised mortality rate (standardised to the 1996 census population) was higher for Maori (1.6 per 100 000) than for European (0.9). There was only one fatality recorded for a Pacific Islands person during the nine years, which is largely a reflection of the relatively small number of elderly Pacific Islands people in New Zealand.

Immunisation coverage: The uptake of influenza vaccine in New Zealand increased each year from 1990 to 1999 (*Figure 1f*). Since 1997, when immunisation benefit claim data became available, influenza immunisation rates among those aged ≥65 years increased from 39% in 1997 to 44% in 1998 and 55% in 1999 (*Table 1*). Coverage in people <65 years with chronic medical conditions was estimated at 19% for 1999.

Comparison of surveillance data: The average annual cumulative incidence rates (based on the influenza season from May to September, averaged for 1993-98) were 2780.3 consultations per 100 000, 6.8 hospitalisations per 100 000 (0.2% of the consultation rate), and 0.9 deaths per 100 000 (13.2% of the hospitalisation rate). The three surveillance systems all identified 1996 as a particularly severe influenza season. Otherwise, the annual cumulative incidence rates for the three systems were not well correlated over this period, partly because of the small number of years of available data. All of the influenza surveillance systems identified the highly seasonal nature of the disease, with peak incidence rates typically around July. The systems each identified different age distributions of disease burden, as described above.

Discussion

Influenza infections are a predictable cause of morbidity and mortality. The impact of influenza in New Zealand over the past 10 influenza seasons has been substantial in terms of general practitioner consultations, hospitalisations and deaths. On average about 2.7% of the population consult a general practitioner annually because of an ILI. A number of community and family-based studies have shown that, during average influenza seasons,

the overall attack rates are often 10-20%.⁷ Among schoolchildren and in old peoples' homes, attack rates of 40-50% are not unusual.⁸ Influenza caused an average of 278 documented hospital admissions and 34 fatalities a year during the 1990s. It is well recognised that such data underestimate the impact of influenza.⁹ Influenza modelling carried out by the Ministry of Health for 13 years from 1980-92 suggested there were 435 deaths annually, 8.7 times the actual number of influenza deaths recorded.²

Antigenic changes in the influenza virus result in variations in the annual impact of influenza. In the 10 years since 1990, the influenza A(H3N2) viruses. Consistently, influenza B has shown increased activity in alternate years.¹⁰ Epidemics caused by influenza A(H3N2) are often associated with increased mortality.⁷ This was seen in New Zealand during the 1996 A/Wuhan/359/95 (H3N2) epidemic, when 464 hospitalisations and 94 deaths were recorded as due to influenza.

The impact of influenza varies greatly by age and, to a lesser extent, by ethnicity. The highest rates of infection are in the very young, whereas the worst outcomes are in the elderly. Rates of hospitalisation and death are higher in Maori than other ethnic groups.

The impact of influenza can be reduced by annual vaccination. Influenza vaccines are recommended for persons at risk of developing complications following infection because of their age or because of an underlying chronic condition.² There are now many studies which confirm the clinical efficacy of influenza vaccines in such populations, if the vaccine strains of influenza are identical or similar to the circulating strains. A meta-analysis of 20 cohort studies on vaccine effectiveness in elderly people estimated the use of the vaccine was associated with a 56% reduction in respiratory illness, 53% reduction in pneumonia, 50% reduction in hospitalisations, and 68% reduction in deaths.¹¹ In addition, there is good evidence for the use of vaccine in both those under 65 years who are at risk of the complications of influenza¹² and those in the workplace.¹³

Influenza vaccines are most effective when their antigenic content is similar to the influenza A and B strains in circulation. New Zealand has been an active participant in the WHO's worldwide influenza surveillance network, and contributes to the Australian vaccine formulation meeting. The match between the formulation of each year's vaccine and the strains which have actually circulated has been remarkably close.¹⁴

The use of influenza vaccine has increased steadily in New Zealand since 1990.¹⁵ Although influenza vaccination has been recommended for at-risk groups for several years, it was not made available free to those ≥ 65 years until 1997. This single policy change has resulted in an accelerated increase in vaccine use, with an increase from 39% coverage among this age group in 1997 to 55% in 1999. This policy was extended to include free vaccination for risk groups < 65 years in 1999. It is estimated that 19% of this group received vaccine in 1999 based on immunisation benefit claim data. However, this is likely to be an underestimate, as a number of people in this group would have received vaccination through employer-subsidised schemes.

