Annual report of the National Influenza Surveillance Scheme, 2002

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Abstract

Surveillance for influenza in Australia in 2002 was based on notifications to the National Notifiable Diseases Surveillance system from all states and territories, national and state-based sentinel practice consultations for influenza-like illness and reports of influenza virus isolations from a laboratory network. The impact of influenza was assessed by absenteeism data from a major national employer. Influenza A was the dominant type, 99 per cent of which were subtype H3N2 with only a single H1 isolate, which was identified as H1N2.The H3N2 isolates were closely related to the vaccine strain A/Moscow/10/99 and the A/Panama/2007/99, with less than one per cent showing genetic variation. Influenza B made up 21 per cent of circulating influenza and the majority of B strains were of the B/Victoria lineage, but had a haemagglutinin closely related to the B/Hong Kong/330/2001 strain.This strain was associated with two outbreaks but a proportion of vaccinees with the 2002 vaccine showed protective antibody titres.The 2002 influenza vaccine was given to 77 per cent of Australians over 65 years. *Commun Dis Intell* 2003;27:162–172.

Keywords: influenza, surveillance, vaccine, general practice, strain

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Introduction

Influenza is an acute, self-limiting upper respiratory tract infection. Complications, including lower respiratory tract infection (in particular secondary pneumonia and exacerbation of chronic obstructive pulmonary disease) and exacerbation of cardiopulmonary disease may occur.[1] Influenza-related morbidity (measured as excess hospitalisation) and mortality may result from these complications. Although influenza infection affects all age groups, the rates of serious morbidity and mortality tend to be highest among those aged 65 years and over, Aboriginal and Torres Strait Islanders and those with chronic medical problems. Young infants and pregnant women are also at increased risk of hospitalisation from influenza.

Influenza outbreaks usually occur during winter months in temperate climates (peaking between December and March in the Northern Hemisphere and June and September in the Southern Hemisphere), but may occur throughout the year in tropical regions. Even though the complication rate may be low, the overall high attack rate during epidemics leads to a considerable increase in hospitalisations and mortality. In Australia in 2001, pneumonia and influenza were underlying causes of 2,702 deaths (ICD–10 codes J10–J18; 2.1% of all deaths).[2] Influenza pandemics occur every 10 to 30 years. During these pandemics a quarter or more of the global population may be infected within a short period and the rates of illness and death from influenza can increase dramatically.

Influenza viruses are successful human pathogens because of their ability to vary their two external proteins, haemagglutinin (H) and neuraminidase (N). Mutations cause a gradual change in these proteins called 'antigenic drift', which results in annual epidemics of influenza. The greater the change in these proteins, the less likely it is that the virus will be recognised by immune cells primed by exposure to earlier infections or vaccines, and the greater the epidemic potential. At irregular intervals, there are more dramatic changes in the viral proteins, called 'antigenic shift', which are a result of either direct introduction of avian influenza viruses into the human population or a re-assortment between human and avian viruses which is believed to occur in an intermediate host such as pigs. These 'shifts' result in the emergence of a new influenza virus. In the absence of immunity to these new viruses, there is a rapid spread of influenza with dramatically increased rates of morbidity and mortality. After the pandemic of 1918 the H1N1 virus circulated widely in the human population. The Asian and Hong Kong pandemics in 1957 and 1968 introduced the H2N2 and H3N2 subtypes. There have been no major 'antigenic shifts' causing pandemics of influenza since 1968, however, the H1N1 subtype reappeared in the human population in 1977. Since 1977, influenza A (H1N1), A (H3N2) and influenza B viruses have been widespread globally, varying in frequency temporally and geographically.[3]

The formulation of influenza vaccines for use in Australia is determined annually by the Australian Influenza Vaccine Committee after review of the viruses circulating locally and internationally and after consideration of the World Health Organization (WHO) recommendations made in September. Influenza vaccination is provided free to non-indigenous Australians aged 65 years and above and indigenous Australians aged 50 years and above and is recommended for individuals with a range of underlying risk conditions, for pregnant women and for individuals who may transmit influenza to those with risk conditions.[4]

An effective national surveillance system is an essential component of a program for the control of influenza. Influenza surveillance is a mix of laboratory reporting of isolates and clinical diagnosis of influenza-like illness in sentinel practice schemes. Influenza surveillance aims to ensure the provision of timely information to public health departments, health care providers and the general public about levels of influenza activity and circulating strains. The major objectives of such surveillance include:

(i) early detection of epidemics to enable the implementation of public health measures such as vaccination of the 'at risk' groups, control campaigns and provision of clinical services;

(ii) characterisation of the nature of the epidemic;

(iii) isolation and antigenic characterisation of circulating influenza viruses to assist in the formulation of the following season's vaccine and to provide new vaccine strains; and

(iv) evaluation of the impact of the epidemic and associated public health measures.

This annual influenza report provides a summary of the surveillance methods and data for 2002.

