Influenza in the Pacific

ANNE KELSO1,2 AND PATRICK C. READING1

World Health Organization Collaborating Centre for Reference and Research on Influenza and Department of Microbiology and Immunology, The University of Melbourne, Australia

SUMMARY

Influenza A and B viruses cause significant human disease worldwide through regular outbreaks and epidemics of seasonal influenza, and occasional pandemics when a novel influenza A virus emerges. Whereas Australia and New Zealand have well-established systems for community and laboratory-based surveillance of influenza, most other countries of the Pacific are only beginning to develop such systems with the support of various global and regional agencies and networks. Here we describe the role of the World Health Organization Global Influenza Surveillance Network and other organizations in laboratory-based influenza surveillance in the region and review some of the available data on seasonal and pandemic influenza in the developed and developing countries of the Pacific. The particular features of the Pacific Island countries and territories as small dispersed island communities, together with the greater susceptibility of indigenous people to the severe effects of influenza, highlight the importance of developing local laboratory-based surveillance systems. Such systems will improve the understanding, detection and control of seasonal influenza while also providing early warning of the emergence of potential pandemic viruses.

Introduction

Human-adapted influenza A and influenza B viruses circulate continuously in the world, causing significant mortality, morbidity and economic loss. It is estimated that as many as 5%-10% of the world’s population are infected with an influenza virus each year, resulting in 3-5 million severe cases and about 250,000-500,000 deaths globally per annum (1). Although infection can induce a long-lived protective antibody response, population immunity selects for variant influenza viruses whose major surface proteins, the haemagglutinin (HA) and the neuraminidase (NA), have mutated sufficiently to avoid antibody neutralization (‘antigenic drift’). Such seasonal influenza viruses cause outbreaks and epidemics in many areas of the world each year.

New influenza A viruses, not previously adapted to humans, emerge at irregular intervals and cause pandemics. Molecular evolutionary analyses of the viruses from the last four pandemics, in 1918-1919 (reconstructed viruses), 1957, 1968 and 2009, have indicated that all arose by reassortment of the genomes of avian, swine and/or human influenza viruses, introducing a new HA and, in most cases, a new NA to which the great majority of the global human population lacked protective immunity (‘antigenic shift’) (2). Because most people are immunologically naive to the pandemic virus, a large proportion of the world’s population may be infected within the first year or two of its emergence.

Information about the impact of seasonal and pandemic influenza viruses on countries of the Pacific is highly variable in scope and depth. Most data are available for the industrialized countries of Australia and New Zealand, which have well-established infectious disease surveillance and health

1 World Health Organization Collaborating Centre for Reference and Research on Influenza, 10 Wreckyn St, North Melbourne, Victoria 3051, Australia
2 anne.kelso@influenzacentre.org
care systems. Important data have also been collected from time to time in less developed countries, often through the singular efforts of individual researchers and institutions. However, with recognition of the potential impact of an influenza pandemic, highlighted by the emergence of severe acute respiratory syndrome (SARS) in 2003 and the re-emergence of highly pathogenic avian A(H5N1) influenza in 2003-2004, recent years have seen the strengthening of surveillance networks in the Pacific as elsewhere.

Here we outline the role of the World Health Organization (WHO) and other international and regional institutions in influenza surveillance in the Pacific, then review some of the published and other available data on the impact of seasonal and pandemic influenza in this region. Finally we try to draw these threads together to identify some of the significant questions and needs for influenza surveillance, particularly in less developed countries and territories of the Pacific.

Influenza surveillance in the Pacific

Effective laboratory-based surveillance systems for influenza, as for many other infectious diseases, are limited in the Pacific outside Australia and New Zealand. The remoteness, individuality and scarce resources of some of the 22 Pacific Island countries and territories (PICTs) have impeded the development of such systems. With little access to influenza vaccines and many other challenges to the delivery of health care, it is not surprising that influenza has not been a priority. Nevertheless, laboratory-based surveillance has been established in some countries with the support of international networks, as outlined below.

