World Health Organization
Western Pacific Region

WPSAR
Western Pacific Surveillance and Response Journal

Volume 4, Number 4, 2013, Pages 1–29
p-ISSN: 2094-7321 e-ISSN: 2094-7313

Western Pacific Surveillance and Response

Open access journal with continuous publication

Western Pacific Surveillance and Response (WPSAR) is an open access journal dedicated to the surveillance of and response to public health events. The goal of the journal is to create a platform for timely information sharing both within our region and globally to enhance surveillance and response activities. WPSAR is a continuous publication which means articles will be published online as soon as they have completed the review and editing process. Every three months articles will be batched for a print issue. It is a publication managed by the World Health Organization Regional Office for the Western Pacific.

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p-ISSN: 2094-7321
e-ISSN: 2094-7313

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Efficient use of social media during the avian influenza A(H7N9) emergency response

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During the 2013 outbreak of human infections of avian influenza A(H7N9), the Centers for Disease Control and Prevention (CDC) used official data released by the World Health Organization (WHO) and the Chinese government to keep United States public health officials informed of updates of the outbreak.[1] The Chinese central government released official avian influenza A(H7N9) data via its web sites (e.g. National Health and Family Planning Commission[2]), their official news agency (Xinhua News Agency) and their official newspapers (e.g. *People's Daily*, Beijing). In addition, official avian influenza A(H7N9) information was released by Chinese provincial and municipal governments such as Shanghai Municipal Bureau of Health,[3] Jiangsu Department of Health[4] and Zhejiang Department of Health.[5] Prior studies have discussed the role of social media in the early detection of disease outbreaks[6–9] and the facilitation of community-level discussion.[10] In this perspective, we focus on the use of social media by public health agencies to disseminate and obtain official outbreak information during a public health emergency response.

Weibo (literally, microblog) is a category of Chinese microblogging sites that are similar to Twitter. Both Twitter and *weibo* are social media that allow users to post a 140-character long message online. *Weibo* has become popular in China since August 2009 when Twitter became unavailable to users in mainland China. As of December 2012, 309 million people were reported to be *weibo* users in China as compared to the global 500 million registered Twitter users as of July 2012. There are several different providers of *weibo*, including *Sina Weibo*, *Tencent (QQ) Weibo*, *Sohu Weibo*, *Baidu Weibo*, *ifeng Weibo*, *NetEase Weibo* and others. Most *weibo* users live in China; a random sample of users of *Sina Weibo* found that 1.6% of users were from countries other than China.[11]

Social media platforms provide a new channel through which public health agencies release official information, either by posting new outbreak information directly or by guiding people to official web sites. The 2013 H7N9 outbreak was the first time that WHO used Twitter for initial release of official outbreak information.[12] Likewise, the Chinese central government, some of its provincial and municipal governments and the Chinese official news agency released some official outbreak information via *weibo* nearly simultaneously with their web site press releases (the exact time of information release is known for *weibo* but often not for web sites; **Table 1**). An official list of Chinese provincial and municipal health authorities' *weibo* accounts can be found at the web site of the National Health and Family Planning Commission.[13] Social media, like Twitter and *weibo*, are used by WHO and the Chinese authorities to direct attention of online communities towards their official web site press releases (**Table 1**). *Weibo* users can also post text longer than 140 characters as an image attached to their *weibo* post, which is known as a long *weibo*. The Chinese government used this function to post press releases on *weibo*. An example of a long *weibo* post containing a whole press release by the Shanghai Municipal Government[14] can be found in **Table 1**.

Social media platforms can help CDC epidemiologists obtain official information more efficiently because

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Submitted: 17 September 2013; Published: 31 October 2013
doi: 10.5365/wpsar.2013.4.3.005

Table 1. **Examples of outbreak information released online and through social media by the World Health Organization and the Chinese national, provincial and municipal health authorities**

Organization	Social media/ web site	Title and content (web site address)	Date/time (if available)	Language
World Health Organization (WHO)	https://twitter.com/WHO (@WHO)	China has notified WHO of three human cases infected with #influenza A(#H7N9) http://goo.gl/Wf7Fy #Flufighter (https://twitter.com/WHO/status/318764531029516288)	1 April 2013 16:39:21 GMT	English
	http://www.who.int/	Human infection with influenza A(H7N9) virus in China. Global Alert and Response – Press release (http://www.who.int/csr/don/2013_04_01/en/index.html)	1 April 2013*	English
Chinese central government – National Health and Family Planning Commission	http://e.weibo.com/u/2834480301 (健康中国)	卫生和计划生育委员会就上海、安徽3例患者感染H7N9禽流感展开疫情答问http://t.cn/zTwy8Jw 确诊患者的所有密切接触者目前均未发现类似病例。 [Translated: The National Health and Family Planning Commission provides a Q&A session on epidemiologic information regarding the three patients infected with H7N9 avian influenza in Shanghai and Anhui (http://t.cn/zTwy8Jw) up to the present; there are no similar cases found among all the close contacts of the confirmed cases (http://e.weibo.com/2834480301/zq0QvBVsl).]	31 March 2013 11:38 GMT	Chinese
	http://www.moh.gov.cn/	上海、安徽发生3例人感染H7N9禽流感确诊病例 [Translated: three confirmed cases of human infections of H7N9 avian influenza in Shanghai and Anhui – Press release] (http://www.moh.gov.cn/mohwsyjbgs/s3578/201303/44f25bd6bed14cf082512d8b6258fb3d.shtml)	31 March 2013*	Chinese
Chinese provincial/ municipal government – e.g. Shanghai Municipal Government	http://e.weibo.com/shanghaicity (上海发布)	【上海两例人感染H7N9禽流感病例密切接触者未发现异常情况】#要闻#经国家卫生和计划生育委员会组织专家确诊并依法向社会公布，上海市发现2例人感染H7N9禽流感病例。截至目前，所有密切接触者均未发现类似症状和发病情况。今年以来上海流感、肺炎发病率与近三年同期相比并无明显上升。详见长微博。 [Translated: (Two cases of human infection of H7N9 avian influenza in Shanghai. Abnormal situation has not been observed among close contacts.) #Headline# The National Health and Family Planning Commission has organized experts to confirm and has followed the law to report to the society, that two cases of human infection of avian influenza have been discovered in Shanghai. Up to the present, there has not been any similar symptoms or disease onset observed among all close contacts. Since the beginning of this year, the clinical attack rates of influenza and pneumonia in Shanghai are similar to the same period in the past three years and no obvious increase has been observed. For details, see long *weibo* post.] (http://e.weibo.com/2539961154/zq0AU9VMR)	31 March 2013 11:00 GMT	Chinese
	http://wsj.sh.gov.cn/ http://www.smhb.gov.cn/	上海、安徽发生3例人感染H7N9禽流感确诊病例 [Translated: three confirmed cases of human infections of H7N9 avian influenza in Shanghai and Anhui – Press release] (http://wsj.sh.gov.cn/website/b/103667.shtml)	31 March 2013*	Chinese

* The precise release time for the official press releases was not available as the webpages did not carry a stamp of their release time. Nonetheless, based on our experience, the online press releases and the social media posts were released by WHO and the Chinese authorities nearly simultaneously.

information from multiple sources can be obtained from a central access point. During the avian influenza A(H7N9) outbreak, a team at CDC followed the social media accounts of multiple official sources so that new outbreak information from WHO and the Chinese health authorities would automatically come to the team's attention. When new case data were released by WHO or the Chinese government at its national, provincial or municipal level via Twitter and/or *weibo*, the event was re-tweeted by social media users; thus even a message originating on a Chinese-language web site of a provincial health department would be rapidly noticed worldwide and quickly rise to the team's attention. While these social media posts might include information that was already available elsewhere, they did alert epidemiologists to the release of new

information through official sources, allowing the team to gather additional information from official web sites if available (**Table 1**) and obviating the need for constant monitoring of multiple news sources and web sites, such as individual web sites of the many local Chinese health departments.

The use of Chinese social media, like *weibo*, coupled with the necessary Chinese language and cultural knowledge, enabled CDC epidemiologists to gather the Chinese official data so that it could be translated, contextualized and interpreted in an efficient manner during the A(H7N9) emergency response. To ensure timely and complete understanding of an outbreak situation, it may be helpful for epidemiologists to track social media, including Twitter and *weibo*, in addition to traditional methods of communication.[7] Our experiences in the 2013 avian influenza A(H7N9) outbreak could be relevant to other outbreaks in other countries and to public health agencies of other nations.

Disclaimer

The findings and conclusions in this report are those of the authors and do not necessarily represent the official position of the CDC.

Conflicts of interest

None declared.

Funding

None

Acknowledgement

We want to thank the team members: Fiona Havers, Su Su, Kira Christian and the Global Disease Detection Operations Center, Center for Global Health. We thank Fiona Havers, Wenkai Li, Martin I Meltzer, Scott Santibanez and Michael Washington for their comments on earlier versions of this manuscript.

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13. *Weibo matrix*. Beijing, National Health and Family Planning Commission, 2013 (http://www.moh.gov.cn/zhuzhan/wbjz/weibo.shtml, accessed 16 October 2013).
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Invasive meningococcal disease in elderly people, New South Wales, Australia, 1993 to 2012

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Little information is available publicly on invasive meningococcal disease (IMD) in elderly people in Australia. This study analysed IMD notifications data from New South Wales between 1993 and 2012 to determine the distribution of IMD among people aged 65 years and older and to describe the characteristics of IMD in this age group compared to younger age groups with respect to notification trends, serogroup distribution and mortality rates. Following introduction of a childhood vaccination programme against meningococcal type C in 2003, notification rates in all age groups decreased, but the proportion of IMD notifications in people aged 65 years and over rose significantly (from 4% to 6%, $P = 0.01$). Mortality rates from IMD in those aged 65 years and older were significantly higher than overall rates (32% compared to 5%, $P < 0.01$). Serogroup Y accounted for 23% of infections in the elderly compared to 3% in people aged under 65 years ($P < 0.01$). As the population ages, the elderly may account for a higher number of IMD cases in Australia. Protocols at the state and national level should be updated to provide guidance on the clinical and public health management of elderly people with IMD.