Surveillance methods

Surveillance of influenza in Australia is based on six sets of data:

1. notifications required by legislation to state and territory health departments and nationally reported to the National Notifiable Diseases Surveillance System (NNDSS);
2. laboratory diagnosis including virus isolation and serology by laboratories participating in the Laboratory Virology and Serology Reporting Scheme (LabVISE);
3. subtype data of influenza virus isolates forwarded by LabVISE laboratories provided by the WHO Collaborating Centre for Reference and Research on Influenza;
4. consultation rates for influenza-like illness diagnosed by sentinel general practitioners;
5. absenteeism data of workers from a national employer; and
6. hospitalisation and mortality data.

National Notifiable Diseases Surveillance System

The Communicable Diseases Network Australia (CDNA) brings together communicable disease epidemiologists in all Australian states and territories.[5] The CDNA has revised the list of diseases to be notifiable across all jurisdictions. From January 2001, this included laboratory-confirmed influenza for the first time. In 2002 all states and territories reported influenza to NNDSS.

Laboratory surveillance

LabVISE is a national scheme of sentinel laboratories. In 2002, 12 laboratories contributed to this scheme, although not all provided reports each month. Laboratory reports of influenza are sent to LabVISE all year round. Although viral isolation remains the gold standard for influenza diagnosis and surveillance, most reports have relied on the detection of viral antigen and serological markers. Nucleic acid detection by the polymerase chain reaction is now in use for diagnosis.[3]

WHO Collaborating Centre for Reference and Research on Influenza

The WHO Collaborating Centre for Reference and Research on Influenza contributes reports on the subtypes and antigenic analysis of influenza viruses isolated throughout the year. This information is used to monitor the characteristics of influenza strains present in Australia and the rest of the world, to assess the suitability of the current vaccine (by measuring the degree of antigenic match between circulating strains and the current vaccine) and to determine the composition of vaccine for the following influenza season.

Standard nomenclature for influenza viruses is based on type, the place where they were first identified, sequential number and year of isolation. For example, A/Sydney/5/97 was first isolated in Sydney in 1997 and was influenza A isolate number 5 for that year.

The WHO Collaborating Centre for Reference and Research on Influenza conducts detailed antigenic analysis on all isolates received from Australian laboratories, and laboratories throughout Oceania and South East Asia, using conventional serological techniques. A geographically and temporally representative sample of isolates, together with any strains demonstrating uncharacteristic reactions during antigenic characterisation are further analysed by genetic sequencing of the viral haemagglutinin antigen and, for a proportion of these, the neuraminidase antigen. Studies are also conducted with panels of pre-and-post vaccination human sera to determine the likely effectiveness of current vaccines against recently circulating viruses to provide data that assists in vaccine formulation decisions.

Sentinel general practitioner surveillance

Sentinel general practitioner surveillance schemes detect and record clinical diagnoses of influenza-like illness (ILI). Participation is voluntary in all sentinel general practice surveillance systems, leading to variation in the number of contributors. The Australian Sentinel Practice Research Network (ASPREN) collects data at a national level. In addition, data are collected through the New South Wales Influenza Surveillance Scheme, the Victorian Influenza Surveillance Scheme, Western Australian sentinel general practices and the Northern Territory Tropical Influenza Surveillance Scheme. The case definition for a clinical diagnosis of ILI varies between sentinel surveillance schemes (Table).

Table. Case definitions of influenza-like illness used in Australian sentinel practice schemes, 2002[6]

Program	Case definition
Victorian State program	Fever, cough, fatigue
Western Australia State program	Fever, cough, fatigue
New South Wales State program, Northern Territory and ASPREN	Six of the following symptoms with sudden onset (<12 hours previously): cough, rigours or chills, fever, prostration and weakness, myalgia, redness of mucous membranes, influenza in close contacts

Sentinel general practices contributing to the ASPREN scheme are mostly located in capital cities and larger regional centres on the east coast of Australia. In 2002, the average number of contributing practices was 51 (range 32–65) each week. These practices together reported on an average of 5,674 (range 2,081–7,476) consultations per week.

The Northern Territory Tropical Influenza Surveillance reported cases of ILI throughout the year from between 8 and 12 centres in the tropical northern regions, reporting on between 469 and 1,092 consultations per week. Data were reported as the rate per 1,000 consultations on a weekly basis.

The New South Wales Influenza Surveillance program collects clinical reports from New South Wales practitioners who are part of ASPREN and from seven Public Health Units, four rural and three metropolitan (Southern New South Wales, New England, Mid North Coast, Macquarie, Illawarra, Central Coast, Northern Sydney, Western Sydney and South Eastern Sydney). The total number of participating practices varied from 14 to 58 per reporting period with a mean of 3,513 consultations across participating practices per week. Reports were published weekly in 2002 between 4 May and 28 September.

The Victorian Infectious Diseases Reference Laboratory, the WHO Collaborating Centre for Reference and Research on Influenza and the Department of Human Services contributed to the Victorian Influenza Surveillance in 2002. Reports were published fortnightly between 5 May and 29 September. The sentinel practices reporting to the scheme in 2002 varied between 16 and 23 metropolitan and 7 to 15 rural practices, which reported on between 5,348 and 8,509 consultations each fortnight. ILI was reported per 100 patients and converted to a rate per 1,000 consultations to allow comparisons with other sentinel schemes.