The WHO Global Influenza Surveillance Network

Recognizing the potential for influenza viruses to cause devastating pandemics as in 1918-1919, the WHO Global Influenza Surveillance Network (GISN) was established in 1948 to facilitate the early detection of new influenza virus variants (3). By late 2010, the network had expanded to include 135 WHO National Influenza Centres (NICs) in 105 countries, 5 WHO Collaborating Centres for Reference and Research on Influenza (CCs), 11 WHO H5 Reference Laboratories and 4 Essential Regulatory Laboratories. This large and growing network is coordinated by the Global Influenza Programme at the WHO’s headquarters in Geneva.

WHO NICs are usually national or provincial diagnostic or reference laboratories. Their roles in GISN are to undertake influenza surveillance within their country, to report regularly on influenza prevalence to the WHO and to send a selection of recent influenza virus-containing clinical specimens or virus isolates to one of the WHO CCs in London, Atlanta, Melbourne, Tokyo or Beijing.

At the WHO CC, the viruses undergo detailed antigenic and genetic characterization, antiviral drug susceptibility testing and other analyses as required. The collective data from the many thousands of viruses analysed by the WHO CCs each year enable the major lineages of influenza A and B viruses circulating in humans to be determined and reveal the emergence and spread of, for example, antigenic drift variants and drug-resistant strains. WHO CCs also isolate some viruses directly from clinical specimens into embryonated hens’ eggs in order to have a suite of potential vaccine candidate strains at hand in the event that seasonal influenza vaccines require updating because of antigenic drift in one or more of the three component viruses.

The five WHO CCs, along with representatives of the H5 Reference Laboratories and the Essential Regulatory Laboratories and other experts, meet in February and September each year to review the available data and to assist WHO in making recommendations on suitable virus strains to be included in influenza vaccines for the coming winter in the northern hemisphere and the southern hemisphere, respectively (4). If strain changes are recommended, appropriate candidate vaccine viruses isolated in the WHO CCs are distributed to the vaccine manufacturers.

The ability to update influenza vaccines with contemporary circulating viruses is one of the benefits of the virological surveillance undertaken by GISN. Of even greater global importance is the frequent and increasingly comprehensive monitoring of influenza viruses circulating in humans. This allows
the rapid detection and reporting of significant changes in seasonal influenza viruses—such as the emergence and rapid global spread of oseltamivir-resistant seasonal H1N1 viruses in 2007-2008 (5,6)—and, most importantly, increases the chance of early detection of a novel influenza A subtype with pandemic potential.

GISN and other contributing laboratories in the Pacific

In addition to the WHO CC in Melbourne, there are eight WHO NICs in the Pacific (Figure 1). Five of these are in Australia and New Zealand (at the Victorian Infectious Diseases Reference Laboratory in Melbourne, the Institute of Clinical Pathology and Medical Research in Sydney, PathWest in Perth, the Institute of Environmental Science and Research (ESR) in Wellington and Auckland Hospital in Auckland). Another three are in Fiji (at the Center for Communicable Disease Control, Mataika House, in Suva), New Caledonia (at the Pasteur Institute in Noumea) and Papua New Guinea (PNG) (at the PNG Institute of Medical Research in Goroka). All of the WHO NICs in the Pacific are active in submitting a selection of influenza viruses, either as clinical specimens or cultured isolates, to the WHO CC in Melbourne.

Laboratory-based influenza surveillance in the Pacific is greatly enhanced by the activities of a number of other public health laboratories in the region which are not currently designated as WHO NICs but have developed with strong support from other regional institutions (Figure 1). The Secretariat of the Pacific Community (SPC) is an intergovernmental organization that provides technical and policy advice to PICTs (7). In collaboration with partner organizations, including the Pasteur Institute of New Caledonia (IPNC) and the WHO CC in Melbourne, SPC has coordinated and implemented a program funded by the United States (US) Centers for Disease Control and Prevention (CDC) aimed at improving influenza surveillance in the Pacific. This project has allowed PICTs to improve the assessment of influenza burden, their laboratory-based surveillance and their pandemic influenza preparedness (8). Other institutions, such as the Pacific Paramedical Training Centre.
Although only about 40 deaths per annum Pacific enter the GISN.