The 1997 policy change has also seen the development of influenza awareness programmes promoting the benefits of influenza immunisation. The ElderCare Canterbury programme, a collaborative effort between 17 healthcare providers initiated in 1998, has seen immunisation coverage in Canterbury increase to 65.7% in 1999 among those ≥ 65 years of age. The formation of the National Influenza Immunisation Strategy Group, with the aims of educating the public on the seriousness of influenza and healthcare professionals on the benefits of immunisation, should also contribute to improving coverage.

This review is the first to correlate, over a multiple-year period, the main New Zealand influenza surveillance data sources. While these data sources identified many similarities in incidence time trends and the characteristics of affected cases, there were also

important differences. The relatively poor correlation by year between sentinel data and hospitalisations is probably a reflection of changes in the operation of these surveillance systems during the 1990s. There was a trend towards reduction in reporting rates from the sentinel system while recorded influenza hospitalisations increased. The increase in hospitalisations is likely to be due to improvements in coding as well as the general increase in hospitalisations that occurred over this period. Ultimately, we might expect to see declines in hospitalisation and mortality rates as immunisation coverage increases in the targeted population groups, though it is probably too early to detect such trends.

Influenza surveillance remains a cornerstone of our strategy to control influenza. It ensures the provision of timely information to the Ministry of Health, healthcare providers and the general public about levels of activity and circulating strains. The early detection of seasonal increases and epidemics supports the implementation of public health strategies, such as immunisation of risk groups, targeted use of influenza antivirals, and management of the seasonal demand on clinical services. The isolation and antigenic characterisation of influenza viruses is critical for the formulation of the following season's vaccine. Surveillance information is essential to the planning of improved influenza vaccine coverage and treatment, and to identify the emergence of novel viruses with the potential to cause pandemics.

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Low and late influenza activity in 2000

National influenza surveillance in 2000 was undertaken between May and September using a sentinel network of 90 general practices. On average 87 practices, with a total patient roll of 321 027, participated each week.

Participating practices recorded the number of consultations for influenza-like illness each week and the age group of each suspected case. Influenza-like illness was defined as acute upper respiratory tract infection, characterised by abrupt onset, and at least two of the following: fever, chills, headache and myalgia. Each practice was also asked to collect swabs from up to three patients each week. The swabs were sent to the regional virus laboratories for viral isolation and strain identification.

During the surveillance period, 2239 consultations for influenza-like illness were reported, and the average weekly consultation rate was 32.5 per 100 000 patient population. This rate is the lowest since rates have been available from the sentinel surveillance system which began in 1991 (refer to lead article in this issue), and less than a third of the 1999 rate of 112.3. The consultation rate remained consistently low throughout the 22 weeks covered by the sentinel surveillance system (Figure 1). However, based on isolations of influenza virus in the five regional virus laboratories, there appeared to be a late peak of activity in September (week 38) and considerable ongoing activity until almost the end of the year (Figure 2).

Figure 1: Weekly consultation rates for influenza-like illness in New Zealand, 1996-2000