In Western Australia, between 4 and 13 metropolitan and 1 to 2 rural practices reported ILI from 3 June to 28 October 2002. The number of consultations in the sentinel practices was not recorded and the data were presented as the number of cases of ILI per practice for each week.

Absenteeism surveillance

Australia Post, a major nationwide employer, continued to provide sick leave absenteeism data during 2002 between March and September. Absenteeism, defined as an absence due to illness for at least three consecutive days, was reported as a rate per 100 employees per week.

Hospitalisation data

To assess the impact of influenza on hospitalisation, the Australian Institute of Health and Welfare made available data on hospital separations and average length of stay in public and private hospitals. Information was accessed by ICD–10AM code that classifies influenza under two categories: cases of influenza where the virus was identified (J10) and cases where the virus was not identified (J11). Data for the 2000–01 financial year was the most recent available at the time of writing this report.

Results

The influenza surveillance data presented here are limited and should be interpreted with caution. Laboratory-confirmed influenza represents a small proportion of all influenza cases in the year and consequently the estimation of the circulating strains is based on a small sample. Definitions of ILI vary between sentinel practices (Table) which make comparisons of influenza prevalence difficult. In addition, definitions of ILI have varied from year to year, so comparisons of data across years are complex.

National Notifiable Diseases Surveillance System

In 2002, 3,780 laboratory-confirmed cases of influenza were reported to the NNDSS. All jurisdictions submitted reports although few reports of confirmed infections were received from Tasmania due to limited access to laboratories for testing for influenza. The number of notification received in 2002 were threefold greater than those received in 2001. This was in part due to incomplete reporting in 2001, the first year in which laboratory-confirmed influenza was a nationally notifiable disease.[7]

The notifications to NNDSS by month of report are shown in Figure 1. Notifications showed a peak in August (1,188 notifications). The breakdown of laboratory-confirmed influenza cases reported to NNDSS by age and sex is shown in Figure 2. The age specific rates were highest among children aged less than five years (119 cases per 100,000 population), and among persons 85 years or older (31 cases per 100,000 population). Although the overall male to female ratio for influenza in 2002 was 1.1:1, in children aged less than five years, there was a higher rate of influenza among males (130 cases per 100,000 population) than females (100 cases per 100,000 population).

Figure 1. Notifications of laboratory-confirmed influenza to the National Notifiable Diseases Surveillance System, Australia, 2002, by month of onset

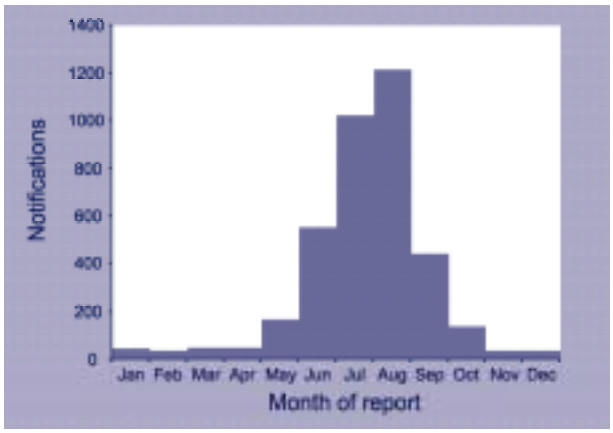
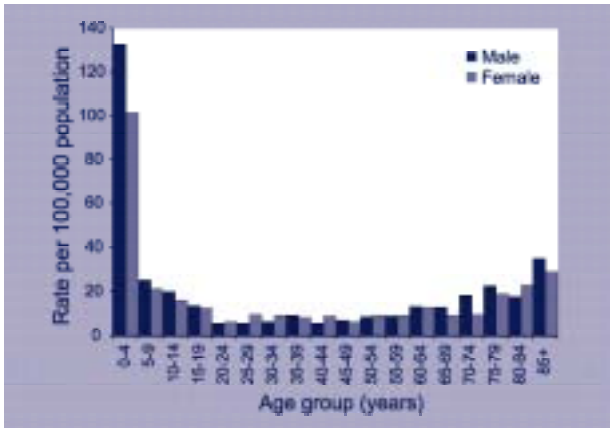


Figure 2. Notification rates of laboratory-confirmed influenza received by the National Notifiable Diseases Surveillance System, Australia, 2002, by age and sex



Laboratory surveillance

In 2002, a total of 2,345 laboratory diagnoses of influenza were made in participating laboratories of the LabVISE scheme. These were 1,798 influenza A and 547 influenza B diagnoses. The ratio of influenza A to B in 2002 was 3:1, which was lower than the 2001 ratio of 4:1. The overall influenza report showed a low level of activity until week 22 (3 June), when there was an increase in reports from 14 per week to approximately 45 per week. This was followed by a major peak of 169 reports per week in week 30 (29 July), then a decline to baseline (14 reports per week) by week 43 (28 October, Figure 3). The peak of influenza activity occurred earlier than in 2001 (Figure 4). In 2002, the peak in influenza B virus activity (week 22) preceded the peak of influenza A activity (week 30, Figure 3).