Seasonal influenza in the Pacific

The health and economic burden of disease from influenza in the Pacific is likely to be significant. In Australia, for example, one analysis attributed an annual average of 310,000 general practitioner consultations and 18,400 hospitalizations to influenza at a direct cost to government of $115 million (10). Although only about 40 deaths per annum are directly attributed to influenza in Australia, the impact on mortality from other conditions is understood to be much higher. Little comparable information is available for most other countries of the Pacific. In general, however, both disease severity and mortality from influenza are likely to be higher in less developed communities due to such factors as concomitant infections, poorer nutrition, limited access to health care, the absence of community influenza vaccination programs, different approaches to health and the impact of community lifestyle on virus transmission.

Isolated populations of more remote PICTs may be at particular risk because their infrequent exposure to influenza viruses results in lower cross-reactive immunity induced by earlier virus strains. For example, in 1964, an A(H2N2) outbreak occurred on several isolated islands in the Yap district: few inhabitants had pre-epidemic antibodies to this subtype and almost the entire population was affected, with high morbidity and a case fatality rate of 1%-6.5% (11). In 1964 also, serological evidence was obtained that the population of the Caroline island of Fais had not been exposed to influenza A viruses since 1924 when the 1918 pandemic virus first reached this remote place (12), or to influenza B viruses since 1940 (11). Such prolonged lack of exposure to influenza viruses may be less common today when few communities are truly isolated from the rest of the world.

In PNG, influenza has been detected in the wet and dry seasons and may cause sporadic outbreaks and large epidemics, despite low overall population density and the remoteness of many villages (13). A serological survey conducted amongst residents of 47 remote villages in the Western Province in 2001-2002 confirmed circulation of type A (H1N1 and H3N2) and type B viruses, with some differences noted in prevalence rates between different villages; the peak of seroprevalence was consistent with increased influenza activity during the wet season (16). The impact of isolation on immunity noted above may account for the observation in 1964 that disease caused by a
type B influenza virus became more severe as it spread from the east coast into the highlands, where it was associated with over 100 deaths (13).

Analyses of viruses submitted by various PICTs to the Melbourne WHO CC indicate that the influenza virus strains circulating throughout the Pacific are generally similar to those isolated elsewhere in the world (unpublished data). Indeed on several occasions over the last 15 years, viruses isolated by the Melbourne WHO CC from specimens provided by laboratories in the Pacific have been recommended by WHO for inclusion in seasonal influenza vaccines for the northern and southern hemispheres (Table 1).

**Pandemic influenza in the Pacific**

Influenza pandemics differ from seasonal epidemics in several ways. The overall burden of influenza is likely to be markedly higher in a pandemic than during inter-pandemic periods. Pandemic influenza may occur outside the normal influenza season and with two or more waves of infections within a year. Pandemics also alter the circulation of seasonal viruses: in the three pandemics of the 20th century, the previously circulating influenza A virus subtype was rapidly replaced by the new virus which then, after a period of pandemic spread, settled down into a seasonal pattern of circulation until it in turn was replaced by the next pandemic virus. Thus, H1N1 probably replaced an H3-like virus in 1918-1919, H2N2 replaced H1N1 in 1957 and H3N2 replaced H2N2 in 1968. The re-emergence of H1N1 in 1977, probably from a laboratory, did not cause a global pandemic nor did it lead to replacement of H3N2, presumably because most of the world’s population had been exposed to related viruses before 1957.