Invasive disease caused by *Neisseria meningitidis* occurs when bacteria enter a normally sterile site such as blood (causing septicaemia) or cerebrospinal fluid (causing meningitis).[1] Transmission is by respiratory droplets and up to 10% of the population may harbour *Neisseria meningitidis* in their nasopharynx without disease.[1] Asymptomatic carriage is higher in household contacts of patients with meningococcal disease (12.4%).[2] In Australia, the highest rates of invasive meningococcal disease (IMD) are among children under 5 years and young adults between 15 and 24 years of age.[3]

Five serogroups (A, B, C, W135 and Y) are responsible for the majority of IMD worldwide,[4] with serogroups B and C the most commonly reported in Australia.[1] Serogroup C vaccine is the only meningococcal vaccine routinely offered to all children in Australia under the National Immunization Programme. The national meningococcal C vaccination programme, introduced in Australia in January 2003, comprises ongoing inclusion of the vaccine in the National Immunization Programme schedule at one year of age, as well as a catch-up programme until 2006 for children aged 2–19 years.[3,4] Large decreases have since been observed in serogroup C infections, with a 92% decline in the number of notifications of this serotype in the 15–24 year age group.[4] Across all age groups, notification of IMD in Australia decreased by half from a rate of 2 per 100 000 to 1 per 100 000 population between 2004 and 2010.[1]

The state of New South Wales (NSW; the largest Australian state) has a population of over 7.2 million; 1 million (14%) are aged 65 years and over. The population is ageing, and the number of people in this age group grew by 22% between 2001 and 2011.[5] Throughout Australia, the proportion of the population aged 65 years and over is projected to increase to just under a quarter of the total population by 2056.[6] Confirmed and probable cases of IMD in NSW have been notifiable by clinicians and laboratories since 1990 under the NSW Public Health Act (**Box 1**). Case notification and surveillance data are entered into the NSW Notifiable Conditions and Information Management System, including details on age and serogroup information where available.[7]

Little has been reported on IMD incidence in the elderly internationally, with no reports published in Australia. In late 2012, the Hunter New England Local

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Submitted: 30 September 2013; Published: 9 December 2013
doi: 10.5365/wpsar.2013.4.4.001

Box 1. **Meningococcal disease case definitions, New South Wales, Australia**

Case	Definition
Confirmed	Requires either laboratory definitive evidence, OR laboratory suggestive evidence AND clinical evidence. Laboratory definitive evidence • Isolation of *Neisseria meningitidis* from a normally sterile site; OR • Detection of specific meningococcal DNA sequences in a specimen from a normally sterile site by nucleic acid amplification testing. Laboratory suggestive evidence • Detection of Gram negative diplococci in Gram stain of specimen from a normally sterile site or from a suspicious skin lesion; OR • High titre IgM or significant rise in IgM or IgG titres to outer membrane protein antigens of *Neisseria meningitidis*. Clinical evidence • A disease which is compatible with invasive meningococcal disease in the opinion of the treating clinician.
Probable	Requires clinical evidence only: • The absence of evidence for other causes of clinical symptoms; AND EITHER • Clinically compatible disease including haemorrhagic rash; OR • Clinically compatible disease AND close contact with a confirmed case within the previous 60 days.

Health District in northern NSW was notified of a case of IMD in a resident of an aged care facility. The response to this case revealed a lack of published guidance with respect to the public health management of IMD in the elderly, particularly in the definition of close contacts and administration of clearance treatment.

The aim of the current epidemiological investigation was to examine IMD trends in NSW with a particular focus on the proportion of notifications in people aged 65 years and over, mortality rates and serogroup distribution. Implications for the prevention and management of IMD are also discussed.

METHODS

IMD notification data for the period 1993 to 2012 for NSW were sourced from the NSW Ministry of Health. Analysis was performed using Microsoft Excel, Statistical Analysis Software (SAS) Enterprise Guide 5.1 (SAS Institute Inc. Seattle, WA, USA) and STATA 11 (Stata Corp. 2009, College Station, TX, USA). IMD notification rates were calculated using mid-year NSW population data from the Australian Bureau of Statistics and proportions were compared using the chi-squared (χ^2) test.

Annualized notification rates by age groups (10–14, 0–14, 15–24, 25–64 year olds and people aged 65 years and over) were compared over two periods, 1993 to 2002 and 2003 to 2012, corresponding to periods before and after introduction of the national childhood meningococcal C vaccination programme in 2003. The 0–14 year age group was further divided into 0–4, 5–9 and 10–14 years. Wider age ranges were chosen to facilitate comparison between people aged 65 years and over and younger age groups, particularly those aged under 25 who are considered most at risk of IMD. Rate ratios and 95% confidence intervals were calculated for each age group to compare 2003 to 2012 notification rates with 1993 to 2002.

Analysis by serogroup was conducted for 2003 to 2012, the years for which serogroup information was recorded for more than 70% of notifications; cases diagnosed by polymerase chain reaction were not further typed. Case fatality rates (CFRs) were also limited to 2008 and 2012 due to missing data before this time.

This project used de-identified data routinely collected under the NSW Public Health Act. The project was further deemed a quality improvement exercise and an ethics waiver was granted by the Hunter New England Local Health District Human Research Ethics Committee.

RESULTS

Notifications by age group

Between 1993 and 2012, there were 2995 notifications of IMD in NSW. Annual notifications peaked at 253 in 2000, decreasing to 66 in 2012 (**Figure 1**). The age group with the highest number of notifications each year was 0–14 year olds; the lowest was observed for the 65 years and over age group (**Figure 1**).

Over the 20-year period, there were 153 notifications (5.1%) in those aged 65 years and over. The

Figure 1. **Invasive meningococcal disease notifications by year and age group, New South Wales, Australia, 1993 to 2012**

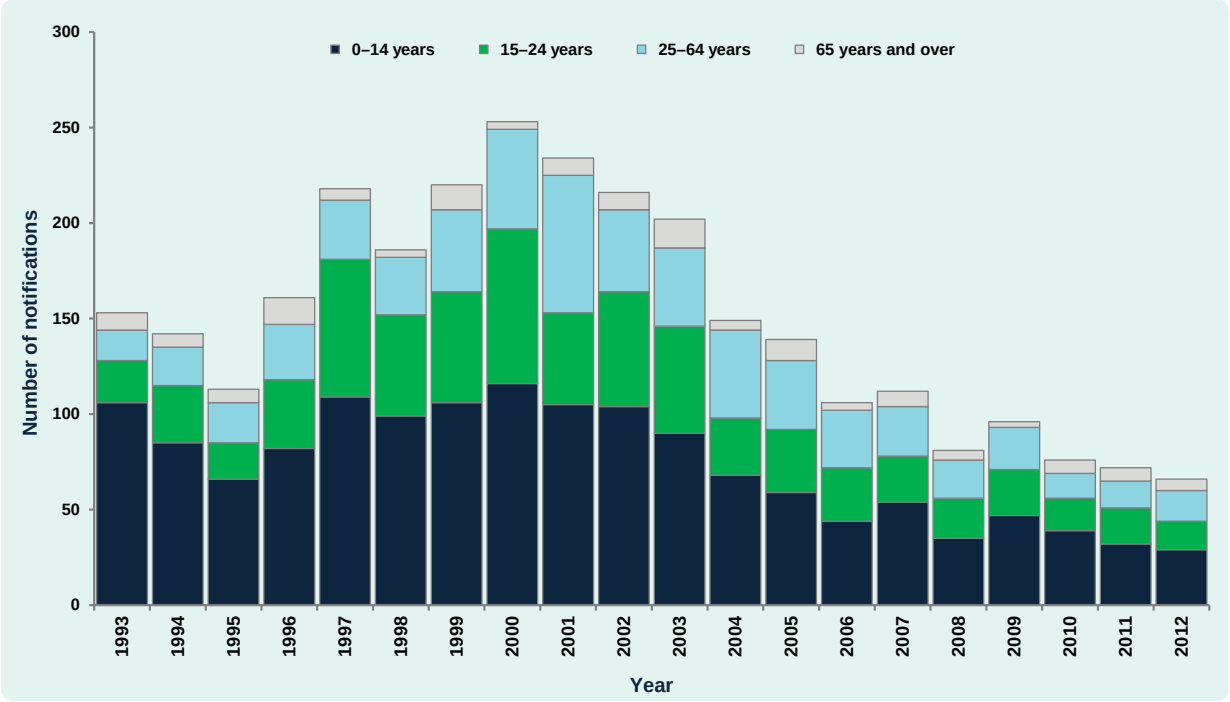


Table 1. **Changes in invasive meningococcal disease notification rates by age group, New South Wales, Australia, 1993 to 2002 and 2003 to 2012**

Age group (years)	1993 to 2002			2003 to 2012			Rate ratio (95% confidence interval)	*P*-value
	n	%	Rate per 100 000	*n*	%	Rate per 100 000		
0–14	978	51.5	74.1	497	45.2	37.04	0.50 (0.45–0.56)	< 0.001
0–4	*701*	*36.9*	*159.8*	*371*	*33.8*	*82.79*	*0.52 (0.46–0.59)*	*< 0.001*
5–9	*151*	*8.0*	*34.1*	*76*	*6.9*	*17.18*	*0.50 (0.38–0.66)*	*< 0.001*
10–14	*126*	*6.6*	*28.7*	*50*	*4.5*	*11.08*	*0.39 (0.28–0.53)*	*< 0.001*
15–24	479	25.3	54.3	267	24.3	28.34	0.52 (0.45–0.61)	< 0.001
25–64	357	18.8	10.8	264	24.0	7.12	0.66 (0.56–0.77)	< 0.001
65 and over	82	4.3	10.2	71	6.5	7.37	0.72 (0.53–0.99)	0.045
Total	**1896**	**100.0**	**30.0**	**1099**	**100.0**	**15.81**	**0.53 (0.49–0.57)**	**< 0.001**

proportion of IMD notifications for those aged 65 years and over significantly increased from 4% in the period 1993–2002 to 6% in the period 2003–2012 ($P = 0.01$). Although annualized rates per 100 000 population decreased significantly across all age groups between 1993–2002 and 2003–2012, the decrease was primarily for younger age groups compared with those aged 65 years and over (**Table 1**).

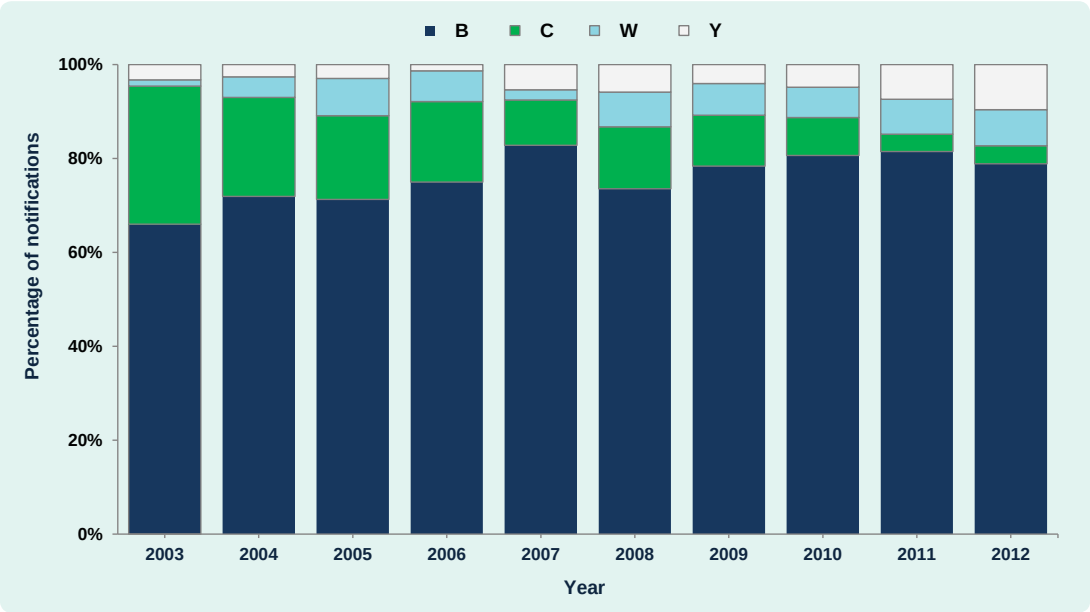
Case fatality rates

Mortality data were available for 366 of 390 IMD notifications between 2008 and 2012, with 19 deaths recorded: three in 0–14 year olds, one in 15–24 year olds, 10 in 25–64 year olds and six in those aged 65 years and over. The CFR for those aged 65 years and over was significantly higher than the overall CFR (32% [6/19] compared with 5% [19/366]; $P < 0.01$) and also than the CFR for the 0–4 year old age group (1% [3/127]; $P < 0.01$), those historically at greatest risk of mortality.

