
Summer outbreak of respiratory disease in an Australian prison due to an influenza A/Fujian/411/2002(H3N2)-like virus

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SUMMARY

An outbreak of influenza A occurred in a prison system in New South Wales, Australia in January 2003 during the southern hemisphere summer. This report documents only the third confirmed outbreak of influenza in a prison environment. The outbreak investigation included case ascertainment, state-wide surveillance, a case-control study and interventions to limit the outbreak such as infection control, quarantine, cohorting of cases, and the use of antiviral medication for prophylaxis. A total of 37 clinical cases were identified. Influenza A virus was detected in 11 of the 22 respiratory tract specimens collected. The virus was typed as an influenza A/Fujian/411/2002 (H3N2)-like virus. This strain subsequently became the predominant virus strain during the northern hemisphere winter and the following 2003 Australian southern hemisphere winter influenza season.

INTRODUCTION

Transmission of influenza and other respiratory diseases is facilitated by crowded and confined environments of which prisons are an example. Yet despite this increased potential for the spread of respiratory infections in prisons, to our knowledge only two previous outbreaks of confirmed influenza in a modern prison have been described [1, 2].

We report an outbreak that occurred in a large remand prison with a highly mobile population, and in contrast to the earlier reports, occurred in the middle of summer.

In January 2003, the Public Health Unit of Justice Health (the public sector health-care provider for prisoners in New South Wales, Australia) was notified of a cluster of 12 cases of acute illness, characterized by pharyngitis, fever, malaise, coryza and microscopic haematuria. The 12 cases were all prisoners from one accommodation unit of Prison A, which is a large metropolitan reception prison within a state network of 29 geographically discrete prisons, with at that time a static population of approximately 7900 prisoners. There are approximately 12 000 individuals who pass through the system per year. Prison stays are often short and there are 18 000 receptions (individuals entering or re-entering prison on remand or sentenced) annually. There are approximately 200 receptions per week and 1400 to 2100 movements of prisoners into and out of the prison each week, mostly between prison and court. Prison A has

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† The Prison Influenza Outbreak Investigation Team is given in the Appendix.

890 beds and the standard accommodation units have 64 beds.

METHODS

Investigations and interventions

The initial notification to the Public Health Unit was for 12 cases of acute pharyngitis. Following the initial notification, a case definition was devised. Suspected cases were defined as any person presenting with any three of the following symptoms of an influenza-like illness: fever, pharyngitis, myalgia, prostration, coryza, cough or headache. We recommended that prisoners with symptoms should not leave their cells and if they shared a cell – their cell-mates be relocated to another cell as soon as possible. Staff were encouraged to stay at home while symptomatic.

We also recommended that documented contacts (prisoners, health-care staff and custodial officers) be assessed, have samples collected for laboratory testing, and offered prophylactic treatment with 75 mg oseltamivir once daily, if within 48 h of exposure.

Surveillance

Active surveillance was commenced and clinic staff were asked to identify and report any suspected cases. State-wide active surveillance was commenced throughout the 29 prisons, and contact was made with the state infectious diseases epidemiologist and public health laboratories to assess the level of respiratory viral activity in the general community.

A site visit to accommodation units and prisoner work areas was conducted by public health staff on the day following the initial notification, by which time, the number of cases had risen to 19. Cases were interviewed to ascertain details of their illness including: date of onset, names of their immediate contacts, the date of their last family visit, where they worked within the prison, names of cell-mates and whether or not their cell-mates had been sick. Clinical samples were also collected at this time.

Case-control study

On the second day after notification, a case-control study was undertaken. A self-administered questionnaire was given to all prisoners from the affected accommodation unit ('cases'), and to all prisoners from an adjacent accommodation unit as 'controls'. The control accommodation unit was chosen because

the units were comparable in terms of demographic mix, stability and profile. The questionnaire sought information on symptomatology, utilization of health services, tobacco use, hand-washing behaviour, food sharing habits, work location, accommodation conditions, contact with other ill persons, and history of influenza vaccination. Results were entered into a statistical database (Epi-Info software, version 6.04b, US Centers for Disease Control and Prevention, Atlanta, GA, USA).

Laboratory testing

A single throat, and two nasal swabs were collected and placed in viral transport medium and transported at 4 °C. Samples were tested using a direct immunofluorescence assay (DFA) where smears of deposits from nose and throat swabs were acetone-fixed and stained with fluorescein-conjugated monoclonal antibodies against influenza A and B haemagglutinin and nucleoprotein, respiratory syncytial virus, parainfluenza viruses and adenovirus (Chemicon International, Temecula, CA, USA). Deposits were inoculated into shell-vial monolayers of Madin–Darby Canine Kidney (MDCK) cells, stained by DFA for influenza viruses after 48–96 h incubation, then typed using monoclonal antibodies to influenza A H3 and H1 (Chemicon International). Material from influenza A DFA-positive shell-vials were passaged in MDCK cell tube cultures for further typing. RNA was extracted from the remaining original clinical sample using the High Pure Viral RNA kit (Roche Diagnostics GmbH, Mannheim, Germany) according to the manufacturer's instructions. A multiplex reverse transcriptase–polymerase chain reaction (RT–PCR) was then carried out using influenza A H3N2, influenza A H1N1 and influenza B specific primers in the haemagglutinin region and cycling conditions as described elsewhere with amplicons visualized by ethidium bromide staining following electrophoresis on 2% agarose [3].