**The 20th century pandemics**

The pandemics of the last century demonstrated that the impact of new influenza

---

**TABLE 1**

**Viruses from Pacific countries recommended by the World Health Organization for inclusion in seasonal influenza vaccines**

<table>
<thead>
<tr>
<th>Type (subtype)</th>
<th>Influenza virus</th>
<th>WHO vaccine recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td>A(H1N1)</td>
<td>A/New Caledonia/20/99</td>
<td>SH 2000 – 2007 \n NH 2000/1 – 2006/7</td>
</tr>
<tr>
<td></td>
<td>A/Solomon Islands/3/2006</td>
<td>NH 2007/8 \n SH 2008</td>
</tr>
<tr>
<td></td>
<td>A/Brisbane/59/2007</td>
<td>NH 2008/9 – 2009/10 \n SH 2009</td>
</tr>
<tr>
<td></td>
<td>A/Wellington/1/2004</td>
<td>SH 2005</td>
</tr>
<tr>
<td></td>
<td>A/Perth/16/2009</td>
<td>SH 2010 – 2011 \n NH 2010/11</td>
</tr>
</tbody>
</table>

WHO = World Health Organization
SH = southern hemisphere
NH = northern hemisphere
Dates indicate the winters to which the recommendations apply
A viruses on mortality and morbidity can vary markedly between pandemics and between populations. Estimated deaths attributed to the pandemic viruses ranged from about 1 million in 1968 to 50 million in 1918-1919 (about 2.5% of the world’s population), due at least in part to differences between the viruses themselves. However, the large literature on these pandemics also highlights the markedly higher mortality experienced in developing countries compared with the developed world, in isolated compared with urbanized communities and, in some countries, in indigenous compared with non-indigenous communities (17). For example, it is estimated that the 1918-1919 pandemic virus killed about 4.2% of the Maori population compared with 0.55% of the caucasian population in New Zealand (17).

The effects of the 1918 pandemic were particularly severe in some of the PICTs (reviewed in 18). Western Samoa (Samoa today) was probably the worst affected with over 7500 deaths, about 25% of the population, attributable to pandemic influenza. By contrast, neighbouring American Samoa introduced strict maritime quarantine and recorded no cases or deaths. Elsewhere in the Pacific, pandemic influenza claimed the lives of 16%, 6% and 5% of the populations of Tonga, Nauru and Fiji, respectively, while PICTs serviced exclusively by Australian vessels, including the Solomon Islands, Kiribati and Vanuatu, were spared due to maritime quarantine imposed on incoming and outgoing ships. Quarantine of arriving vessels protected Australia only until January 1919, after which the virus spread rapidly and caused approximately 12,000 deaths (0.24% of the population).

As noted above, the A(H2N2) epidemic in remote islands of the Yap district in 1964 was probably the first exposure of that population to the 1957 pandemic virus and mortality was high (11). During the 1968 pandemic, the new A(H3N2) virus quickly spread through many previously isolated areas of PNG, contributing to the deaths of more than 3000 people over a few months in 1969 (13).

The A(H1N1) 2009 pandemic

The 2009 influenza pandemic displayed both similarities with and differences from the 20th century pandemics:

- The speed and intensity of modern airline travel ensured that the virus reached most major ports within a few weeks of its first detection in Mexico and the USA in late April. Cases were confirmed in all continents within 14 weeks of the first known case in Mexico.

- The new virus quickly became the predominant influenza virus circulating in humans. Within a few months, the previous seasonal A(H1N1) viruses had almost disappeared.

- The virus preferentially affected younger people, especially children and teenagers. As a result, schools were a major avenue of spread and children were frequently the origin of household infections.

The experience of the 2009 pandemic in the Pacific mirrored that in many other parts of the world.

In Australia, there had been little seasonal influenza activity before the arrival of the pandemic virus. The first active case of A(H1N1) 2009 was identified in Melbourne in a traveller from the US on 20 May but the virus had probably been circulating there undetected and confirmed case numbers escalated rapidly from that point (19). While timing varied around the country, Australia overall experienced a single pandemic wave which peaked in the third week of July and then declined to baseline by September-October (20). The predominant seasonal influenza viruses detected in the 2009 winter were of the A(H3N2) subtype but their numbers were low compared with detections of the pandemic A(H1N1) 2009 virus. The first cases of pandemic influenza in New Zealand were detected in two high school groups who returned from North America on 25 and 28 April (week 18) (21,22). These cases were apparently contained and the pandemic wave started in earnest in weeks 24 and 25. Seasonal A(H1N1) viruses were also circulating in New Zealand at that time but were rapidly overtaken by A(H1N1) 2009 viruses.