Notifications by serogroup

Between 2003 and 2012, serogroup information was available for 64/71 (90%) notifications in those aged 65 years and over and for 787/1028 (77%) notifications in people under 65 years of age. During this period, there

Figure 2. **Invasive meningococcal disease notifications by year and serogroup, New South Wales, Australia, 2003 to 2012**



was a gradual reduction in the proportion of infections caused by serogroup C and an increase primarily in the proportion caused by serogroup B (**Figure 2**).

Of the 64 notifications in those aged 65 years and older, 30 (47%) were serogroup B, 10 (16%) were serogroup C, 15 (23%) were serogroup Y and 9 were (14%) serogroup W. For those under 65 years of age, 605 (77%) were serogroup B, 125 (16%) were serogroup C, 21 (3%) were serogroup Y and 35 (4%) were serogroup W. The difference in proportions attributable to serogroup Y infection between the two age groups was statistically significant ($P < 0.01$).

Across all age groups, the proportion of IMD notifications attributable to serogroup Y significantly increased from 3% (17/539) in the period 2003–2007 to 6% (19/312) in the period 2008–2012 ($P > 0.05$). For those aged under 65 years, the proportion of notifications attributable to serogroup Y experienced a slight but steady increase between 2003 and 2012, while for older age groups there was variability from year to year (**Figure 3**).

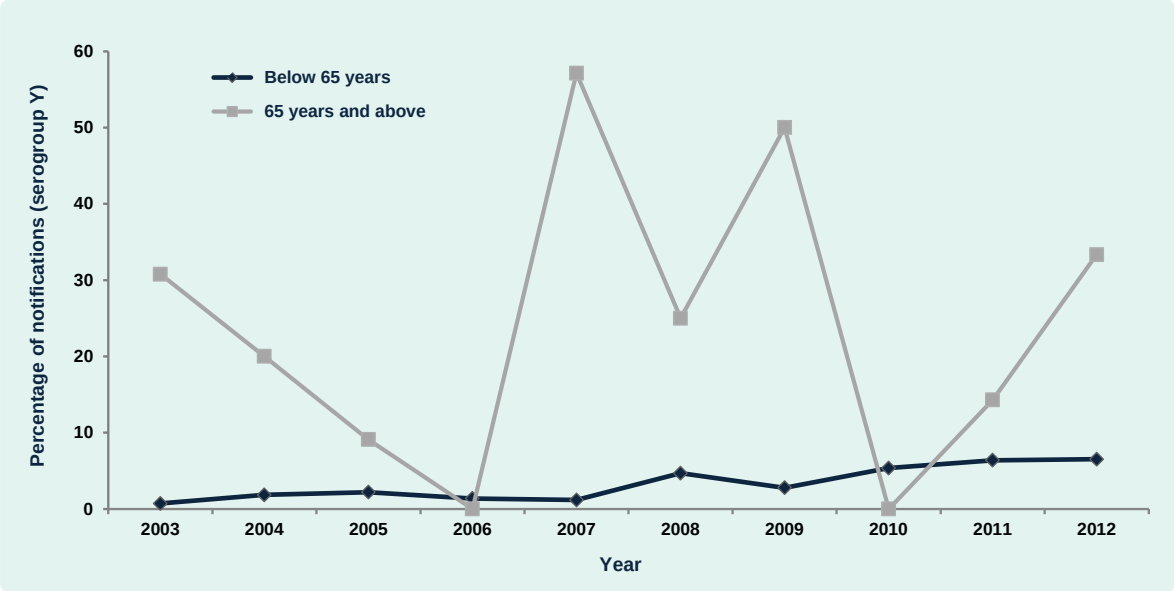
DISCUSSION

The absolute number of IMD cases in NSW residents aged 65 years and over has remained low over the past 20 years, and rates for all age groups have decreased, most likely as a result of the herd immunity effect of the childhood meningococcal C vaccination programme. However, the 65 years and over age group constituted an increasing proportion of all notifications had a higher proportion of infections of serogroup Y. Almost one third of cases resulting in death.

An increase in the proportion of IMD due to serogroup Y has occurred in NSW during the past 10 years. This has been previously reported throughout Australia, although statistical significance was not demonstrated.[8] Research in the United States of America and United Kingdom has also found a higher prevalence of serogroup Y in older people.[9,10] Higher CFRs have previously been reported in older people with IMD (particularly among those with underlying medical conditions) and among serogroup Y cases even after controlling for age.[9–11] This may explain the higher CFR in older persons in this study; they had a higher proportion of infection with serogroup Y.

Potential increases in the number of IMD cases among the elderly, as a result of an ageing population, have implications not only for the clinical management of these cases but also for the public health response. The current *Guidelines for the early clinical and public health management of meningococcal disease in Australia* recommend provision of clearance antibiotics to household contacts or household-like contacts in

Figure 3. **Proportion of invasive meningococcal disease notifications attributable to serogroup Y by year and age group, New South Wales, Australia, 2003 to 2012**



settings such as schools or universities but do not specifically address aged care or residential facilities.[12] A similar lack of specificity is also found in other national and regional guidelines (**Table 2**) with no particular guidelines on meningococcal disease available for the Western Pacific Region.

Outbreaks in residential aged care facilities have been reported, but specific studies are limited and provide contradictory advice.[13] Prophylaxis was administered following a case of meningococcal disease in an aged care facility in the United States of America in 1997; a further case was then reported in a patient who had refused prophylaxis.[14] A carriage study in the United Kingdom conducted after a case of meningococcal disease was diagnosed in a nursing home conversely found that no residents or caregivers had the same sero-subtype of *Neisseria meningitidis* as the index case, and the study concluded that prophylaxis was not necessary in such settings.[15]

The analyses on serogroup and mortality presented in this study should be interpreted with caution given the amount of missing information, particularly between 1993 and 2002 and because the estimates among older people are based on relatively small numbers. It is also likely that mortality was underestimated across all age groups, given the difficulty in determining a single cause of death. However, as IMD is a notifiable and serious disease, affected patients are likely to present in a health -care setting and be tested and notified by a treating physician, thus the notification data should approximate incidence.

CONCLUSION

The epidemiology of IMD in NSW, Australia has changed following the introduction of the childhood vaccination programmes, with a higher proportion of infections in those aged 65 years and over attributable to serogroup Y. Similar analysis should be conducted at the national and regional levels to determine whether similar trends are occurring across Australia and other countries that have introduced childhood meningococcal C vaccination programmes. As the Australian population is ageing, there may be increases in the number of notifications of IMD among people aged 65 years and over, including cases and potentially outbreaks in aged care facilities. Such an increase would have serious implications given the higher mortality rates in this group. National protocols should be updated to provide clinical and public health guidance for this age group.

Conflict of Interest

None declared.

Funding

None.

Table 2. **Recommendations on use of chemoprophylaxis in general and in aged care facilities following a case of invasive meningococcal disease – various guidelines**

Country or Region	Guideline	Recommendations on use of chemoprophylaxis in general and in aged care facilities
Australia	*Guidelines for the early clinical and public health management of meningococcal disease in Australia*. Canberra, Australian Government of Health and Ageing and Communicable Diseases Network Australia, October 2007.[12]	"The household contacts of a case, including recent visitors who have stayed overnight in the 7 days preceding the onset of the case's illness should receive clearance antibiotics and vaccination. … Those who share the same dormitory, military barrack or hostel bunkroom as a case are, in effect, household contacts." p39 No specific mention of aged care facilities.
United Kingdom	*Guidance for public health management of meningococcal disease in the United Kingdom*. London, Health Protection Agency United Kingdom, March 2012 (http://www.hpa.org.uk/webc/hpawebfile/hpaweb_c/1194947389261).	"Chemoprophylaxis should be offered to close contacts of cases… in the following categories: (a) Those who have had prolonged close contact with the case in a household type setting during the seven days before onset of illness. Examples of such contacts would be those living and/or sleeping in the same household (including extended household)… .Prophylaxis not indicated (unless already identified as close contacts) for … residents of nursing/residential homes." p22
United States of America	*Prevention and Control of Meningococcal Disease: Recommendations of the Advisory Committee on Immunization Practices*. Atlanta, United States Centers for Disease Control and Prevention, March 2013 (http://www.cdc.gov/mmwr/preview/mmwrhtml/rr6202a1.htm).	"Antimicrobial chemoprophylaxis of close contacts of a patient with invasive meningococcal disease is important to prevent secondary cases. Close contacts include: (1) household members (2) child-care center contacts, and (3) anyone directly exposed to the patient's oral secretions... … in the 7 days before symptom onset." p23 No specific mention of aged care facilities.
Canada	*Guidelines for the Prevention and Control of Meningococcal Disease*. Ottawa, Public Health Agency of Canada, May 2005 (http://www.phac-aspc.gc.ca/publicat/ccdr-rmtc/05vol31/31s1/).	"Chemoprophylaxis should be provided to close contacts … ." p7. Close contacts defined as including: – household contacts of a case – persons who share sleeping arrangements with a case. p3 No specific mention of nursing homes other than as potential location for organization based outbreaks. p4
New Zealand	*Communicable Disease Control Manual – Neisseria meningiditis invasive disease*. Wellington, New Zealand Ministry of Health, May 2012 (http://www.health.govt.nz/publication/communicable-disease-control-manual-2012).	Contact defined as, "Anyone who has had unprotected contact with upper respiratory tract or respiratory droplets from the case during the 7 days before onset of illness to 24 hours after onset of effective treatment." p3 "Public health follow-up is important for household contacts and contacts that have had similarly close exposure. Examples of such contacts are: • those sleeping at least one night in the same household, dormitory, military barrack, student hostel bunkroom (not residents of nursing or residential homes who sleep in separate rooms) as the case … ." p4
Europe	*Public health management of sporadic cases of invasive meningococcal disease and their contacts*. Stockholm, European Centre for Disease Prevention and Control, October 2010 (http://www.ecdc.europa.eu/en/publications/publications/1010_gui_meningococcal_guidance.pdf).	"Chemoprophylaxis with an antibiotic regime that eradicates carriage is recommended for household contacts of a case of IMD." p12 No definition of "household contacts" or mention of aged care facilities specifically.