Influenza isolates were further serotyped using haemagglutination inhibition at the WHO Collaborating Centre for Reference and Research on Influenza, Melbourne, Australia. The genetic sequence of a single isolate from the outbreak was determined by sequencing the amplicon (1165 bp) following PCR using specific primers targeting the haemagglutinin 1 (HA1) domain of H3N2 and H1N1 [4]. The sequence was aligned with existing influenza A (H3N2) sequences in the Los Alamos National Laboratory

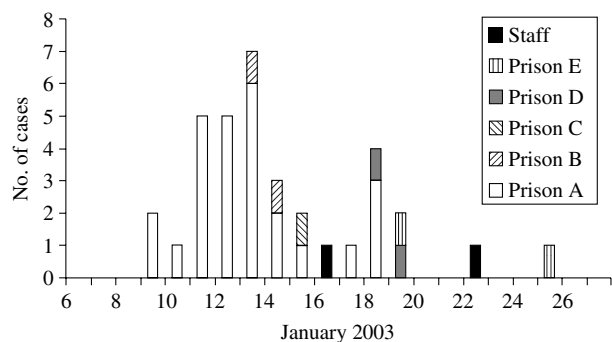


Fig. 1. Summer outbreak of influenza A in an Australian prison – epidemic curve.

Influenza Database [5], using BLASTN [6] and an unrooted neighbour-joining distance tree drawn.

RESULTS

Surveillance

Thirty-seven cases were notified (35 prisoners, one health-care staff and one custodial officer) that satisfied the case definition (see Fig. 1). The first 20 cases were prisoners from the same accommodation unit of Prison A, and were notified during the first 3 days of the outbreak. Another eight cases from six other units within the same prison were identified. A further eight cases were identified in four other prisons within the state. It is highly probable that there were more cases than we ascertained as there was an increase in presentations to the clinic in the few days prior to formal notification, and because of prisoner mobility, ill inmates could have been released to the community prior to case ascertainment.

The epidemic curve of the outbreak had two peaks. The first was day 5, the day of notification. At this stage the outbreak was confined to one prison accommodation unit. The second peak was on days 10 and 11, with five new cases notified. The outbreak had spread to other accommodation units of Prison A and to other prisons (Prisons B, C, D, E) within the state via transfers of prisoners (see Table).

Case-control study results

A self-administered questionnaire was given to prisoners in two adjoining residential wings. Twenty-five cases were identified out of a total number of 108. The age of prisoners ranged between 19 and 58 years. All prisoners were male as were the health-care worker and custodial officer.

Univariate analysis demonstrated close correlation between our case-definition and symptoms reported by cases. A significant association (OR 4.80, 95% CI 1.55–15.15) was found between influenza-like illness and sharing a cell with a symptomatic cell-mate. There was no association between influenza-like illness and history of influenza vaccination, work history, smoking (prevalence of 80%), country of birth, food habits or hand-washing behaviour.

Laboratory results

Twenty-two of the 37 cases had respiratory tract samples collected for DFA and culture, and 20 of these had material remaining for RT-PCR testing. Influenza A virus was detected in 11 (50%). There were nine cases that were influenza A H3N2 RT-PCR positive (five by RT-PCR alone with negative DFA and isolation, three with influenza also detected by both DFA and culture, and one that was DFA positive and culture negative). There were two samples where influenza was detected by both DFA and isolation, but were negative by RT-PCR – in both cases no visible pellet was left after centrifuging for DFA and culture preparation. No other isolates of influenza virus had been made in the previous 2 months in laboratories in the state.

The five isolates were serotyped as influenza A/Fujian/411/2002-like, similar to influenza A strains that were detected late in the northern hemisphere 2002/2003 winter. This was the first reported detection of this strain in the southern hemisphere. Analysis of the alignment of the haemagglutinin nucleotide sequence of one of the five isolates (influenza A/Sydney/015/03; accession number ISDN38234) showed 99% similarity with influenza A/Fujian/411/2002 (ISDN38157), 96.2% to influenza A/Moscow/10/99 (ISDN13277) and 97.1% to influenza A/Panama/2007/99 (ISDNCDA001), an influenza A/Moscow/10/99-like virus that was a component of the 2003 southern hemisphere influenza vaccine. It was 97.1% similar to another influenza A/Moscow/10/99-like virus (influenza A/Sydney/118/2000; ISDN13379), and 96.1% to influenza A/Sydney/5/97 (ISDNASYD97), viruses first isolated in our laboratory. An unrooted neighbour-joining distance tree is shown in Figure 2.