Although rates of laboratory testing were high in Australia and New Zealand, especially early in the pandemic wave, the data suggested that many cases were mild and therefore would not have been formally diagnosed. As the pandemic progressed,
However, it also became apparent that some people suffered severe illness. About two-thirds of severe and fatal cases were associated with risk factors, including pregnancy, asthma, chronic obstructive pulmonary disease, diabetes, obesity, malignancy and immunosuppressive medication; a significant proportion of the remaining cases were in previously healthy, young people (23,24). While most health care systems coped well with the extra demands imposed by pandemic cases and policies, intensive care units in Australia and New Zealand carried an exceptional load of severely ill influenza patients, many requiring mechanical ventilation, with unprecedented extent and duration of use of extracorporeal membrane oxygenation to manage acute respiratory distress syndrome (23,25).

Indigenous people were disproportionately represented among serious cases: Aboriginal and Torres Strait Islander Australians were 10 times more likely to be hospitalized than other Australians (26) and Maori and Pacific Islander people were, respectively, 5 and 7 times more likely to be hospitalized than those of European origin (27).

Several authors have collated data from PICTs as outlined below. While case numbers might be under-reported in these countries compared with industrialized nations, it is likely that hospitalizations and deaths that occurred in hospitals have been comprehensively recorded in at least some of the PICTs.

The Western Pacific Regional Office of the WHO (28) noted that, during 2009, cases of A(H1N1)2009 were reported by all but three Pacific Island countries, Niue, Pitcairn Islands and Tokelau. Incidence was often very high in countries with small populations, including Cook Islands, the Marshall Islands, New Caledonia, Palau, Tuvalu, and Wallis and Futuna, where it ranged between about 200 and 540 per 100,000 population. Overall, PICTs recorded 21 deaths, or 0.22 per 100,000, compared with 0.08/100,000 for the whole Western Pacific Region. Most deaths, in the Pacific as elsewhere, were recorded in people aged 15-64 years.

In the French territories of the Pacific, New Caledonia, French Polynesia and Wallis and Futuna, the first pandemic wave commenced at a different time in each territory and then lasted approximately 8 weeks (29). Cases were detected among travellers from Australia, the USA and France but school exchanges with Australia and New Zealand and the return of students from holidays or study abroad appear to have contributed to community spread. Estimated attack rates ranged from 16-18% in New Caledonia to 38% in Futuna; numbers of hospitalizations and deaths were small and linked especially to diabetes, heart and lung disease, obesity, neuromuscular diseases in children and Oceanic origin.

The higher vulnerability of indigenous populations of the Pacific to pandemic influenza noted in 1918-1919 was observed again in 2009 and was apparently reflected in both clinical attack rates and the risk of severe disease and death (30). For example, in addition to the Australian and New Zealand experience noted above, the death rate among indigenous inhabitants of New Caledonia was 5.3 times higher than among non-indigenous people; the relatively high attack rates recorded in the French territories of the Pacific may therefore reflect the high proportion of indigenous people in those populations (30). Some of the contributing factors are likely to be the same as in earlier pandemics: for example, poverty, malnutrition, crowding, bacterial co-infection, high rates of pregnancy, poor access to health services and the small size of some islands. A role for genetic factors also has not been excluded. Other factors may differ from those of the past, such as obesity, diabetes, asthma and HIV-associated immunosuppression. However, the contribution of such factors may also vary between countries. For example, the incidence of diabetes, obesity and chronic respiratory diseases is higher among indigenous than non-indigenous people in Australia and New Caledonia, whereas the incidence of chronic respiratory disease is lower among Pacific peoples than Maori and others in New Zealand (27,30).