The seasonal pattern of influenza A and B activity between 1996 and 2002 is shown in Figure 5. The seasonal pattern in 2002 closely resembled that in 1997 with a relatively higher numbers of influenza B isolates, peaking earlier in the season than influenza A.

Figure 3. Laboratory reports of influenza, Australia, 2002, by type and week of specimen collection

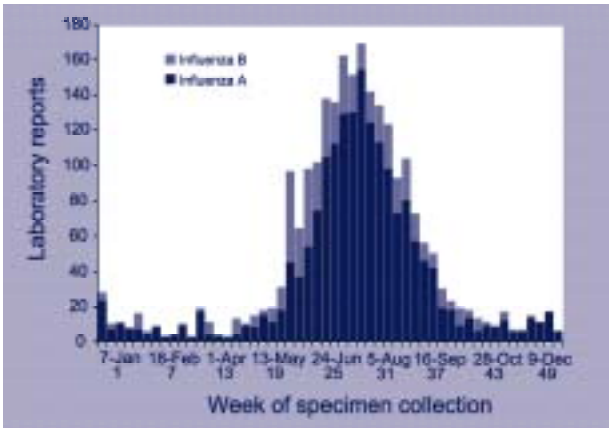


Figure 4. Laboratory reports of influenza, Australia, 2001 and 2002, by month of specimen collection

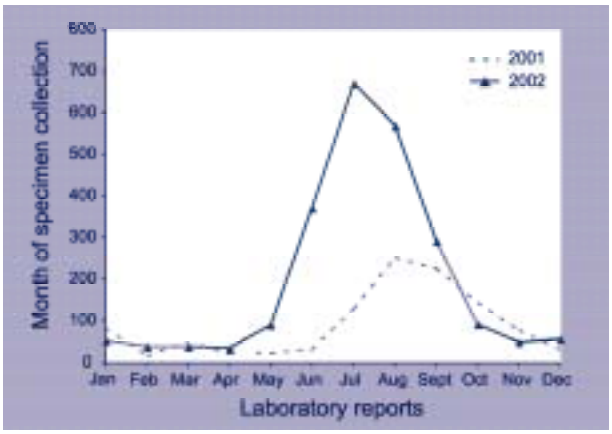
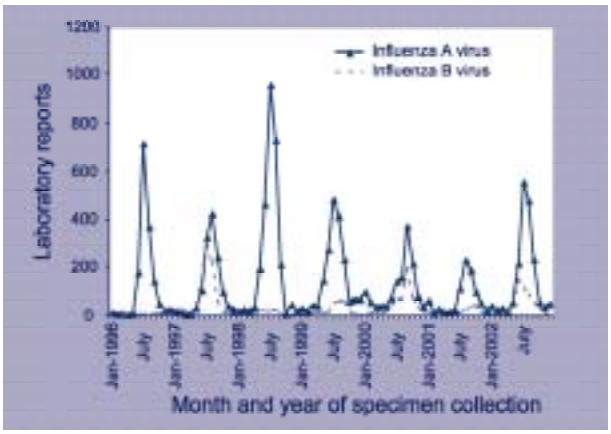


Figure 5. Laboratory reports of influenza, Australia, 1996 to 2002, by type and month of specimen collection



WHO Collaborating Centre for Reference and Research on Influenza

The WHO Collaborating Centre for Reference and Research on Influenza received 1,412 isolates and specimens that yielded viable viruses for antigenic analysis, 800 more than in 2001. Of these viruses 1,110 (78.4%) were influenza A(H3), 302 (21.5%) were influenza B and there was a single A(H1) strain. The variable region of the haemagglutinin was sequenced for 95 strains (1H1, 66H3, 28B) strains and the neuraminidase in 44 strains (27A and 17B) strains. The majority of A(H3) viruses were antigenically closely related to the A/Moscow/10/99 reference strain and the A/Panama/2007/99 vaccine strain with eight (<1%) strains showing some evidence of genetic variation. The neuraminidase was confirmed as N2 in all of 26 H3 viruses for which sequencing was undertaken. Sequencing of H3 haemagglutinin demonstrated continued genetic heterogeneity but no clear line of evolution at the moment (Figure 6). The single A(H1) strain was demonstrated to have a haemagglutinin that was antigenically and genetically closely related to A/New Caledonia/20/99 but an N2 neuraminidase genetically similar to those found on recent A(H3N2) viruses. Hence this virus belongs to the genetic reassortant lineage A(H1N2) which was first reported during the 2001–02 Northern Hemisphere season.[8] The A(H1N1), A/New Caledonia/20/99-like viruses continued to circulate in some countries while viruses of the A/Bayern/7/97 lineage have not been seen recently.