DISCUSSION

This report describes an outbreak of influenza A infection that was unusual as it occurred during the

Table. Characteristics of prisons with reported cases of influenza

Prison	Security	Characteristics	Bed capacity	No. of cases
A	Maximum security, metropolitan	For sentenced and remand prisoners, male	890	28 prisoners, 1 staff member
B	Minimum security, rural	Sentenced prisoners, male	256	2 prisoners
C	Medium and minimum security, rural	Sentenced and remand prisoners, male and female	268	1 prisoner
D	Minimum security, metropolitan	Sentenced prisoners, male	350	2 prisoners, 1 staff member
E	Maximum security, metropolitan	For sentenced and remand prisoners, male	332	2 prisoners

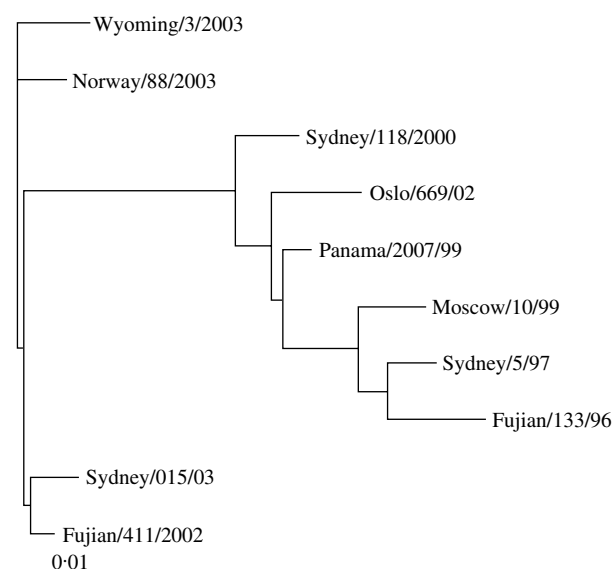


Fig. 2. Phylogenetic relationship of the haemagglutinin gene nucleotide sequence of an influenza A (H3N2) isolate from the prison outbreak, influenza A/Sydney/015/03, to other influenza A (H3N2) sequences. An unrooted neighbour-joining distance tree was constructed using sequences from the Los Alamos National Laboratory Influenza Database [5] Reference influenza A strains and their accession numbers are Wyoming/3/2003 (ISDN38155), Norway/88/2003 (ISDN38160), Sydney/118/2000 (ISDN13379), Oslo/669/2002 (ISDN13294), Panama/2007/99 (ISDNCDA001), Moscow/10/99 (ISDN13277), Sydney/5/97 (ISDNASYD 97), Fujian/133/96 (AF180603), Sydney/015/03 (ISDN 38234) and Fujian/411/2002 (ISDN38157).

middle of summer. To our knowledge it is only the third documented influenza outbreak in a prison, somewhat surprising as prisons are crowded environments, which facilitate transmission of respiratory diseases. One previously described outbreak occurred during the peak winter influenza season in a closed, stable psychiatric ward of a prison hospital where prisoners were housed in single cells [1] and the

infection did not spread beyond that ward. The second prison outbreak also occurred during the winter influenza season [2]. Summer influenza outbreaks [7, 8] have been reported in other crowded environments, such as cruise ships [9–11] a nursing home [12] and an oil-rig in tropical northern Australia [13].

This outbreak began in a crowded 890-bed remand prison, where each cell may accommodate between one and six prisoners. Occupancy rates are consistently near 100%. The prisoner population is highly mobile and the prison has up to 200 new receptions per week. This outbreak also affected a custodial officer and health-care staff. The frequent movement of prisoners between prisons allowed the spread of the outbreak from Prison A to four other prisons in the state, Prisons B, C, D and E. The initial notification was of a cluster of 12 prisoners from one accommodation wing in Prison A, who had presented to the clinic with pharyngitis, fever, running nose, malaise, and on examination, microscopic haematuria. The initial provisional diagnosis was either an adenovirus, group A streptococcus or a toxic reaction to ingestion of contraband alcoholic ‘gaol brew’ that had been detected in the prison a few days earlier. The latter was discounted early in the outbreak investigation, and once laboratory confirmation of an infectious agent – influenza A virus was confirmed.

The State Health Department was contacted following laboratory confirmation of this outbreak, but only 224 doses of the 2002 influenza vaccine were available and, therefore, vaccination was not considered as a useful outbreak response intervention.