In recent years, the Melbourne WHO CC has received approximately 2500 influenza virus samples (clinical specimens and cultured virus isolates) per annum from around the Asia-Pacific region, with relatively small numbers from PICTs. Following the emergence of the pandemic virus, however, the WHO CC was requested by several countries to confirm first cases and subsequently received markedly elevated numbers of samples from a larger number of PICTs than in previous years (Table 2).
Detailed antigenic and genetic analyses of these viruses showed that they were closely related to those circulating in Australia, New Zealand and other parts of the world and would therefore be covered by pandemic vaccines containing the reference virus A/California/7/2009.

After the decline of the pandemic wave in September-October 2009, Australia and New Zealand detected only sporadic cases of influenza until the winter of 2010, when both countries experienced a moderate influenza season (31,32). In New Zealand the great majority of typed viruses were A(H1N1) 2009 while in Australia typed viruses comprised approximately 70% A(H1N1) 2009 with the remainder including both type B and, to a lesser extent, A(H3N2) viruses. Clinical reporting systems suggest that the virulence of circulating A(H1N1) 2009 viruses has not changed significantly from 2009 (32). The lower influenza prevalence reported in 2010 is likely to reflect lower testing rates and, more importantly, elevated population immunity to A(H1N1) 2009 due to exposure during 2009 or subsequent vaccination, as indicated by several recent serological surveys (eg, 33-35).

Recent information is lacking for most PICTs. However, New Caledonia has reported that influenza activity detected between July and September 2010 was mostly due to type B viruses (36). Compared with 2009, the WHO CC in Melbourne has received smaller numbers of samples from a more limited range of PICTs during 2010. Of those analysed at the time of writing, viruses from Fiji, Guam and PNG have mainly been A(H1N1) 2009 and the remainder (from the Federated States of Micronesia and New Caledonia) have been type B viruses.

These data support the idea that the 2009 pandemic virus is following a similar pattern

<table>
<thead>
<tr>
<th>Countries</th>
<th>2006</th>
<th>2007</th>
<th>2008</th>
<th>2009</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of PICTs submitting viruses</td>
<td>3</td>
<td>4</td>
<td>7</td>
<td>15</td>
</tr>
<tr>
<td>Number of samples received from:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fiji</td>
<td>0</td>
<td>0</td>
<td>5</td>
<td>465</td>
</tr>
<tr>
<td>French Polynesia</td>
<td>20</td>
<td>6</td>
<td>0</td>
<td>59</td>
</tr>
<tr>
<td>Guam</td>
<td>0</td>
<td>21</td>
<td>19</td>
<td>275</td>
</tr>
<tr>
<td>New Caledonia</td>
<td>40</td>
<td>16</td>
<td>2</td>
<td>38</td>
</tr>
<tr>
<td>Papua New Guinea</td>
<td>0</td>
<td>8</td>
<td>39</td>
<td>223</td>
</tr>
<tr>
<td>Solomon Islands</td>
<td>3</td>
<td>0</td>
<td>13</td>
<td>39</td>
</tr>
<tr>
<td>Total from selected PICTs</td>
<td>63</td>
<td>51</td>
<td>78</td>
<td>1099</td>
</tr>
<tr>
<td>Total from all PICTs</td>
<td>63</td>
<td>51</td>
<td>99</td>
<td>2074</td>
</tr>
</tbody>
</table>

*The table shows the total number of PICTs that submitted virus samples in the indicated years, the total numbers of samples received from PICTs and the numbers submitted by selected individual PICTs.*
of circulation to seasonal influenza viruses in the Pacific, as observed elsewhere in the world and consistent with the WHO's declaration of the end of the pandemic on 10 August 2010. A(H3N2) and type B viruses are co-circulating with A(H1N1) 2009 to varying extents in different countries while the previous seasonal A(H1N1) lineage is no longer being detected.