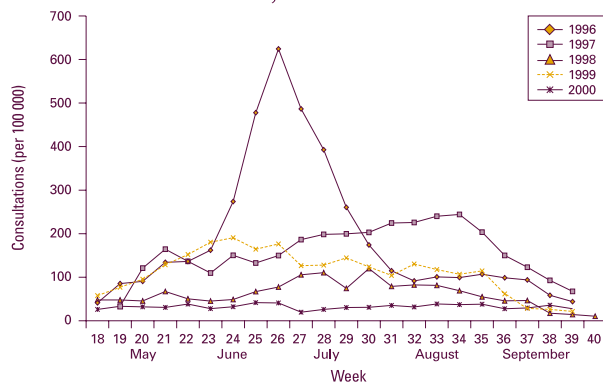
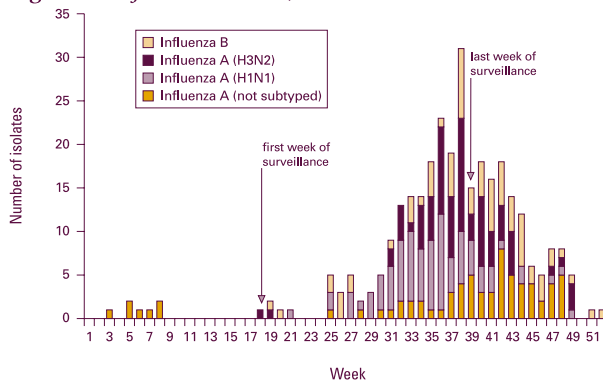
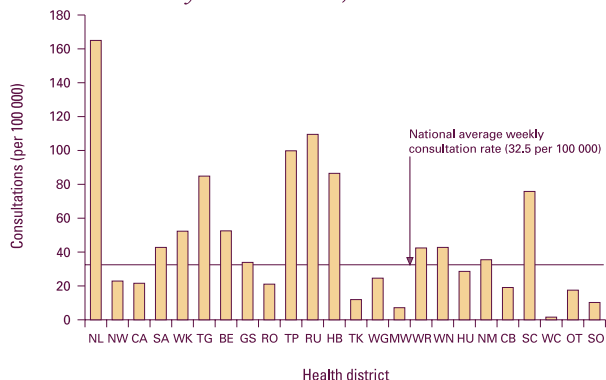


Figure 2: Influenza isolates, 2000



Consultation rates varied between health districts, with rates of more than twice the national average in Northland (165.0 per

Figure 3: Average weekly consultation rates for influenza-like illness by health district, 2000



100 000), Ruapehu (109.5), Taupo (99.8), Hawkes Bay (86.5), Tauranga (84.8), and South Canterbury (75.8) (Figure 3).

Infants and pre-schoolers were the most likely to be seen by a general practitioner for an influenza-like illness. The age-specific average weekly consultation rates were: infants less than one year of age, 43.8 per 100 000; 1-4 year olds, 38.7; 5-19 years, 32.6; 20-64 years, 36.5; and ≥ 65 years, 15.6. The lower rate in those ≥ 65 years of age is likely to be due, at least in part, to higher levels of vaccination in this age group.

Influenza virus was isolated from 73 (8.5%) of the 854 swabs submitted by the sentinel practices. Sixty-two (84.9%) of these isolates were influenza A. The remaining 11 were influenza B. Of the 50 influenza A isolates further typed, 41 (82%) were subtype H1N1, and 9 (18%) were subtype H3N2. Based on all influenza virus isolations made by the regional virus laboratories during 2000 (Figure 2), both subtypes of influenza A and influenza B circulated throughout the season.

Characterisation of the influenza viruses isolated during the 2000 winter indicated a need for a change in the influenza A (H3N2) and influenza B components of the vaccine for the 2001 winter. Accordingly, the 2001 Southern Hemisphere winter influenza vaccine has the following composition:

- A/New Caledonia/20/99 (H1N1)-like strain
- A/Moscow/10/99 (H3N2)-like strain
- B/Sichuan/379/99-like strain

This composition differs from the vaccine used in the last (2000-01) Northern Hemisphere winter, but is the same as that recommended for the next (2001-02) Northern Hemisphere winter.

Influenza immunisation is especially recommended for those at increased risk of complications from influenza due to their age or medical condition (see the *Immunisation handbook* for details). Influenza vaccination has been free for people ≥ 65 years of age since 1997, and since 1999 has been extended to younger people with chronic illnesses who are at risk of developing complications from influenza. Based on immunisation benefit claims data, vaccination coverage in 2000 was 59% among those ≥ 65 years and 28% among those < 65 with chronic illnesses. Optimally, influenza vaccine should be given in March or April, but it can be given later. The immunisation benefit can only be claimed for vaccinations given before 1 July.