Acknowledgements

Praveena Gunaratnam is employed as a trainee on the NSW Public Health Officer Training Programme, funded by the NSW Ministry of Health. She undertook this work based at the Hunter New England Population Health. The Programme is offered in partnership with the University of NSW.

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Surveillance of avian influenza viruses in Papua New Guinean poultry, June 2011 to April 2012

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We investigated the circulation of avian influenza viruses in poultry populations throughout Papua New Guinea to assess the risk to the poultry industry and human health. Oropharyngeal swabs, cloacal swabs and serum were collected from 537 poultry from 14 provinces of Papua New Guinea over an 11–month period (June 2011 through April 2012). Virological and serological investigations were undertaken to determine the prevalence of avian influenza viruses. Neither influenza A viruses nor antibodies were detected in any of the samples. This study demonstrated that avian influenza viruses were not circulating at detectable levels in poultry populations in Papua New Guinea during the sampling period. However, avian influenza remains a significant risk to Papua New Guinea due to the close proximity of countries having previously reported highly pathogenic avian influenza viruses and the low biosecurity precautions associated with the rearing of most poultry populations in the country.

Influenza virus is a major respiratory pathogen that infects an average of 5–15% of the global population each year, with approximately 500 000 human deaths related to influenza annually.[1] Currently all known influenza A viruses are naturally maintained in aquatic birds.[2] Occasionally these influenza viruses of avian lineage cross natural species barriers and infect other susceptible bird species and/or mammals including humans, pigs and horses. The interspecies transmission of highly pathogenic avian influenza (HPAI) virus to poultry populations often results in devastating disease outbreaks.

In 1996, a HPAI strain of H5N1 emerged in South-East Asia and extended throughout several Asian, Middle Eastern, African and European countries. Its re-emergence in 2003 resulted in the death of more than 62 million birds in Thailand alone, almost half of which were backyard poultry.[3] Death caused by infection and preventive measures (such as depopulation) implemented to control the spread of the HPAI H5N1 virus resulted in considerable socioeconomic burdens for many of the affected countries.[4] The recent emergence of a novel H7N9 virus in China (March 2013) has increased fears about the spread of influenza viruses with pandemic potential from poultry populations.[5] The transmission of these viruses over long distances by migrating birds is a concern for countries such as Papua New Guinea that have large poultry populations with few biosecurity precautions.

Poultry production accounts for 45% of the total annual livestock production in Papua New Guinea, and poultry consumption is second only to pigs.[6] The short turn-around time, ease in rearing, market demand and high income from poultry production makes it more profitable than most other livestock rearing in Papua New Guinea. Most poultry farming in the country is conducted in semi-enclosed areas or free-ranged village settings. Relatively few poultry farms are commercialized and therefore do not have high biosecurity settings to reduce potential introduction of influenza viruses into the poultry population. The free-ranged village/backyard chickens are often raised together with other animals within the same pen (e.g. pigs and ducks). The village chickens also have unrestricted access to water and feed sources that may be utilized by wild birds, thus increasing the risk of exotic disease transmission.

In this paper we report a cross-sectional study to determine the presence of circulating avian influenza

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Submitted: 21 October 2013; Published: 19 December 2013
doi: 10.5365/wpsar.2013.4.4.004

Table 1. Summary of the poultry* sampling sites in Papua New Guinea

Sampling site (Town, Province)	Number of sub-sites	Biosecurity classification			Total
		Low	Medium	High	
Daru, Western Province	18	69 (13)	0	43	112 (13)
Goroka, Eastern Highlands Province	5	25	28 (9)	0	53 (9)
Mt Hagen, Western Highlands Province	6	15 (3)	20 (2)	24	59 (5)
Mendi, Southern Highlands Province	2	0	6	0	6
Lae, Morobe Province	4	27 (4)	36 (8)	25	88 (12)
Kavieng, New Ireland Province	7	20	8	0	28
Port Moresby, Central Province	4	8	14 (5)	0	22 (5)
Madang, Madang Province	1	0	22 (9)	0	22 (9)
Rabaul, East New Britain Province	6	10	10 (2)	0	20 (2)
Kimbe, West New Britain Province	8	25	5	2	32
Vanimo, West Sepik Province	1	20 (7)	20	0	40 (7)
Kundiawa, Simbu Province	1	2	2	0	4
Wabag, Enga Province	8	6 (2)	12	0	18 (2)
Alotau, Milne Bay Province	11	15	17 (6)	0	32 (6)
TOTAL	**82**	**242 (29)**	**200 (41)**	**94**	**536 (70)**

* Samples in brackets were from ducks (unknown species) with the remaining from chickens.

viruses and the seroprevalence of neutralizing antibodies to avian influenza viruses in poultry populations across Papua New Guinea.

MATERIALS AND METHODS

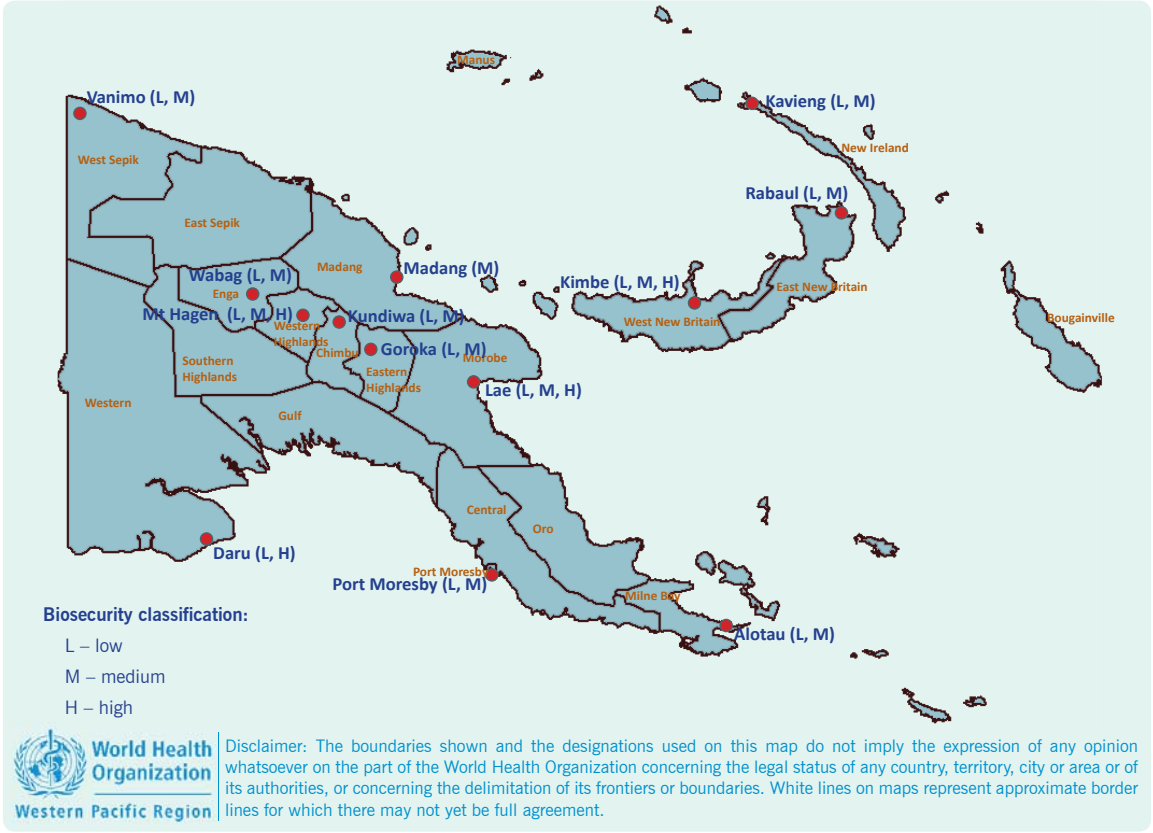
Oropharyngeal swabs, cloacal swabs and serum were obtained from 536 poultry (466 chickens and 70 ducks) from 82 sub-sites within 14 selected provinces from June 2011 to April 2012 (**Table 1** and **Figure 1**). Qualified field officers from the Papua New Guinea National Agriculture Quarantine and Inspection Authority carried out the sampling during their routine surveillance programme, adhering to the guidelines of the Food and Agriculture Organization of the United Nations (FAO) for avian sampling.[7]

Sampling was conducted in three types of biosecurity settings: high, medium and low. These classifications were based on the amount of exposure the sampled poultry population had to other birds and/or animals. Thus, poultry sites with little-to-no exposure to other animals or birds were classified as high (e.g. commercial farms); sites with some exposure were classified as medium (e.g. semi-enclosed farms); and sites with unlimited exposure were classified as low biosecurity containment (e.g. free-range village chickens).

Oropharyngeal swabs, cloacal swabs and serum were obtained from poultry and sent at 4 °C to the laboratory for analysis. Upon arrival at the laboratory, the samples were stored at –80 °C (–20 °C for sera) until required for analysis. Total RNA was extracted from oropharyngeal and cloacal swabs using the QIAamp Viral RNA Minikit (Qiagen, Hilden, Germany), according to the manufacturer's instructions. The extracted RNA was tested for the presence of influenza A virus by real time reverse-transcriptase polymerase chain reaction (PCR) assays supplied by the Centers for Disease Control and Prevention (Atlanta, GA, USA). Samples positive or equivocal for avian influenza viruses were further tested for influenza A/H5 and A/H7 using previously published assays.[8] Aliquots of all samples were sent to the Center of Excellence for Influenza Research and Surveillance, St Jude Children's Research Hospital (Memphis, TN, USA) for isolation and subtyping of avian influenza virus isolates.

A total of 36 paired oropharyngeal and cloacal samples collected from farms and provinces that had samples deemed equivocal were passaged three times in 10-day old embryonated chicken eggs. A sample was considered negative for isolation if no virus was isolated upon three passages. For increased sensitivity in detection of viral genome, deep-sequencing was also

Figure 1. **Map of Papua New Guinea showing the 14 provinces where sampling was conducted**



performed on the equivocal samples. Briefly, viral RNA was extracted, transcribed to cDNA and subjected to whole-genome amplification according to previously published methods.[9] The resulting PCR products were then library-prepped and sequenced on the Illumina MiSeq platform (Illumina, San Diego, CA, USA) using the paired-end sequencing chemistry. After removal of MiSeq indices, analysis was performed using CLC Genomics Workbench 6.5 (CLC bio, Aarhus, Denmark) using the following process: for quality trimming sequence reads were filtered at the quality-limit threshold of 0.05; short reads and reads with more than two ambiguous bases were removed. Remaining reads were then de novo assembled using the fast-contig mapping mode at the minimum contig length of 200 base pairs; paired-reads were aligned using the scaffold option. Assembled contigs were then subjected to BLASTn search against the National Center for Biotechnology Information (Bethesda, MD, USA) database for viral sequences.