Figure 6. Evolutionary relationships between influenza H3 haemagglutinins (HA1 region)

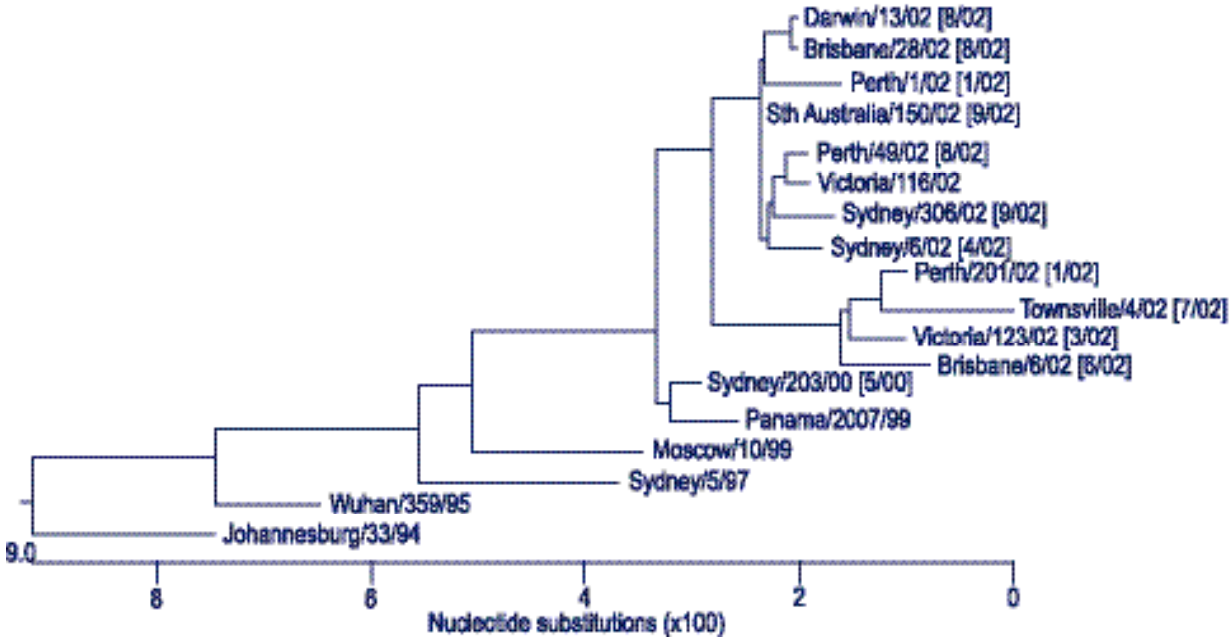
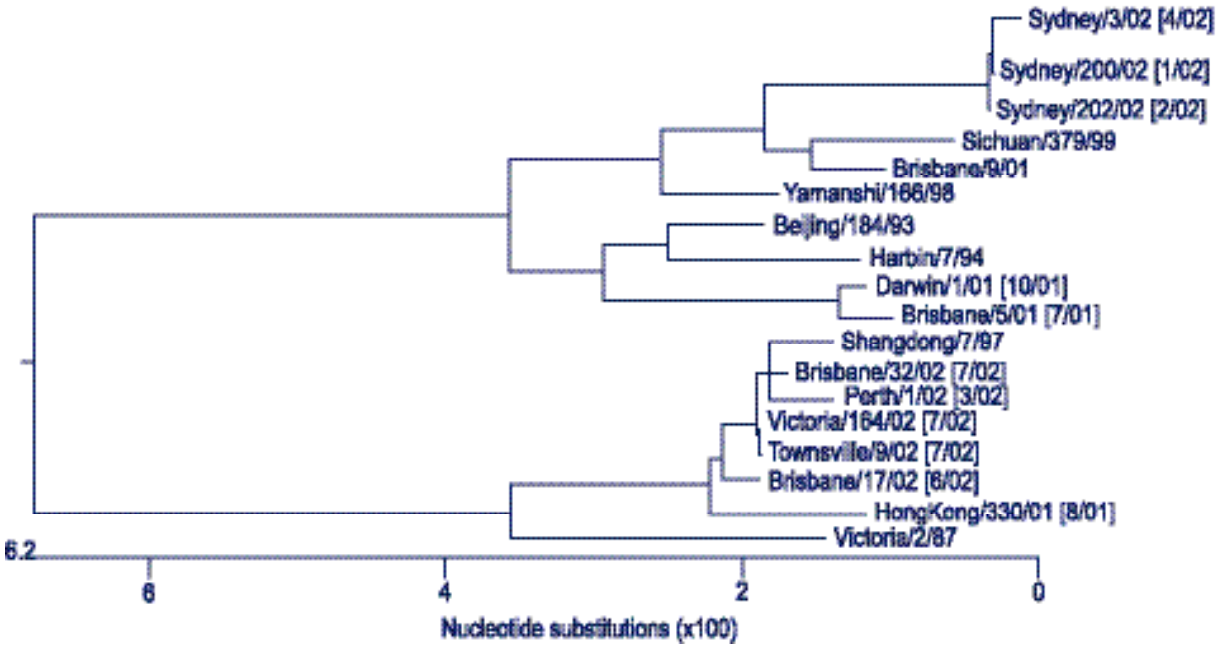


Figure 7. Evolutionary relationships between influenza B haemagglutinins (HA1 region)



The Australian influenza B viruses isolated during 2002 mainly (291 of 302 isolates) belonged to the lineage often referred to as the B/Victoria lineage which has not been seen in Australia for a decade. While this lineage of viruses had continued to circulate in Asia it had been absent from other regions for many years, re-emerging in some areas of Europe and North America during the 2001–02 winter.[9] Viruses isolated in Australia had a haemagglutinin that was antigenically and genetically closely related to the B/Hong Kong/330/2001 reference strain (Figure 7). However, by sequence analysis the neuraminidase of the isolates was found to be similar to that of the B/Sichuan/379/99-like viruses of the alternate lineage indicating that a genetic reassortment event had also occurred for these viruses. Some viruses isolated in the Northern Hemisphere 2001–02 winter were similarly found to be genetic reassortants.[9] The remaining 11 influenza B strains analysed belonged to the previously circulating lineage, nine were antigenically close to B/Sichuan/379/99 while two were more closely related to the older B/Harbin/7/94 reference strain.