Surveillance data

National surveillance data - December 2000

Disease ¹	Current year - 2000 ²			Previous year - 1999			Trends - December 2000
	Dec 2000 cases	Cumulative total year-to-date	Current rate ³	Dec 1999 cases	Cumulative total year-to-date	Previous rate ³	
AIDS	0	27	0.7	2	33	0.9	
Campylobacteriosis	917	8414	232.5	1016	8163	225.6	*
Cholera	0	0	0	0	1	0	
Creutzfeldt-Jakob disease	1	4	0.1	0	1	0	300
Cryptosporidiosis	28	773	21.4	23	977	27.0	***
Dengue fever	0	7	0.2	0	9	0.2	
Gastroenteritis ⁴	49	732	20.2	68	604	16.7	***
Giardiasis	114	1687	46.6	109	1793	49.6	
<i>H influenzae</i> type b disease	1	13	0.4	1	10	0.3	
Hepatitis A	7	108	3.0	4	119	3.3	
Hepatitis B (acute) ⁵	7	78	2.2	6	94	2.6	
Hepatitis C (acute) ⁵	3	83	2.3	6	96	2.7	
Hydatid disease	0	3	0.1	0	8	0.2	
Influenza ⁶	4	251	6.9	1	798	22.1	***
Lead absorption	26	125	3.5	8	153	4.2	
Legionellosis ⁶	5	70	1.9	8	69	1.9	
Leprosy	2	5	0.1	1	10	0.3	
Leptospirosis	8	101	2.8	7	59	1.6	***
Listeriosis	2	22	0.6	2	19	0.5	
Malaria	5	113	3.1	5	46	1.3	*** 146
Measles	1	67	1.9	8	107	3.0	**
Meningococcal disease	41	477	13.2	42	508	14.0	
Mumps	4	50	1.4	5	56	1.5	
Paratyphoid	2	21	0.6	1	17	0.5	
Pertussis	347	4147	114.6	207	1046	28.9	*** 296
Rheumatic fever	3	118	3.3	1	65	1.8	***
Rubella	1	26	0.7	3	35	1.0	
Salmonellosis	150	1805	49.9	112	2077	57.4	***
Shigellosis	9	115	3.2	11	147	4.1	*
Tetanus	0	1	0	1	6	0.2	
Tuberculosis	22	358	9.9	34	449	12.4	**
Typhoid	2	21	0.6	0	9	0.2	*** 133
VTEC/STEC infection	4	70	1.9	1	64	1.8	
Yersiniosis	22	396	10.9	43	503	13.9	***

Notes: 1 Other notifiable infectious diseases reported in December: Nil

2 These data are provisional

3 Rate is based on the cumulative total for the current year (12 months up to and including December 2000) or the previous year (12 months up to and including December 1999), expressed as cases per 100 000

4 Cases of gastroenteritis from a common source or foodborne intoxication (eg, staphylococcal intoxication or toxic shellfish poisoning)

5 Only acute cases of this disease are currently notifiable

6 Surveillance data based on laboratory-reported cases only

7 Percentage change is the difference between the number of cases in the current year (12 months up to and including December 2000) and the previous year (12 months up to and including December 1999). This difference is expressed as a percentage of the number of cases in the previous year.