Sera were analysed for the presence of influenza A virus antibodies using the IDEXX AI MulitS Screen ELISA (IDEXX Laboratories, Rydalmere, Australia), according to the manufacturer's instructions. All serum samples were individually tested on three separate occasions to ensure the validity of results.

RESULTS

Influenza A virus was not detected in any of the oropharyngeal or cloacal swabs (n=536 each). Four samples had results recorded as equivocal as crossing-threshold values of 36–40 were detected. These samples were tested for influenza A/H5 and A/H7 using real-time PCR; however, all of the samples were negative. Further analysis of these samples using egg inoculation and next-generation sequencing at St Jude Children's Research Hospital (Memphis, TN, USA) resulted in no detection of influenza A virus.

Despite all serum samples being tested on three independent occasions, influenza A antibodies were not detected in any of the samples. Positive and negative control reactions supplied with the kits confirmed the validity of the results.

DISCUSSION

This paper is the first to investigate the presence and distribution of avian influenza viruses in poultry populations in Papua New Guinea. Influenza virus and antibodies were not detected in any of the samples, suggesting that there is low (or no) circulation of avian

influenza viruses in poultry in the country. Poultry and wild bird surveillance programmes in other countries, such as Australia and New Zealand, have also found low prevalence of circulating avian influenza viruses.[10]

The failure to detect avian influenza viruses in poultry does not necessarily mean that Papua New Guinea is at low risk for an outbreak. The recent detection of H5N1 in West Papua (Indonesia)[11] is a concern for Papua New Guinea as this region shares a land border with West Papua.The recent outbreak of Newcastle Disease virus in poultry populations in the north-west region of Papua New Guinea[12] highlights the potential for the incursion of exotic diseases into this region. Indeed the maiden outbreak of chikungunya was first detected in this region[13] before subsequently spreading throughout much of the country.

Papua New Guinea is in close proximity to South-East Asian countries endemic for the H5N1 and H7N9 viruses.[3,5] The spread of these viruses through the migration of waterfowl may be a potential source of incursion into non-endemic areas.[14] Although wild bird surveillance studies have shown that there is a low prevalence of avian influenza viruses in Australia, and an absence of HPAI,[10] avian influenza introduction from this direction is also a possibility given the nomadic migration of some duck species between Australia and Papua New Guinea.[15] H5N1 has not been reported in the Pacific region since its re-emergence in 2003, despite being detected in the West Papua province of Indonesia. Previous studies have suggested that the Pacific region is protected from the incursion of HPAI influenza viruses by the uncommon migration of waterfowl across Wallace's Line.[16] However, it is important that active surveillance continues so that outbreak mitigation steps can be rapidly implemented in the event of incursion of these viruses. In particular, future surveillance studies should focus on wild waterfowl and the potential for the introduction of avian influenza viruses through migration and nomadic movements of these birds.

In this study we report that there is no evidence of avian influenza circulation in Papua New Guinean poultry populations. However, there are some limitations to this study. A cross-sectional analysis for avian influenza viruses may not be sufficiently sensitive when a low prevalence of virus is circulating. The short lifespan of poultry bred for meat and the low number of samples collected from each site may have contributed to the non-detection of avian influenza viruses and antibodies. Therefore, it is recommended that long-term sentinel surveillance should be established at sites where there is a risk of avian influenza introduction, such as sites close to border crossings and lakes used by waterfowl.

Although wild waterfowl migration routes are unlikely to be the source of exotic avian influenza introduction, the landborder with West Papua (Indonesia) and the poultry husbandry practices in Papua New Guinea mean that there is still a relatively high risk of introduction into the country. The introduction of HPAI viruses into Papua New Guinea could create a huge socioeconomic burden. Poultry provides the only source of protein consumption for many people in rural regions, and a large outbreak may have far-reaching health implications. Poor diagnostic capacity at a national level[17] and limited outbreak response and mitigation capabilities may not be sufficient to contain an avian influenza outbreak.

Conflict of interest

None declared.

Funding

None.

Acknowledgements

This work was funded by the National Institute of Allergy and Infectious Diseases, the National Institutes of Health, under contract number HHSN266200700005C. Marinjho Jonduo was supported by a Partnership in Health Research Training Program, funded by Esso Highlands Papua New Guinea Limited.

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Surveillance for arboviral zoonoses in New Zealand birds

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Introduction: Given the significant burden that emerging infectious diseases place on global economies and public health, the monitoring and mitigation of, and early response to, potential infectious diseases are of the highest priority. The objective of this study was to survey for known and other potential arboviral zoonoses in multiple bird species at four locations in New Zealand.

Methods: Common bird species were targeted for blood sampling during two southern hemisphere summers. Sera from each period (n = 185 and n = 693) were screened in an epitope blocking enzyme immunoassay for flavivirus antibody detection. In the first season, testing for antibodies to specific alphaviruses was conducted on samples with sufficient sera (n = 22). In the second season, blood clots (n = 544) were screened for viral presence by polymerase chain reaction (PCR) for alphaviral and flaviviral RNA, and virus isolation (n = 146) was conducted.

Results: Flavivirus antibodies were detected in 13 species, and one Australasian gannet (*Morus serrator*) from one site was positive for antibodies to Ross River virus. PCR tests and virus isolation were all negative.

Discussion: Evidence for flavivirus exposure in seabirds at Kaikoura Peninsula and Cape Kidnappers suggests that viruses isolated from seabirds and associated ticks in New Zealand in the late 1970s are still present. Evidence for flavivirus exposure in passerines at Kaikoura Peninsula, Cape Kidnappers and Mokoia Island is novel. The Ross River virus finding is also new and supports the hypothesis that migratory seabirds are an import pathway for such agents into New Zealand.

Emerging infectious diseases (EIDs; disease-causing agents that rapidly increase in host range, geographic range or prevalence) are a well-recognized threat to public health globally,[1] and the rate of disease emergence has risen since the middle of the 20th century.[2] Risk analysis indicates that emergence is driven by multiple factors including socioeconomic circumstances,[2,3] climate and land-use changes,[4,5] and pathogen pollution (the anthropogenic global movement of pathogens).[6] Given the significant burden that EIDs place on global economies and public health,[1,7] the monitoring and mitigation of, and early response to, potential infectious disease threats are of the highest priority.[4,8] These global concerns are reflected in New Zealand with an increase in active surveillance for potential disease threats being advocated for the benefit of native wildlife, domestic stock and public health.[9–15]

Four potential viral zoonoses associated with wildlife have previously been documented in New Zealand: three flaviviruses (Johnston Atoll virus,[16,17] Saumarez Reef virus and an unnamed Hughes group virus[17]) and one alphavirus (Whataroa virus[18]). The flaviviruses are all tick-borne viruses that have remained largely unstudied since their detection in the late 1970s. Johnston Atoll virus is closely related to the Quaranfil group of viruses, which have been isolated from symptomatic humans,[16] and it has been hypothesized that humans may also be susceptible to infection with Johnston Atoll virus.[16,19] Saumarez Reef virus is believed to have been responsible for febrile illness in meteorological workers on the Saumarez and Frederick reefs in Australia.[20] A closely related Hughes group virus, Soldado virus, has been implicated as a cause of human illness overseas.[21] The Whataroa virus is a mosquito-borne alphavirus that belongs to the Sindbis virus subgroup that has had a known public health impact in several countries.[22] Whataroa virus has been detected only in bird populations and two endemic mosquito species (*Culex pervigilans* and *Culiseta tonnoiri*) to date, around Whataroa township on New Zealand's South Island.[18]

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Submitted: 5 August 2013; Published: 6 November 2013
doi: 10.5365/wpsar.2013.4.3.002

The ecology and host-associations of all four viruses are poorly understood. In this study we conducted wildlife surveillance for these and other potential viral zoonoses at two locations where viruses were previously recorded (Kaikoura Peninsula and Cape Kidnappers; **Figure 1**) and two locations where occurrence was likely (Muriwai Beach for tick-borne viruses and Mokoia Island for mosquito-borne viruses). These locations are also potential import pathways for infectious agents into New Zealand; for example, migratory seabirds and their ticks may be able to transport infections such as West Nile virus into the country.[23] This potential import pathway has been discussed by various researchers globally,[24–27] and the risk to New Zealand needs to be determined.

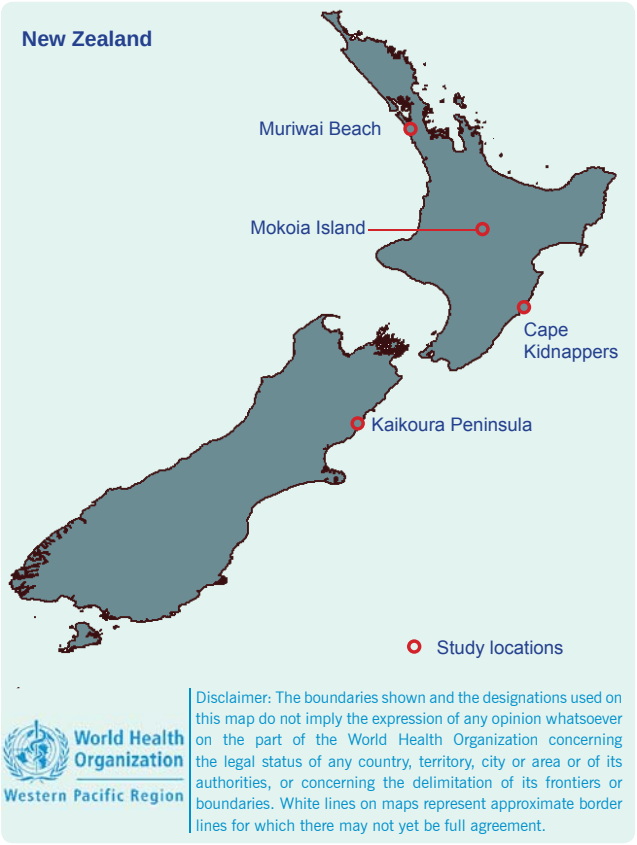
METHODS

Survey sites

The Kaikoura Peninsula, on the north-east coast of New Zealand's South Island, is where Saumarez Reef virus and the unidentified 'Hughes group' arbovirus were isolated from ticks associated with both the red-billed gull (*Larus novaehollandiae*) and white-fronted tern (*Sterna striata*) colonies in the 1970s and where the Hughes group virus was isolated from the blood of a red-billed gull.[17] The presence of these viruses suggests a potential import pathway of migratory seabirds.[23] Red-billed gulls can move over 300 km after breeding, with some evidence of trans-oceanic straggling.[28] Large numbers of white-fronted terns migrate from New Zealand to Australia; the farthest recovery of a banded bird was 2970 km from Kaikoura to South Australia (**Figure 2**).[28]

Cape Kidnappers, a peninsula on the east coast of New Zealand's North Island, has the country's largest mainland colony of the migratory Australasian gannet (*Morus serrator*). In the 1970s, Johnston Atoll virus was isolated from ticks associated with these gannets, in addition to the unidentified Hughes group arbovirus also isolated on the Kaikoura Peninsula.[16,17] Most young Australasian gannets cross the Tasman Sea within three months of life,[28] remain in Australian waters until they are two to three years old (**Figure 2**), then return to their natal gannetries at three years of age as non-breeding or roosting birds – another potential import pathway.