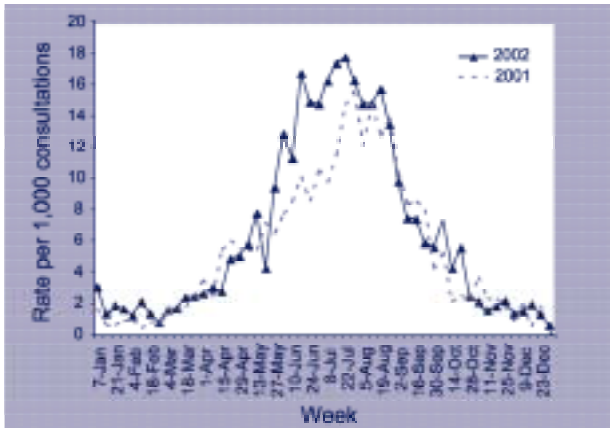
Based on the antigenic and genetic analyses and post-vaccination human serology studies conducted at the WHO Collaborating Centre for Reference and Research on Influenza the 2002 Australian influenza vaccine represented a good antigenic match for the circulating influenza A viruses but only for a minority of the influenza B strains.

After a decade of absence B/Hong Kong/330/2001-like viruses began to spread from Asia into other regions in 2001. However, apart from a small outbreak in Hawaii in May, this spread only became apparent some time after the WHO Consultation on Influenza Vaccine Formulation in September 2001 and the Australian Influenza Vaccine Committee meeting on 11 October 2001 which both recommended a B/Sichuan/379/99-like vaccine strain for the 2002 vaccine. Because there is a substantial antigenic difference between viruses of these two lineages, as expected, the 2002 vaccine produced reduced responses against the new B/Hong Kong/330/2001-like viruses. Nevertheless, around 40 per cent of younger adults and 25 per cent of older adults achieved antibody titres in the protective range against recent strains compared with 95 per cent and 80 per cent against the homologous virus, in younger and older adults respectively.

Sentinel general practice (GP) surveillance

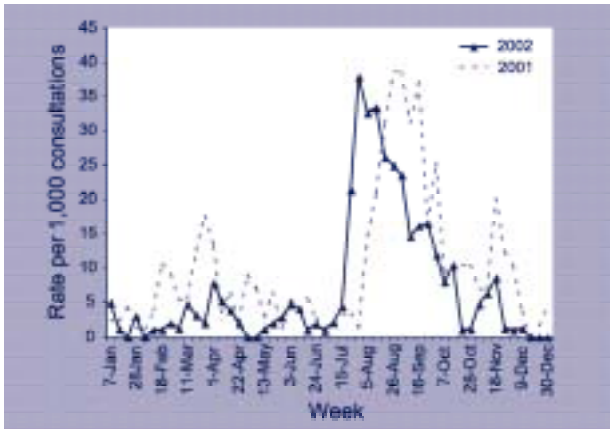
Reports of influenza-like illness to ASPREN practice sites showed a rapid rise starting in week 20 (27 May) and peaking in week 24 (17 June) when reports reached a rate of 16.6 cases per 1,000 consultations, and remained at that level for nine weeks. The peak in rates of ILI was earlier and slightly higher than in 2001 (Figure 8).

Figure 8. ASPREN consultation rates for influenza-like illness, Australia, 2001 and 2002, by week of report



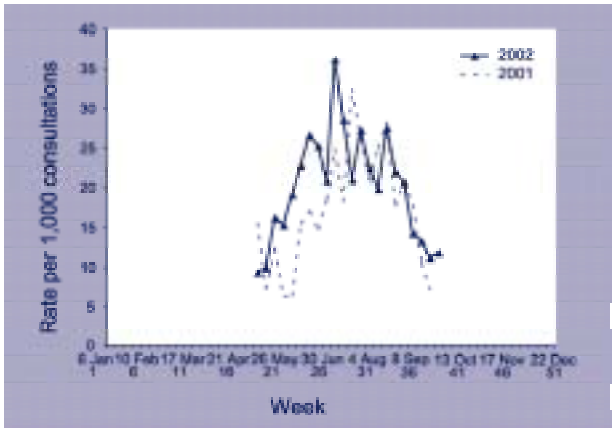
The Northern Territory Tropical Influenza Surveillance Scheme data showed one major peak (38.7 cases per 1,000 consultations) of influenza activity in week 30 (28 July) which was of the same magnitude but four weeks earlier than the peak influenza activity reported in 2001 (Figure 9).