The WHO Consultation on the Composition of Influenza Vaccines for the Southern Hemisphere 2011, held in late September 2010, concluded, from detailed strain analyses of viruses received by the four WHO CCs and data submitted by NICs, that A(H1N1) 2009 viruses have not yet undergone significant antigenic drift and remain closely related to the vaccine strain A/California/7/2009 (36). This was also true for the A(H1N1) 2009 viruses recently submitted to the Melbourne WHO CC by countries of the Pacific. Although a new genetic subclade emerged in Australia, New Zealand and Singapore during 2010, it has remained antigenically similar to A/California/7/2009 to date (37).

**Strengthening influenza surveillance in the Pacific**

Although the available data indicate that both seasonal and pandemic influenza can cause significant illness in people of the Pacific, there are many gaps in our knowledge of this disease in the less developed countries of the region. These gaps include information on the contribution of influenza infection to the health and economic well-being of Pacific communities, on the seasonality of influenza activity, on major avenues of virus transmission and on clinical, environmental and cultural risk factors for severe disease and death from influenza infection. Such information would assist governments, aid agencies and other international authorities in allocating resources for effective influenza detection, prevention and treatment, particularly for the most vulnerable indigenous populations.

Laboratory-based surveillance of influenza infection is critical for obtaining this information, supported by robust communicable disease surveillance – for example, of influenza-like illness – in order to identify outbreaks and monitor disease burden (18). However, laboratories in the PICTs face significant challenges in the adoption of contemporary testing technologies: they often lack suitable facilities and equipment, laboratory technicians may have limited formal training, and they may not have access to external quality control and proficiency testing procedures. It is therefore important that laboratory techniques are appropriate to local conditions and are supported by training and external quality control. Work is also needed in other areas, including sample collection, storage and shipping to reference laboratories, and education of laboratory staff and clinicians in the use and limitations of different testing technologies for surveillance versus diagnosis and clinical management.

The work being undertaken by the WHO, SPC, PPHSN, ESR, CDC and other agencies and networks to establish laboratory and field surveillance for influenza deserves strong and continuing support. There is also an important role for the more established laboratories, not only in Australia and New Zealand, but also in, for example, Fiji, New Caledonia, PNG and Guam, in mentoring and supporting smaller PICT laboratories. The justification need not be altruistic. Strong laboratory-based surveillance is a cornerstone of pandemic preparedness, building local awareness of influenza and improving the chance of detecting and responding to an emerging outbreak in the region.

**ACKNOWLEDGEMENTS**

We particularly thank our colleagues in the WHO National Influenza Centres and other laboratories in the Pacific who submit samples to the Melbourne WHO Collaborating Centre to support the WHO Global Influenza Surveillance Network. We are also grateful to the Secretariat of the Pacific Community for allowing us to use a modified version of their map of the Pacific. The Melbourne WHO Collaborating Centre for Reference and Research on Influenza is supported by the Australian Government Department of Health and Ageing. Financial support was also received from the National Health and Medical Research Council (Program Grant No 567122).

**REFERENCES**

mediacentre/factsheets/fs211/en/index.html


10 Newall AT, Scuffham PA. Influenza-related disease: the cost to the Australian healthcare system. Vaccine 2008;26:6818-6823.


17 Mathews JD, Chesson JM, McCaw JM, McMvernon J. Understanding influenza transmission, immunity and pandemic threats. Influenza Other Resp Viruses 2009;3:143-149.


19 Kelly HA, Mercer GN, Fielding JE, Dowse GK, Glass K, Carcione D, Grant KA, Effer PV, Lester RA. Pandemic (H1N1) 2009 influenza community transmission was established in one Australian state when the virus was first identified in North America. PLoS One 2010;5:e11341.


22 Laurie K, Barr I, Kselo A, Jones N. Limited novel influenza A (H1N1) 09 infection in travelling high-school tour group. Influenza Other Resp Viruses, in press.


34 Gilbert GL, Cretikos MA, Hueston L, Doukas G, O’Toole B, Dwyer DE. Influenza A (H1N1) 2009 antibodies in residents of New South Wales, Australia, after the first pandemic wave in the 2009 southern hemisphere winter. *PLoS One* 2010;5:e12562.