Surveillance data

Surveillance data by health district - December 2000

Cases this month Current rate¹

Disease	Cases for December 2000, ² and current rate ^{1,2} by health district ^{3,4}																							
	Northern				Midland							Central						Southern						
	Northland	NW Auck	Central Auck	South Auck	Waikato	Tauranga	Eastem BOP	Gisborne	Rotorua	Taupo	Taranaki	Ruapehu	Hawkes Bay	Wanganui	Manawatu	Wairarapa	Wellington	Hutt	Nelson-Marl	West Coast	Canterbury	South Cant	Otago	Southland
AIDS ³	0				0							0						0						
	1.3				0							0.7						0.6						
Campylobacteriosis	15	86	27	49	71	18	3	9	12	2	23	0	48	13	15	6	72	44	17	5	228	21	51	22
	149.5	210.0	225.0	181.5	266.4	153.4	101.4	153.0	141.0	185.7	190.9	119.4	226.5	141.6	113.7	197.6	305.5	218.7	138.9	178.9	352.6	402.3	301.1	356.6
Cholera	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Creutzfeldt-Jakob disease	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0
	0	0	0.3	0	0.9	0	0	0	0	0	0	0	0.7	0	0	0	0	0	0	0	0.3	0	0	0
Cryptosporidiosis	2	0	1	0	0	0	1	1	1	0	0	0	7	0	1	1	1	0	0	1	4	1	7	0
	17.5	7.9	10.1	10.2	41.0	14.2	4.0	24.0	68.2	19.5	14.0	0	27.2	11.4	23.3	31.2	17.3	32.4	0.9	37.0	20.4	75.4	33.6	37.7
Dengue fever	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	0	0	1.4	0	0	0	0	0	0	0	0	0	0	0	0	0.4	0	0	0	0	0	1.3	0	0
Gastroenteritis	0	7	4	3	3	0	0	0	0	0	1	0	0	0	0	0	4	0	0	0	16	1	9	1
	4.4	17.8	22.8	14.0	11.2	14.2	8.0	2.2	13.9	159.6	32.8	0	0	0	2.7	7.8	14.4	6.8	13.7	6.2	61.3	41.5	20.8	5.4
Giardiasis	2	13	14	11	15	6	2	0	1	1	1	0	6	0	1	1	16	5	4	4	6	0	3	2
	34.3	58.1	74.3	38.9	59.5	70.0	17.9	35.0	60.4	52.1	15.0	23.9	65.5	14.7	21.9	23.4	71.2	43.0	12.9	123.4	34.1	36.5	28.4	19.8
H influenzae type b disease	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	1.5	0	0.3	0.9	0.3	2.7	2.0	0	0	0	0	0	0	0	0	0	0.8	0	0	0	0.3	0	0	0
Hepatitis A	1	1	2	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	2	0	0	0
	0.7	1.5	2.6	4.7	0.7	3.5	2.0	15.3	3.1	0	1.9	0	0.7	0	0	1.6	1.5	1.7	0	10.9	3.8	1.2	1.8	
Hepatitis B	0	0	2	0	3	0	0	0	0	0	0	0	0	0	1	0	0	0	1	0	0	0	0	0
	1.5	1.0	3.5	0.6	4.3	1.8	2.0	4.4	1.5	3.3	0	6.0	3.5	0	2.0	2.6	1.6	2.3	1.7	0	3.1	3.8	1.7	0.9
Hepatitis C	0	0	0	1	0	0	0	1	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0
	0.7	1.0	0	0.6	1.0	16.8	4.0	2.2	13.9	0	0.9	0	7.7	0	0.7	0	1.6	3.0	2.6	0	2.6	2.5	2.9	0.9
Hydatids	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	0	0	0	0	0.3	0	0	2.2	0	0	0	0	0	0	0	0	0.4	0	0	0	0	0	0	0
Influenza ⁵	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	1	0	0	0	2	0	0	0	0
	0.7	0.3	19.1	0	9.9	0	0	0	0	0	0	0	0	0	0	6.6	0	0	0	33.9	0	3.5	0	
Lead absorption	1	0	2	0	0	0	0	1	0	0	0	0	2	1	2	1	1	0	0	1	10	2	2	0
	2.2	0.3	1.2	1.2	4.6	1.8	2.0	8.7	3.1	3.3	0.9	0	3.5	4.9	7.3	2.6	3.3	0	3.4	3.1	8.3	16.3	4.6	1.8
Legionellosis ⁵	0	0	0	0	1	1	0	0	0	0	0	0	0	0	0	2	0	0	1	0	0	0	0	0