Figure 9. Consultation rates for influenza-like illness,Northern Territory, 2001 and 2002, by week of report



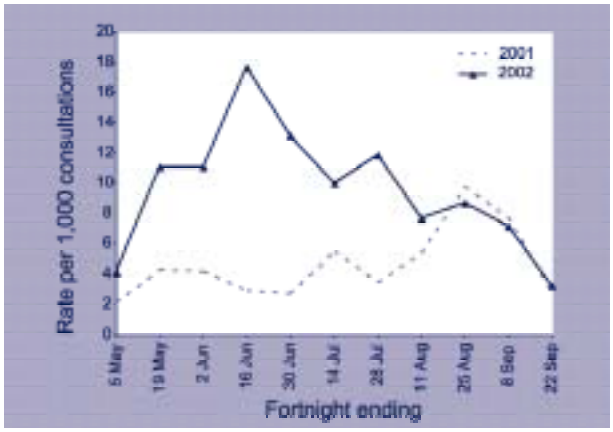
In New South Wales, influenza-like illness reports peaked in week 27 (7 July) at 36.2 cases per 1,000 consultations (Figure 10). In contrast with the peak consultation rate in 2001, the peak activity of influenza in New South Wales in 2002 was earlier and marginally higher.

Figure 10. Consultation rates for influenza-like illness, New South Wales, 2001 and 2002, by week of report



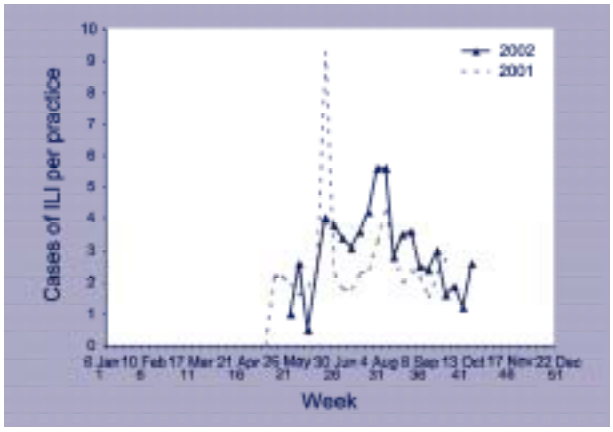
In Victoria, the reporting rate of influenza-like illness in 2002 peaked at 17.6 cases per 1,000 consultations in the fortnight ending 16 June (Figure 11). In comparison to the previous year (peak consultation rate on 16 August at 9.7 cases per 1,000 consultations), the peak consultation rate in 2002 was higher and occurred earlier in the year.

Figure 11. Consultation rates for influenza-like illness, Victoria, 2001 and 2002, by fortnight of report



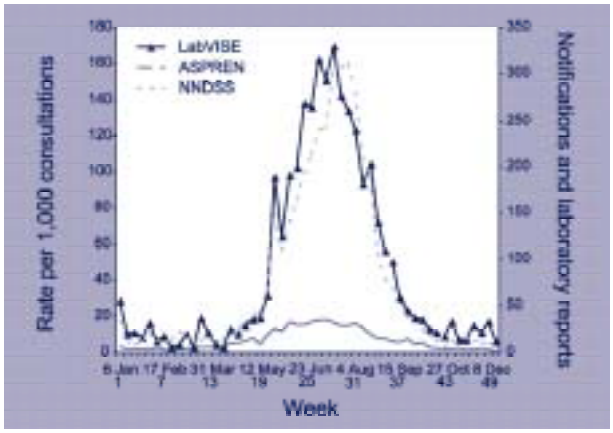
In Western Australia, the peak of reporting of influenza-like illness occurred later than in the eastern states, in week 32 to 33 (11–18 August) at 5.6 cases per practice (Figure 12).

Figure 12. Consultation rates for influenza-like illness, Western Australia,2001 and 2002, by week of report



A comparison of the NNDSS, ASPREN and LabVISE reports is shown in Figure 13. The peak in confirmed influenza notifications received by NNDSS, in reports of influenza-like illness to ASPREN, and in laboratory reports of influenza to LabVISE, overlapped in the period between week 29 to 32 (21 July to 4 August).

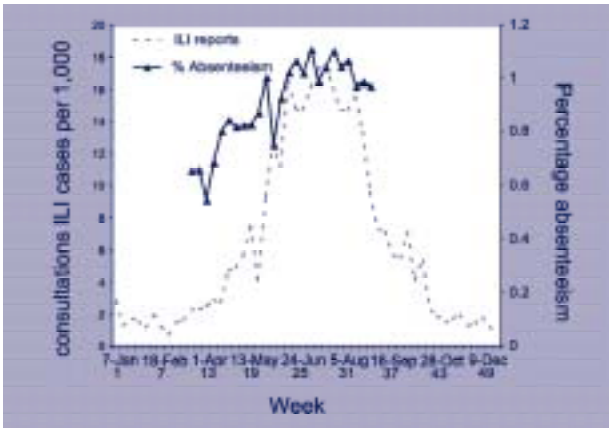
Figure 13. Influenza laboratory reports to LabVISE,notifications to NNDSS and consultation rates in ASPREN, Australia, 2002, by week of report



Absenteeism surveillance

Data supplied by Australia Post suggested an association between the peak in influenza activity and absenteeism. National absenteeism rates were highest at 1.1 per cent in weeks 29 and 30, which coincided with the peak in ASPREN reports of consultation of influenza-like illness (Figure 14).