We speculate that this outbreak resulted from importation from the northern hemisphere of an influenza A/Fujian/411/2002-like virus, a strain that had not been detected previously in Australia. On

1 January 2003 a group of 11 individuals were transferred from a migrant detention centre to the prison, 9 days before the start of the outbreak. The detention centre at any given time holds individuals who could have been in the northern hemisphere only 8–24 h earlier. The detention centre also held over 30 children at that time, a potential source of influenza virus transmission. Unfortunately, clinical details of influenza-like illness in the detention centre were not available. Influenza A/Fujian/411/2002-like viruses were uncommon in the northern hemisphere winter of 2002/2003. For example, influenza A/Fujian/411/2002-like viruses were only 0.75% of all characterized isolates from Europe (and these were only found in Norway and Switzerland in February and March) during the 2002/2003 winter influenza season [14].

An outbreak of influenza due to an influenza A/Fujian/411/2002-like virus occurred in a police residential college in South Africa in May 2003, 4 months after this outbreak, representing the first incursion of this strain into South Africa. The attack rate of 34% and the hospital admission rate of 7% reflects the appearance of a new influenza variant in an otherwise healthy unvaccinated population [15]. Whether the influenza A/Fujian/411/2002-like viruses cause more severe disease is uncertain, although there were descriptions of increased influenza-associated deaths in children in the United States during the 2003/2004 season [16]. It will be important to monitor the clinical disease due to this strain in the coming influenza seasons.

The influenza A/Fujian/411/2002-like virus was isolated during this prison outbreak before it appeared in Europe. As there is no credible link between the outbreak we describe and subsequent European cases, we are left to surmise that this outbreak and the subsequent outbreaks in Europe had different sources, and that summer outbreaks may serve as a sentinel for the following influenza seasons.

Interestingly, during the following southern hemisphere winter of 2003 and in the northern hemisphere winter of 2003/2004, influenza A/Fujian/411/2002-like viruses have replaced the influenza A/Moscow/10/99-like viruses as the commonest influenza A H3N2 isolates. For example, 87% of influenza A (H3N2) viruses characterized in the United States in the 2003/2004 winter, a season that was more severe than the previous three winter seasons, were Fujian/411/2002-like [17]. The influenza A/Fujian/411/2002-like virus was responsible for a winter outbreak of influenza A in an Australian prison (data not shown),

and a moderately severe winter influenza season in Australia.

Rapid laboratory testing is particularly important in the context of unseasonal outbreaks of acute respiratory illness. As demonstrated in this outbreak, detailed serological or genetic typing of influenza isolates assists in determining whether such outbreaks are due to novel strains, or viruses that may be poorly covered by the current vaccine. Antiviral agents may also be useful in managing influenza outbreaks [15, 18]. Our outbreak control measures were constrained by the lack of readily available vaccine, a potential problem in any summer influenza outbreak.

Recommendations for future management of influenza outbreaks should include early clinical and laboratory diagnosis and notification, provision of antiviral treatment for cases and prophylaxis for cell-mate contacts, and successful restriction of movements to enable quarantine of areas of the prison. As we were dealing with an evolving outbreak, with little published experience on how to manage the situation, at least two possible management strategies could have been pursued. Either, to move well cell-mates out of the cell they shared with a symptomatic cell-mate as soon as possible, or to restrict the movement of exposed and potentially infected cell-mates and leave them to share their cell with a symptomatic case. In this outbreak, on identification of a case with influenza-like symptoms, we recommended that the sick prisoner remain in his cell and that his cell-mate/s, if well, be relocated to another cell as soon as possible. Prisoners who were sick were advised to postpone family visits, and not to attend work while symptomatic. A request was made to the custodial authorities that prisoners not be penalized for not attending work. Requests to the custodial authorities were made relating to isolation of sick prisoners and restrictions of movements into and out of the affected accommodation area, guided by previous experience with management of a varicella outbreak [19]. Custodial staff and health-care staff were advised of infection control measures, focusing on hand washing. Staff members were requested not to present for work if they were ill. A sign was placed in the visitors' reception area advising visitors that there were cases of influenza in the prison.

In conclusion, we have described only the third influenza outbreak in a prison. We were able to document transmission and identify the influenza virus, which became the predominant strain during the subsequent Australian influenza season. Prisons may

be considered as sentinel surveillance sites for acute infections.

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APPENDIX

The Prison Influenza Outbreak Investigation Team included: K. A. McPhie, L. Donovan, V. M. Ratnamohan and S. W. Chan from the Centre for Infectious Diseases and Microbiology Laboratory Services, Institute of Clinical Pathology and Medical Research, Westmead Hospital; and R. Gilmour, G. Forrest, M. Cowley, G. Guirguis and R. Matthews from Justice Health, NSW.

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