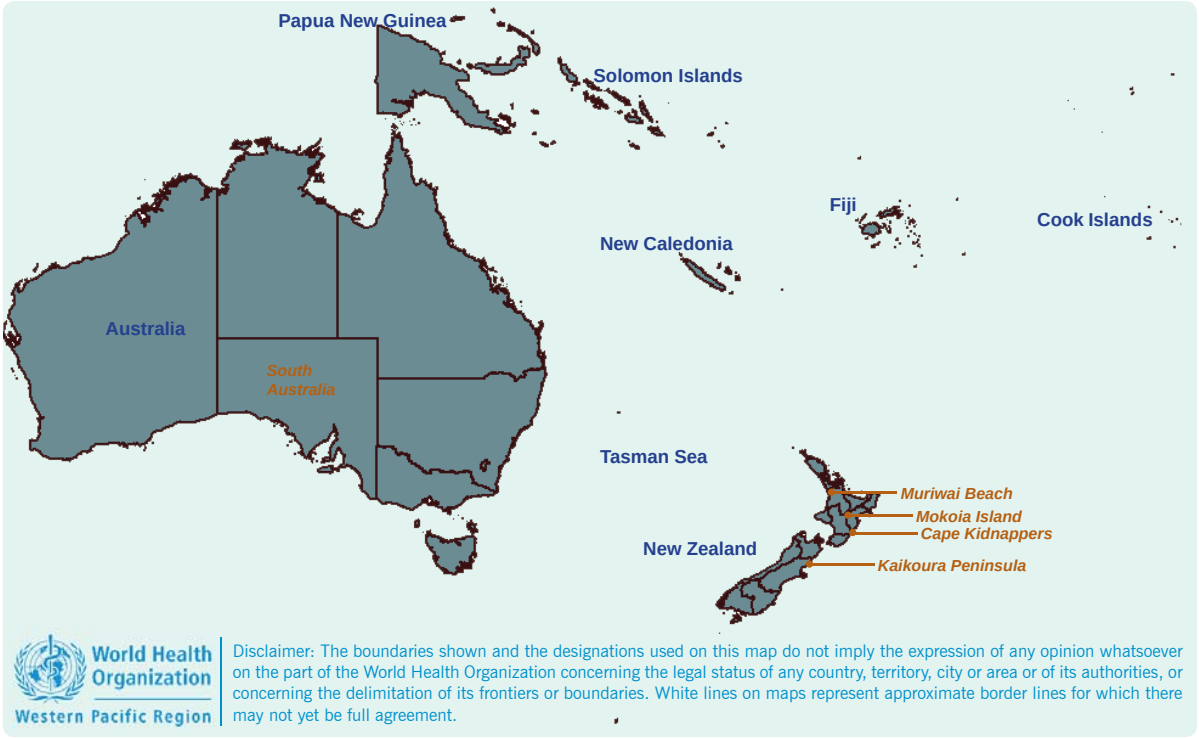
Figure 1. **Map of New Zealand indicating the four study locations**



Muriwai Beach, on the west coast north of Auckland, has three potential import pathways. First, it is a second mainland colony site for migratory Australasian gannets; second, the site is close to major shipping ports and airports in the Auckland Region (both potential sites of entry of exotic vectors); and third, it is a popular tourist destination attracting thousands of overseas visitors each year. Being in the north of the country it also has close proximity to Australia and the Pacific islands (**Figure 2**).

Mokoia Island is a 1.35 km^2 island in the middle of Lake Rotorua in the centre of New Zealand's North Island. Infection of local bird populations by mosquito-borne avian malarial parasites have been documented here,[29] making it a potential site for mosquito-borne viral agents such as Whataroa virus. In addition, the migration of shining cuckoos (*Chrysococcyx lucidus*; a species that breeds on Mokoia Island) to the Bismarck (New Britain Island) and Solomon archipelagos and other Pacific Islands[28,30] offers a potential route of agent incursion

Figure 2. **Oceania regional map**



(**Figure 2**). Mokoia Island is used for endangered bird translocations, representing a pathway for viral spread within the country.

Sampling

The common bird species present at each site were targeted for blood sampling during two southern hemisphere summers – January to March 2008 (all four sites) and November 2008 to February 2009 (Kaikoura Peninsula, Cape Kidnappers and Mokoia Island only). Tuis (*Prosthemadera novaeseelandiae*), North Island robins (*Petroica longipes*), North Island saddlebacks (*Philesturnus rufusater*) and other passerines were caught using mist nets, banded with a numbered metal band (if no band already present) and had a peripheral blood sample collected from the brachial vein. The vein was punctured using a sterile 25–27 g needle (depending on bird size), and blood (no more than 1% body weight) was collected into capillary tubes.

Hand nets were used to catch red-billed gulls and white-fronted terns, and shepherd's crooks were used to catch Australasian gannets. Little blue penguins (*Eudyptula minor*) were taken by hand from burrows as were gulls and terns from nests. Wekas (*Gallirallus australis*) were caught in baited cage-traps, and New Zealand scaup (*Aythya novaeseelandiae*) were caught in mist nets on the shore of Lake Rotorua (in which Mokoia Island lies). Once banded with a numbered metal band (if no band already present), a peripheral blood sample was collected. Gannets, penguins, gulls, terns, scaups and wekas had up to 1.0 ml blood drawn by syringe with a sterile 25 g needle from the metatarsal vein. Gulls and juvenile terns had their brachial vein punctured using a sterile 25 g needle with up to 0.5 ml blood collected into capillary tubes.

Diagnostic testing

Serum samples (collected from $n = 185$ and $n = 693$ individuals during the first and second field seasons respectively) were screened using an flavivirus epitope-blocking enzyme-linked immunosorbent assay described elsewhere[31,32] with the exception that virus-inactivated cell culture lysates were used to coat U-bottom 96-well plates before addition of test samples.[33] Briefly, after washing excess antigen and blocking, sera were added to the 96-well plates in duplicate before the addition of the flavivirus group-reactive monoclonal antibody 3H6 (JCU Tropical Biotechnology Pty Ltd, Townsville, Australia). Binding of the monoclonal antibody was detected following the addition of horseradish peroxidise-conjugated goat anti-mouse antibody and subsequent visualization of enzymatic activity in substrate buffer.

Optical densities were measured and percentage inhibition of the monoclonal antibody by test sera was calculated using negative control sera as the reference. For samples with sufficient sera, those with 30% or greater inhibition were re-tested against 3H6 as well as specific monoclonal antibodies 10C6 (JCU Tropical Biotechnology Pty. Ltd) for Murray Valley encephalitis virus and 3.1112G (Discipline of Microbiology and Immunology, The University of Western Australia, Perth, Australia) for Kunjin virus (both flaviviral agents of incursion concern from Australia[14]). Samples with 50% or greater inhibition on at least one 3H6 test were considered positive for flavivirus antibodies. This criterion was validated as robust in the 50 samples that were re-tested; while some samples up to 40% did not confirm at re-testing, all samples over 40% did.

Testing for antibodies to specific alphaviruses (Ross River virus, Barmah Forest virus and Sindbis virus; arboviral agents of incursion concern from Australia[14,34]) was also carried out on first field season samples with sufficient remaining sera (n = 22) using serum neutralization assays as described elsewhere[35] except that Vero cells were used in place of baby hamster kidney cells. In short, sera were serially diluted in 96-well tissue culture plates and incubated for five days with approximately 50 tissue culture infectious doses of virus and Vero cells. Each well was examined microscopically for cytopathic effect (CPE), and neutralization titres were expressed as the reciprocal of the highest serum dilution where CPE did not occur. Samples with two repeat neutralization titres of at least 40 were considered positive.

Blood clots collected during the second field season (from n = 544 individuals) were screened for viral presence using flavivirus and alphavirus group-specific reverse transcription–PCR tests. Blood clots collected after removal of serum were frozen at –70 °C within four hours of collection. They were then homogenized in sterile virus transport media and the debris pelleted by microcentrifugation. The collected supernatant was extracted directly using the Zymo Viral RNA kit (Zymo Research Corporation, Irvine, CA, USA) and resuspended in ddH2O. For flavivirus testing, we employed the flavivirus nsp5 PCR that uses mFU1 and cFD2 published primers.[36,37] Generic alphavirus PCR was conducted using nsp4 AL-EF and AL-ER primers.[38] Both PCR tests were conducted using Invitrogen Superscript III Platinum Taq Sybr Green one-step qRT–PCR master mix (Life Technologies, Carlsbad, CA, USA) in single-tube reactions. The detection of positive reactions was determined by melt curve analysis of the PCR product followed by gel electrophoresis and DNA sequencing of PCR amplicons.

Virus isolation was performed on clots collected from 146 individuals during the second field season. Supernatants from homogenized clots were inoculated into VeroE6 cell cultures[39] for two passes of five days and monitored for evidence of CPE using a light microscope.

RESULTS

Serology

In the first field season, antibodies to flavivirus were detected in serum samples from a red-billed gull at Kaikoura Peninsula and a North Island saddleback at Mokoia Island (**Table 1**). In the second field season, a relatively high prevalence of antibodies to flavivirus was observed in serum samples from white-fronted terns at Kaikoura Peninsula (**Table 1**). Flavivirus antibodies were also detected at this time in red-billed gulls and passerines at this location; in little blue penguins and passerines at Cape Kidnappers; and in passerines, wekas and New Zealand scaups at Mokoia Island (**Table 1**). None of the 50 repeat-tested samples were specifically positive for either Murray Valley encephalitis virus or Kunjin virus.

Of the 22 first field season samples also tested for antibodies to specific alphaviruses (**Table 2**), one Australasian gannet from Muriwai Beach was positive for antibodies to Ross River virus with two repeat neutralization titres of 80.

PCR and virus isolation

In the second field season, the 544 blood clots (from Kaikoura Peninsula, Cape Kidnappers and Mokoia Island) screened on alphavirus and flavivirus generic PCR tests were all negative (**Table 3**). The 146 clots subject to viral isolation were also negative (**Table 3**); no CPE was observed in any of the cultures after two passages of virus isolation in VeroE6 cells, and no flavivirus PCR products were amplified with RNA extracted from these cell cultures.