Figure 14. Rates of absenteeism and consultation rates for influenza-like illness, Australia, 2002, by week of report



Hospitalisation due to influenza

In 2000–01 there were a total of 2,380 separations in Australian hospitals for influenza. Six hundred and fifty-five of these were cases in which the influenza virus was identified. Altogether influenza was responsible for 9,825 hospital days in 2000–01.

Discussion

In 2002, influenza activity in Australia was moderately increased compared with 2001, as assessed by all surveillance systems. This increase was more evident in temperate regions of Australia than in tropical regions. Although influenza A was predominant, there was increased activity of influenza B compared with 2001. Influenza A and B peaked at different periods, with influenza B peaking early in the flu season (early June) and influenza A peaking later in the season (late July).

Surveillance of influenza in Australia is based on a number of different systems. Although there are continuing problems in comparing data from different surveillance systems, there were some improvements in 2002. The addition of laboratory-confirmed influenza to the NNDSS has lead to a marked increase in the numbers of laboratory isolates of the influenza virus available for analysis. The need for timely data has been addressed by the fortnightly publication of data during the influenza season on the Communicable Diseases Australia Website (http://www.cda.gov.au). This allows the wide dissemination of information on the evolving dimensions of the annual influenza epidemic and provides data for public health action as required. The need to assess circulating influenza strains in a timely manner was demonstrated by the WHO Collaborating Centre for Reference and Research on Influenza during 2002, when the emergence of the B/Hong Kong strain was identified early in the season and assessments of the protective efficacy of the 2002 vaccine were undertaken rapidly.

In Australia in 2002, influenza A isolates were almost entirely H3N2 strains with only a small number of strains showing evidence of genetic variation and were closely related to the A/Panama/2007/99 vaccine strain. The virtual absence of H1N1 strains was in sharp contrast with the predominance of this strain in 2001.[7] Similar low levels of A/H1N1 were observed between 1996 and 1998.[10] The sequence of the single A/H1N1 was shown to have a haemagglutinin related to the vaccine strain, but a neuraminidase related to A/H3N2. This reassortment lineage (H1N2) was detected for the first time in the United Kingdom during the 2001–02 winter, when it made up 54 per cent of all influenza A viruses isolated.[11] The H1N2 virus was isolated more frequently from children aged less than 15 years of age but there was no evidence of the strain causing a pandemic or more severe disease. Preliminary data from the UK for the 2002–03 influenza season, indicates that H1N2 viruses comprised a small proportion of influenza viruses identified to date.[12] In the United States of America, in the 2002–03 season, preliminary data indicated that H1N2 influenza comprised 24 per cent of the strains characterised.[13]

Influenza B isolates showed evidence of a genetic reassortment with haemagglutinin closely related to B/Hong Kong/330/2001 and neuriminidase sequences related to the B/Sichuan/379/99. Similar viruses were identified in the Northern Hemisphere during the 2001–02 winter. Despite differences between the B/Hong Kong/220/2001 viruses and the B/Sichuan/379/99 vaccine strain used in the 2002 Australian vaccine, there was evidence that protective antibody titres were induced in a proportion of vaccinees. There were two reports of outbreaks of influenza due to the B/Hong Kong virus in Victoria, affecting approximately 136 individuals (Graham Tallis and Kerry-Ann O'Grady, Department of Human Services, personal communication).

Reports of the B/Hong Kong strain were also made in 2002 in New Zealand,[14] and in 2002–03 in the UK and the USA.[12,13]

Influenza vaccination of vulnerable populations such as the elderly is important to reduce the morbidity and mortality of annual influenza epidemics. The National Health and Medical Research Council recommends annual vaccination for influenza for all Australians aged over 65 years. A national telephone survey in October and November 2002 showed 76.9 per cent of Australian aged over 65 years received influenza vaccination in 2002.[15] This is a similar vaccination rate in this age group to that in 2001. A recent analysis of the protective efficacy of the influenza vaccine in Americans aged 65 years and older over two influenza seasons, demonstrated a reduction in hospitalisation for pneumonia or influenza of 29–33 per cent and a reduction in deaths from all causes of 48–50 per cent. The same study also noted a significant reduction in hospitalisations for cardiac disease and stroke in the same population.[16] An analysis of mortality associated with influenza and respiratory viruses in the USA showed an increase in influenza-associated death, probably the result of an ageing population.[17] These studies reinforce the need for continued annual influenza vaccination of the elderly.

In response to the emergence of this new influenza B, the recommended Australian influenza vaccine for 2003 has been changed to incorporate B/Hong Kong/330/2001 in place of B/Sichuan/379/99. The influenza A strains in the 2003 vaccine remain unchanged (A/New Caledonia/20/99(H1N1) and A/Moscow/10/99 (H3N2)).

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