	2.9	1.0	1.4	0.3	6.3	0.9	0	0	0	0	0	0	1.6	0.7	5.2	2.5	3.8	0	3.1	3.9	0	2.9	0	
Leprosy	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0
	0	0.3	0	0.6	0.3	0	0	0	0	0	0	0	0	0	0	0.4	0	0	0	0	0	0	0	0
Leptospirosis	0	1	0	1	2	0	0	0	0	0	0	1	1	0	1	0	0	0	0	1	0	0	0	0
	4.4	0.8	0	0.6	4.3	4.4	0	10.9	0	0	3.7	11.9	7.0	4.9	2.0	2.6	0.4	0	7.7	9.3	2.1	17.6	2.9	3.6
Listeriosis	0	0	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	0	0.8	0	1.2	0.7	0.9	0	0	0	0	0	0	0	0	0	0.4	2.3	0	0	0	0.8	3.8	0	1.8
Malaria	0	0	0	1	1	0	0	1	0	0	0	0	0	0	1	0	1	0	0	0	0	0	0	0
	2.2	0.5	0.9	2.0	2.6	0	4.0	2.2	0	3.3	0.9	17.9	0.7	0	39.9	0	2.1	2.3	3.4	0	1.0	2.5	0.6	1.8
Measles	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0
	3.6	2.8	0.9	0.6	0	0	0	0	1.5	3.3	0	6.3	0	0.7	2.6	0.8	3.0	4.3	3.1	3.1	2.5	1.2	4.5	
Meningococcal disease	1	4	6	9	4	1	0	2	2	0	0	1	1	1	0	0	0	0	3	0	4	0	1	1
	18.2	9.9	22.0	34.5	14.9	10.6	23.9	26.2	17.0	3.3	3.7	23.9	13.2	11.4	5.3	13.0	7.4	7.5	4.3	0	4.9	5.0	9.8	5.4
Mumps	0	1	0	1	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	1	0	0	0
	0.7	1.3	2.0	1.5	0	1.8	0	0	1.5	0	0.9	0	4.2	1.6	0.7	0	2.5	0.8	4.3	0	1.6	0	1.2	0
Paratyphoid	0	0	2	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	0	0.3	0.9	0.6	1.7	0	0	0	0	0	0	0	1.4	0	1.3	0	0.4	1.5	0	0	0.8	0	0	0
Pertussis	10	10	15	15	64	8	4	5	6	2	1	7	9	0	9	20	16	25	3	91	1	16	10	
	85.3	56.6	53.2	48.6	164.6	69.2	85.5	94.0	71.3	48.9	10.3	77.6	47.4	1.6	10.0	249.6	54.8	137.2	432.2	622.9	285.6	100.6	154.0	53.0
Rheumatic fever	0	0	0	0	0	0	1	0	1	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0
	10.2	1.5	7.2	9.7	4.3	1.8	11.9	2.2	6.2	0	0	6.0	3.5	0	2.6	2.1	1.5	0	0	0	0	0	0	0
Rubella	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0
	0.7	0.3	0.9	0.3	0	0	0	0	0	0	0.9	0	0.7	0	0	1.6	1.5	1.7	0	2.1	0	1.2	0	0
Salmonellosis	3	8	7	10	7	6	1	2	1	1	3	0	5	3	8	3	15	4	4	0	24	14	16	5
	29.2	35.0	30.7	24.0	40.7	42.6	29.8	37.2	21.7	45.6	24.3	11.9	38.3	27.7	71.1	176.8	57.2	40.7	48.9	24.7	67.5	106.9	107.1	129.4
Shigellosis	0	0	4	0	1	0	0	0	0	0	0	0	0	0	0	1	0	0	0	1	2	0	0	0
	2.2	4.3	6.7	5.6	2.6	0	6.0	4.4	3.1	19.5	0	1.4	0	0	0	3.3	5.3	0.9	0	2.3	2.5	1.7	0	
Tetanus	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	0	0	0	0	0	0	0	0	0	0	0	0	1.6	0	0	0	0	0	0	0	0	0	0	0
Tuberculosis	1	0	5	5	0	0	0	0	0	0	0	0	0	0	0	9	1	0	0	0	0	1	0	0
	5.1	9.4	20.5	21.1	7.6	5.3	9.9	4.4	6.2	3.3	1.9	11.9	8.4	8.1	8.0	5.2	19.3	13.6	1.7	0	2.6	2.5	6.9	3.6
Typhoid	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0
	0	0.5	2.0	2.0	0.3	0	0	0	0	0	0	0	0	0	0	1.2	0	0	0	0	0.3	0	0	0
VTEC/STEC infection	0	0	0	0	2	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	1	0
	0	0.5	0	0.9	6.9	0	4.0	0	3.1	16.3	6.6	0	1.4	3.3	0	0.4	0	0.9	0	2.3	5.0	5.2	0	
Yersiniosis	0	3	4	2	0	1	0	1	2	0	0	0	0	0	2	1	0	2	2	0	2	0	0	0
	3.6	14.0	15.6	8.5	9.6	16.8	13.9	4.4	17.0	9.8	1.9	0	12.5	0	2.7	2.6	12.8	11.3	9.4	27.8	14.0	21.4	4.6	10.8