Table 1. **Confirmed flavivirus antibody-positive serum samples collected from birds in both the first (2007/08 southern hemisphere summer) and second (2008/09 southern hemisphere summer) field seasons**

Common name	Latin name	Number of individuals positive for flavivirus neutralizing antibodies/Total number screened								Total
		Cape Kidnappers		Muriwai Beach		Kaikoura Peninsula		Mokoia Island		
		2007/08	2008/09	2007/08	2008/09	2007/08	2008/09	2007/08	2008/09	
Australian magpie	Gymnorhina tibicen	–	0/1	–	–	–	–	–	–	0/1
Australasian gannet	Gallirallus australis	0/35	0/131	0/57	–	–	–	–	–	0/223
Chaffinch	Fringilla coelebs	-	0/1	–	–	–	0/7	–	–	0/8
Cirl bunting	Emberiza cirlus	-	-	–	–	–	1/1	–	–	1/1
Common starling	Sturnus vulgaris	0/1	0/1	–	–	–	0/3	–	–	0/5
Dunnock	Prunella modularis	0/1	0/5	–	–	–	0/4	–	–	0/10
Eurasian blackbird	Turdus merula	0/2	0/3	–	–	–	1/14	0/1	2/10	3/30
European goldfinch	Carduelis carduelis	-	0/2	–	–	–	0/4	–	–	0/6
Greenfinch	Carduelis chloris	0/3	0/3	–	–	–	1/5	–	–	1/11
House sparrow	Passer domesticus	0/6	0/34	–	–	–	0/11	–	–	0/51
Little blue penguin	Eudyptula minor	–	2/17	–	–	0/7	0/10	–	–	2/34
New Zealand scaup	Aythya novaeseelandiae	–	–	–	–	–	–	–	1/12	1/12
North Island robin	Petroica longipes	–	–	–	–	–	–	0/15	1/38	1/53
North Island saddleback	Philesturnus rufusater	–	–	–	–	–	–	1/38	0/77	2/115
Red-billed gull	Larus novaehollandiae	–	0/18	–	–	1/15	6/104	–	–	7/137
Silvereye	Zosterops lateralis	0/3	0/11	–	–	–	–	–	–	0/14
Song thrush	Turdus philomelos	–	1/3	–	–	–	0/7	–	0/3	1/13
Tui	Prosthemadera novaeseelandiae	–	–	–	–	–	–	0/1	2/28	2/29
Weka	Gallirallus australis	–	–	–	–	–	–	–	1/8	1/8
Welcome swallow	Hirundo neoxena	–	0/3	–	–	–	–	–	–	0/3
White-fronted tern	Sterna striata	–	–	–	–	–	33/102	–	–	33/102
Yellowhammer	Emberiza citrinella	–	1/4	–	–	–	1/8	–	–	2/12
Site totals		**4/288**		**0/57**		**44/302**		**8/231**		

DISCUSSION

The four sites surveyed for viral agents in birds were selected on the basis of previous documentation of potential zoonoses (in seabirds and their associated ticks) and/or the presence of potential import pathways. Our results indicate that these selection criteria were relevant. Evidence suggests the continued presence of previously isolated seabird flaviviruses, the presence of novel avian flaviviral agents and exposure of a migratory species to an alphavirus of incursion concern from Australia. This last result, serological evidence for antibodies to Ross River virus (the most common mosquito-borne pathogen causing human disease in Australia[34]) in an Australasian gannet at Muriwai Beach, is a novel finding of particular relevance to public health.

Although the standard positive criterion for the flavivirus serology conducted is to achieve inhibition of 3H6 on repeat testing, we were frequently unable to obtain sufficient serum for a repeat (particularly from smaller birds). To maximize the utility of our surveys, and prevent biasing against smaller species in our findings, we instead used a criterion of 50% or greater inhibition on at least one test. Although this criterion was validated as robust in the 50 samples that were re-tested (while some samples up to 40% did not confirm at re-testing, all samples over 40% did), our inability to conduct repeat testing on all samples means that cases of just one or two positive results should be interpreted with caution and require follow-up sampling to confirm the evidence for flavivirus infection. In spite of this proviso, we have obtained two strong lines of evidence for such infection.

Table 2. **Confirmed alphavirus antibody-positive serum samples collected from birds in the first field season (2007/08 southern hemisphere summer) for antibodies to specific alphaviruses***

Common name	Location	Number positive	Number negative
Australasian gannet	Cape Kidnappers	0	4
Australasian gannet	Muriwai Beach	1 (RRV)	12
Little blue penguin	Kaikoura Peninsula	0	1
Red-billed gull	Kaikoura Peninsula	0	3
North Island saddleback	Mokoia Island	0	1

* Specific alphaviruses – Ross River virus (RRV), Barmah Forest virus, Sindbis virus. See **Table 1** for species Latin names.

Table 3. **Blood clots collected in the second field season (2008/09 southern hemisphere summer) subjected to alphaviral and flaviviral PCR assays and virus isolation***

Common name	Number of individuals screened for alphaviruses/flaviviruses			Number of individuals screened by virus isolation	
	Cape Kidnappers	Mokoia Island	Kaikoura Peninsula	Cape Kidnappers	Mokoia Island
Australian magpie	–	–	–	1	–
Australasian gannet	54/54	–	–	22	–
Chaffinch	–	–	11/12	1	–
Cirl bunting	–	–	0/1	-	–
Common starling	–	–	1/3	1	–
Dunnock	3/3	–	8/8	4	–
Eurasian blackbird	–	2/2	17/21	2	1
European goldfinch	–	–	5/6	1	–
Greenfinch	–	–	2/7	1	–
House sparrow	9/9	–	34/35	24	–
Little blue penguin	–	–	11/11	17	–
North Island robin	–	30/46	–	–	12
North Island saddleback	–	54/54	–	–	20
Red-billed gull	1/1	–	119/119	11	–
Silvereye	10/10	–	-	12	–
Song thrush	2/2	1/1	8/10	3	1
Tui	–	6/6	–	–	3
Weka	–	3/3	–	–	3
Welcome swallow	–	–	–	3	–
White-fronted tern	–	–	0/103	–	–
Yellowhammer	2/2	–	13/15	3	–
Site totals	**81/81**	**96/112**	**228/351**	**106**	**40**

* All tests were negative. See **Table 1** for species Latin names.

First, serology results from Kaikoura Peninsula suggest that previously isolated flaviviruses from red-billed gulls (the unidentified Hughes group arbovirus) and ticks associated with both red-billed gulls and white-fronted terns (Saumarez Reef virus and the unidentified Hughes group arbovirus) are still present at this site. Targeted sampling at different times of year may be required for successful viral isolation to verify agent identity. With specific tests for flaviviral agents of incursion concern being negative, the flaviviral reactivity detected in little blue penguins at Cape Kidnappers similarly suggests that the viruses previously isolated from ticks associated with Australasian gannets at this site (Johnston Atoll virus and the unidentified Hughes group arbovirus) may also still be present. However, successful viral isolation is again necessary to verify this.

Second, serological evidence for flavivirus exposure in passerines is novel with no prior evidence for such agents being present in such hosts. Targeted sampling at different times of year may once again be required for successful viral isolation to identify the agents present and inform whether this represents a past incursion via a migratory species such as the shining cuckoo. Since human flaviviral infection is as yet unknown in New Zealand,[9] these agents are most likely not a risk to public health.

CONCLUSIONS

The key conclusion that can be drawn from both the results discussed above and previous work is that migratory birds represent a possible import pathway for potential zoonotic agents into New Zealand. Both the past and current evidence for Saumarez Reef virus and Johnston Atoll virus support the hypothesis that this pathway has historically operated to bring such agents into the country. Although birds may not be currently carrying viral particles back into New Zealand, the evidence for Australasian gannet exposure to Ross River virus indicates that incursion from Australia by such a mechanism may be possible. Since the native *Aedes notoscriptus* and *Culex pervigilans* and the introduced *Aedes camptorhynchus*, *Aedes australis* and *Culex quinquefasciatus* mosquitoes are all potentially competent vectors of Ross River virus,[14,40] such incursion could lead to ongoing transmission within the country. With this agent being of public health concern, more thorough surveillance should be carried out at Muriwai Beach to confirm its current status.

Conflicts of interest

None declared.

Funding

This work was funded by the New Zealand Foundation for Research Science and Technology (now Ministry of Business, Innovation and Employment).

Acknowledgements

Thanks to Lindsay Rowe and Jim Mills for field assistance at Kaikoura Peninsula, to Raewyn Edmonds and Te Rūnanga o Kaikoura for iwi approval to work at Kaikoura Peninsula, to Mick Unahi and Ngāti Hawea for iwi approval to work at Cape Kidnappers, to Malcolm Paterson and Ngāti Whatua o Kaipara for iwi approval to work at Muriwai Regional Park, and to Bill Kingi and the Mokoia Island Trust for iwi approval to work on Mokoia Island. Thanks also to Dean Clarke, Morgan Coleman, Keven Drew, Steph Hicks, Pete Lei, Adrian Monks, Maria Barclay, Lauren Best, Kirsten Derry, Mel Farrant, John Potter, Stephanie Shaw, Ellen Schoener, Cleland Wallace, Stefanie Ismar and Katja Geschke for field assistance. Further thanks to Della Orr for help with virology test development, Megan Dymond and Jianning Wang for contributions to PCR test development and Cheryl Johansen for serological testing. This work was conducted under New Zealand Department of Conservation (DOC) Global Concession CA-5160-OTH; DOC Research and Collection Permits NM-22225-RES, ECHB-22299-FAU, AK-22099-FAU, BP-22190-RES, NM-23980-RES, ECHB-24005-FAU and BP-23988-RES; Landcare Research Animal Ethics Authority 07/12/01; New Zealand National Bird Banding Scheme Institutional Permit to Band Birds No. 2007/83.

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Human infections with avian influenza A(H7N9): preliminary assessments of the age and sex distribution

Viroj Wiwanitkit[a]

The recent report on human infections with avian influenza A(H7N9) is very interesting.[1] The age distribution of the patients was studied, and Arima et al. mentioned that "it seems unlikely that elderly men are being overly selected."[1] It is true that the virus can attack any age group. The factors that determine vulnerability to infection in each age group include (1) immunity to infection, (2) exposure to the disease, (3) availability of medical care, and (4) ability of medical personnel to diagnose the illness.

The elderly are more prone to infections due to their weaker health status as compared to the young. In addition, the high number of infections among the elderly might imply that they have no previous immunity to the infection, indicating that avian influenza A(H7N9) is a new infection for the Chinese in our generation (compared to swine flu in which there was evidence of cross-protective immunity among the elderly that might relate to the low number of cases among that group[2]).

Conflicts of interest

None declared.

Funding

None.

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Submitted: 13 September 2013; Published: 31 October 2013
doi: 10.5365/wpsar.2013.4.3.006

Author Response

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We thank Dr Viroj Wiwanitkit for his comments on our preliminary assessment of the age and sex distribution of the human cases with avian influenza A(H7N9) virus infection. To clarify, we posed three scenarios which could possibly explain the preponderance of cases among elderly men reported through China's surveillance system: (1) differential exposure due to gender-associated practices and norms, e.g. more high-risk behaviours among elderly men; (2) differential clinical course post-exposure/infection, e.g. given similar exposures, elderly men have a more severe outcome relative to other age–gender groups; and (3) differential health care-seeking/access behaviour favouring selection of elderly men, e.g. elderly men accessing health care more than other age-gender groups.[1] There may be more than one of these possibilities in operation, and this initial assessment was intended to pose the question to public health practitioners and researchers and to encourage further study into the causes for the distribution observed for

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Submitted: 14 October 2013; Published: 31 October 2013
doi: 10.5365/wpsar.2013.4.4.003

this apparently emerging disease.[2] Our statement that it appeared unlikely that elderly men were being overly selected was addressing this third possibility.

While we agree with the four possibilities listed by Dr Wiwanitkit "that determine vulnerability to infection", we also believe that vulnerability to severe outcomes (i.e. scenario 2, differential outcomes given an infection) is important when assessing surveillance information given that reported surveillance data are often a function of severity. For example, during the 2009 H1N1 pandemic, while infection rates were lower in the elderly (attributed to likely cross-protection from previous H1N1 infection among the elderly survivors) relative to seasonal influenza, once infected, the elderly were still at higher risk of serious complications.[3] In addition, a recent serological study in China found that, while no seropositivity for antibodies specific for H7N9 virus were detected among >1000 individuals among the general population, greater than 6% of the 396 poultry workers tested were positive, indicating that subclinical or non-severe infections are possible.[4]

We agree with Dr Wiwanitkit's statement that "the virus can attack any age group". As we reported (age range 4–87 years) and as reported later in August 2013, China's routine influenza-like illness surveillance detected from outpatient visits six avian influenza A(H7N9) cases that skewed towards a younger profile.[5] Of these, four had complications and were hospitalized. Notably, the non-hospitalized cases were aged two and four years, while those hospitalized were older. This adds to the biological possibility that, once infected with avian influenza A(H7N9), the elderly may suffer more severe outcomes relative to their younger cohort. While acknowledging the wide age range for infection, the distribution of the avian influenza A(H7N9) cases continues to tend towards the elderly (more than half of cases are 60 years or older as of late September 2013, $n = 135$); this distribution remains strikingly different from that of avian influenza A(H5N1) and requires further investigation. As we noted regarding seasonal influenza infections, the elderly are generally more prone to suffer from severe clinical manifestation of influenza virus infection,[3,6,7] and this may be the case for avian influenza A(H7N9).

Lastly, we appreciate Dr Wiwanitkit's comment regarding the possibility of the absence of immunity to the avian influenza A(H7N9) virus among the elderly, hence the true novel nature of the avian influenza A(H7N9) virus in humans causing infection in all ages rather than a detection and/or reporting artefact. We agree with this statement that is supported through extensive phylogenetic and virological analyses,[8] the absence of pre-existing immunity to avian influenza A(H7N9) among high-risk groups before 2013[9] and the lack of cross-reactive immunity in tested patients previously vaccinated against seasonal influenza viruses.[10]

As the winter influenza season in the northern hemisphere approaches with the potential for additional cases of avian influenza A(H7N9), it is imperative that investigations continue with regards to the observed skewed age and sex distributions.

Conflicts of interest

None declared.

Funding

None.

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Western Pacific Surveillance and Response Instructions to Authors

ABOUT WPSAR

The aims of WPSAR are:

1. to provide an open access journal to publish articles on the surveillance of and response to public health events and emergencies in the WHO Western Pacific Region and in areas with relevance to the Western Pacific Region; and
2. to build capacity in communicating epidemiological and operational research within the WHO Western Pacific Region.

Our objectives are:

1. to provide a platform for people working in surveillance and response in the Western Pacific Region to share their scientific and operational findings;
2. to publish a broad range of articles not limited to conventional research articles:
   - to disseminate short reports on outbreak investigations;
   - to publish analyses of surveillance data on communicable diseases;
   - to encourage the publication of evaluations of new and existing surveillance systems;
   - to promote the use of risk assessment for public health by facilitating risk assessment articles;
   - to support preparedness and response to public health events and emergencies through the dissemination of lessons learnt from such events; and
3. to build capacity in communicating epidemiological and operational findings in the Western Pacific Region through pre-submission assistance.

Scope

WPSAR covers all activities related to the surveillance of and response to public health events and emergencies, with a focus on topics that are relevant to the Western Pacific Region. Public health events may be acute or ongoing and can fall under any of the following areas: communicable diseases, natural disasters, food safety, bioterrorism, and chemical and radiological events. Other events and topics may also be considered. Response activities include those for acute events, e.g. responding to natural disasters, or for response to cases or epidemics of disease.

Why publish in WPSAR?

WPSAR is not limited to conventional research. It publishes a broad range of articles, including short outbreak investigation reports, lessons from the field, analyses of surveillance data, evaluations of surveillance systems and risk assessments for public health events. There are limited opportunities to publish these types of articles in other journals. We also accept the more traditional original research, perspectives and case reports/case series articles.

WPSAR is an open access journal, meaning it is free of charge for both readers and authors. It is also a continuous publication, which means articles are published as soon as they have completed the review and editing process.

WPSAR accepts all articles that fit the scope of the journal and that meet the minimum publication standards. We are especially interested in field epidemiology and operational research.

WPSAR also aims to build capacity in scientific writing and encourages submissions from authors with little or no experience in publishing in peer-reviewed journals. The Coordinating Editor often works with new authors on their submissions to ensure that articles fit the scope of WPSAR and meet the minimum standards for publication.

INSTRUCTIONS TO AUTHORS FOR ARTICLE WRITING AND SUBMISSION

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Formatting guidelines

Please submit your article in a Microsoft® Office Word file or a compatible file in English. Double-spaced, 12-point Arial font should be used to format your article. Please remove all automatic formatting including automatic numbering and referencing before submitting.

The format of the article will depend on the article type. Please see below for specific instructions per article type.

Outbreak Investigation Report

A short article describing a field or outbreak investigation including how it was detected, investigated and controlled. Rapid risk assessments undertaken during these investigations are also encouraged. These articles may be considered for rapid publication.

- Structured article with an abstract of ≤ 250 words and sections for introduction, methods, results and discussion
- Structured abstract with sections for objective, methods, results and discussion
- Word limit: ≤ 1500 words
- ≤ 15 references
- ≤ 2 figures/graphs/pictures

More comprehensive investigations can be submitted as Original Research.

Surveillance Report

A summary and interpretation of surveillance data over a given period of time. A description of the surveillance system and the limitations of the data collected must be included.

- Unstructured abstract of ≤ 250 words
- Word limit: ≤ 2000 words
- ≤ 15 references
- ≤ 10 figures/graphs/pictures

Surveillance System Implementation/Evaluation

An article describing the implementation of a new surveillance system or an evaluation of an existing surveillance system used to detect public health events.

- Unstructured abstract of ≤ 250 words
- Word limit: ≤ 2000 words
- ≤ 15 references
- ≤ 3 figures/graphs/pictures

Risk Assessments

An article detailing a risk assessment of a public health threat or event.

- Structured article with an abstract ≤ 250 words and sections for introduction (including risk question/s), risk assessment methodology, results, discussion and recommendations
- Structured abstract with objectives, method, results and discussion
- The results should include an assessment and/or characterization of the hazard, exposure and context, as well as the level of risk or risk characterization. The limitations must also be included. Risk management may be included in the discussion.
- Word limit: ≤ 3000 words
- ≤ 30 references
- ≤ 3 figures/graphs/pictures

Original Research

Original research articles may include epidemiological studies including outbreak investigations.

- Structured article with an abstract of ≤ 250 words and sections for introduction, methods, results and discussion
- Structured abstract with objective, methods, results and discussion
- Word limit: ≤ 3000 words
- ≤ 40 references
- ≤ 5 figures/graphs/pictures

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An article describing a problem faced in field epidemiology or during a public health event and the experience in trying to overcome the problem.

- Structured article with an abstract ≤ 250 words and sections for problem, context, action, lesson(s) learnt or outcome and discussion
- Structured abstract with the headings of problem, context, action, lesson(s) learnt and discussion
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- ≤ 15 references
- ≤ 3 figures/graphs/pictures

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An unstructured article discussing an issue regarding the surveillance of and response to public health events. The scope of the discussion must be clearly defined.

- Word limit: ≤ 1000 words
- ≤ 10 references
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An unstructured article describing an unusual case or series of cases of public health significance. Subheadings may be used to increase the readability of the article.

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An article providing an analysis of a topic for the Western Pacific Region, typically authored by WHO staff as part of their routine work on behalf of Member States. Regional Analyses do not undergo peer review.

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A letter commenting on a previously published article OR a letter commenting on the theme of the issue. Letters do not undergo peer review.

- Word limit: ≤ 500 words
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News items and meeting and conference reports do not undergo peer review. Please contact the Coordinating Editor at WPSAR@wpro.who.int if you intend on submitting such an article.

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Refer to the article type for the limit on illustrations (figures/graphs/pictures). Please insert all illustrations at the end of the article with titles. Each illustration must be referred to in the text and must be understood on its own. Use Microsoft® Office Excel for graphs and Microsoft® Office Word for tables and diagrams. Additionally, please provide a Microsoft® Office Excel spreadsheet of the data used to create a graph. Footnotes should be placed under the illustration and should use the following symbols in superscript format: *, †, ‡, §, ‖, **, ††, etc.

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Reference the most recent and relevant publications. Please use the Vancouver referencing style with in-text citations and a bibliography at the end of the text. Sample references can be viewed on the National Institutes of Health website.

Place the bibliography at the end of the article text and not as footnotes. Write journal names in full. Use superscript sequential numbering for citing references in the text. Place the number after any punctuation. For example:

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Every article is initially screened by the Editorial Team to ensure it fits the scope of the journal. All articles, with the exception of regional analyses, letters to the editor, news items and meeting and conference reports, then undergo external peer review by two reviewers. This blind peer review process ensures that the reviewer does not know the identity of the author(s) and the author(s) do not know the identity of the reviewer. Significant effort is made to make this process timely, but since it relies on the availability and cooperation of persons external to the journal, it can take considerable time.

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The publication process comprises rigorous editing for content and style by an external technical editor, followed by layout and proofreading. Authors may be asked to provide further information or clarifications during these stages. An article is not formally accepted for publication until these stages have been completed and approval has been granted by the Editorial Team. The authors will also have an opportunity to approve the final proof prior to publication on the WPSAR website. The article will be batched with others in the next quarterly issue.

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As per the International Committee of Medical Journal Editors (ICMJE), all authors should have contributed significantly to the article through one or more of the following in each category A, B and C:

A

- Study design
- Data collection
- Data analysis
- Data interpretation

B

- Drafting the article
- Critically revising the article

C

- Final approval of the article for submission

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If authors have taken photographs that are relevant to their article, they may be submitted for consideration for publication on the cover of the issue. Submission of a photograph does not guarantee its publication.

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  - a short title,
  - a brief description of the article of ≤ 50 words,
  - ≤ 7 keywords,
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  - data in an MS Excel spreadsheet for any graphs
  - names and e-mail addresses of two suggested reviewers (optional but recommended);
- acknowledgements, conflicts of interest, ethics statement and funding information (attached as a separate file to ensure a blind review);
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Acknowledgement to WPSAR reviewers 2013

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