Notes: 1 Current rate is based on the cumulative total for the 12 months up to and including December 2000, expressed as cases per 100 000

2 These data are provisional

3 AIDS data given by divisions of the Health Funding Authority

4 Further

Systematic review of water fluoridation

A systematic review of the safety and efficacy of water fluoridation was recently published by the National Health Service's Centre for Reviews and Dissemination, University of York. A total of 214 studies were included in the review, and were of low to moderate quality, highlighting a lack of high quality research on this topic. The results revealed that water fluoridation was associated with a greater proportion of children without caries (median of 14.6% fewer), and a reduction in the average number of teeth affected by caries (median of 2.25 teeth). There was no clear evidence of potential adverse effects except a dose-dependent increase in dental fluorosis. However, the full report on which this paper is based illustrates that the contribution made by water fluoride at levels of ≤ 1 mg/l (ppm) to the prevalence of aesthetically concerning dental fluorosis did not reach statistical significance (McDonagh MS, Whiting PF, Wilson PM, et al. Systematic review of water fluoridation. *BMJ* 2000;

321: 855-9).

Editorial note: The *Drinking water standards for New Zealand 2000* recommend the adjustment of fluoride to a level of 0.7-1.0 mg/l in drinking water in order to prevent dental caries. In May 2000 approximately 1.9 million New Zealanders were receiving water from fluoridation treatment plants. Excessive fluoride during tooth formation can cause dental fluorosis. Sources of fluoride most likely to contribute to such an excess in New Zealand are the ingestion of fluoride toothpaste and/or use of fluoride tablets. Therefore public health dental advice since 1995 has been that, whether the water supply is fluoridated or not, children under the age of five should use no more than a 5 mm smear of fluoride toothpaste on a small brush and be discouraged from swallowing or eating toothpaste. Fluoride tablets are not recommended for children under the age of three years, even in non-fluoridated areas.

Hepatitis viruses transmitted in prisons via injecting equipment

The results of a study in English and Welsh prisons suggest that hepatitis viruses are probably being transmitted in prisons through shared, non-sterile, injecting equipment and that a risk of HIV transmission exists. Eight of the 135 prisons in England and Wales were enrolled in the study: six prisons for adult males, one for females, and one for young (<21 years of age) males. Eighty-three percent (3942) of the inmates eligible for the study completed a risk-factor questionnaire and provided an oral fluid specimen for antibody testing. Among the 3930 prisoners tested, 0.4% were positive for anti-HIV, 8% for anti-hepatitis B core (HBc) and 7% for anti-hepatitis C virus (HCV). Among the adult prisoners, 24% reported having injected drugs; 30% of

whom injected in prison. Three-quarters of those who injected in prisons shared needles or syringes. The presence of antibodies to HCV and HBc was associated with injecting inside prison and the number of previous times in prison (Weild AR, Gill ON, Bennett D, et al. Prevalence of HIV, hepatitis B, and hepatitis C antibodies in prisoners in England and Wales: a national survey. *Commun Dis Public Health* 2000; 3: 121-6).

Editorial note: The Hepatitis Foundation and the Department of Corrections have been running a pilot programme in prisons in the Wellington region to screen for hepatitis B and C, and to vaccinate against hepatitis B. The Department of Corrections is currently reviewing the results of the pilot.

Travel health

Preventing travellers' thrombosis

The importance of travellers' thrombosis, also known as 'flight-related deep vein thrombosis (DVT)' or 'economy class syndrome', is uncertain. So far there is only circumstantial evidence of an association between air travel and venous thrombosis. The few epidemiological studies to date have contradictory findings. Current evidence suggests that any association between symptomatic DVT and air flight is weak, and its incidence much less than the impression given by recent publicity (Geroulakos G. The risk of venous thromboembolism from air travel. *BMJ* 2001; 322: 188).

Editorial note: Despite the probably low incidence of travellers' thrombosis, it would be wise to give travellers general advice on measures that are thought to minimise the risk. The recently published House of Lords Select Committee on Science and Technology's report on the health risk of air travel contains specific advice on this topic (<http://www.parliament.the-stationery-office.co.uk/pa/ld/ldhome.htm>). Travellers with no known predisposing factors should be encouraged to exercise their legs periodically while travelling and to drink plenty of fluids (but not alcohol or caffeine-containing drinks). All travellers should be assessed to identify risk factors for DVT. Minor risk factors include age over 40 years, obesity, extensive varicose veins, and recent minor surgery. Moderate risk factors include recent heart disease, pregnancy, hormone medications and contraceptives, recent leg injury or surgery, and family history of DVT. Substantial risk factors include previous DVT, recent stroke, known clotting tendency, recent major surgery, and current malignant disease. Depending on the degree of risk, travellers could be advised against taking sleeping pills while travelling, given support or compression stockings, prescribed low-dose aspirin, or in extreme cases given heparin or advised to postpone their travel.

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