Swine flu — lessons learnt in Australia

Peter J Collignon

What did we do well in the first year of pandemic (H1N1) 2009, and what can we do better?

In Mexico in April 2009, a new H1N1 influenza strain appeared to be associated with a high mortality rate. This fuelled fears that a highly virulent virus would quickly spread internationally and cause millions of deaths. Appropriately heightened surveillance and controls were put in place, and Australia activated its "well-rehearsed plan for response to pandemic influenza".[1] Across the country by mid May, we had in place accurate polymerase chain reaction (PCR) testing for "swine flu", improved public awareness of infection control and good public health surveillance. By September, Australia was among the first countries with a vaccine available. Now, a year after the virus first emerged, what have we learnt and how could our pandemic response be improved in the future?

Swine flu did spread rapidly internationally. However, by late May, data from the United States spring showed that case-fatality rates were lower than those from seasonal influenza (< 0.1%).[2] But what would happen in the Australian winter? By mid June, we knew that case-fatality rates here were also low.[3] Despite this knowledge, many costly interventions continued, including border control, widespread use of antivirals, school closures and contact tracing, but with little evidence that these made much difference to the overall rate or spread of the virus. Appropriately, when it became obvious that the spread of the virus could not be controlled, the national pandemic plan was modified. A new phase, "Protect", was adopted on 17 June,[1,4] with a greater focus on treating and caring for those patients who were more vulnerable to severe outcomes.

The word "pandemic" can evoke needless fear and panic. This term would be best used when a virus not only spreads widely but also has increased virulence — this latter aspect is currently not considered in the World Health Organization definition.[5] Virulence needs to be measured quickly and accurately. Pandemic plans seem to assume a case-fatality rate of 1% or more. However, a different approach could be better for a virus such as swine flu with a mortality of 0.01% or less — predetermined responses that take into account different levels of virulence, not just the spread of a virus. The US has such a grading system (similar to that used for hurricane severity),[6] but it was not used to guide this public health response. "Real-time" viral spread and activity can be followed with remarkable accuracy using Google Flu Trends.[7]

In the Australian community, the effects of the pandemic (H1N1) 2009 influenza virus were "*at most* like influenza circulation in a season of moderate seasonal activity".[8] Rates of absenteeism from work and school were similar to those seen in the winter of 2007.[1] The 191 associated deaths were substantially fewer than the 3000 estimated yearly deaths from seasonal influenza in Australia.[1,4,9] Although there may have been additional influenza-associated deaths that were not diagnosed by laboratory testing,

> [a] broader measure of all Australian deaths resulting from influenza or pneumonia currently indicates that there have been fewer such deaths than in other influenza or winter seasons.[1]

Some groups, such as Indigenous peoples and pregnant women, were more vulnerable. Pregnant women had a tenfold higher rate of severe complications than others of the same age.[8] Astute clinicians in Melbourne found that pregnant women with complications were often IgG2-deficient. Thus, we now potentially have a marker that identifies those at much greater risk from influenza and also new, related therapeutic options (using gamma globulin).[10]

Intensive care units (ICUs) in Australia managed to cope with the larger numbers of generally younger influenza patients, but had major problems and were, worryingly, very stretched.[1,4] This demonstrated the lack of spare capacity in our hospitals and ICUs — a problem most apparent every winter. Australia's population mortality rate from swine flu was 0.9 per 100 000.[1,4] If a more virulent virus with a 1% case-fatality rate infected 30% of the population, our hospitals and ICUs could not cope, and we would have to find other ways of managing the problem.

Despite the widespread use of costly oseltamivir stockpiles in Australia and elsewhere, there were no obvious effects in terms of slowing or altering the overall epidemic. Antivirals probably benefit individuals who are at high risk of complications, but in the general population the benefits may be marginal.[11] In addition, the recommendations for who should receive antivirals changed with the different declared phases of the pandemic (eg, from "Contain" to "Protect" phases). This led to confusion for both clinicians and the general public — were antivirals to be used to reduce transmission by ill patients, limit disease severity by stopping sick patients getting sicker, or for prophylaxis?

Testing for swine flu was problematic. Most of those infected had only mild disease, but demand for testing was high. Rapid influenza tests had poor sensitivity, and no specific serological tests were available. PCR was the only reliable form of testing, but it is relatively expensive and labour-intensive. Thus, testing was often not available. Testing was also commonly centralised, which meant results were not readily available in a timely fashion, even for ill patients in many hospitals.

Vaccines were also problematic. Australia was one of the first countries to manufacture and distribute a vaccine for pandemic (H1N1) 2009. However, it only became available after the epidemic finished around the end of September, in multidose vials containing thiomersal, and when a large proportion of the population may have been already immune (from recent infection or prior immunity). In vaccine trials, Australian participants had higher-than-expected levels of pre-vaccination cross-reactive antibodies.[1] Thirty per cent of children aged > 3 years and 27% of adults aged 18–65 years had protective antibody levels, with 62% of adults having detectable antibodies.[12,13] Older people are likely to have even higher pre-existing immunity, given their relatively lower rate of pandemic (H1N1) 2009 infection last winter.

In the future, it could be worthwhile to consider another approach to vaccination. Currently, effective vaccines are usually only available "after the horse has bolted". Because of poor matching, seasonal influenza vaccine efficacy varies from 50% to 80%.[14] New vaccines that are safe and more effective, but that

only have to be given once every 5–10 years and protect against a variety of influenza strains, could be a useful development. Large amounts of public money and resources were spent on antivirals and vaccines in Australia during the pandemic (H1N1) 2009 outbreak. Pandemic vaccines cost over $120 million here, widely reported and mass immunisation delivery costs for 20 million doses would likely be another $500 million.

We also saw that infections spread easily. If people are sick, they should not be at work, school or travelling on public transport. Disproportionate fear generated by media reports resulted in many people presenting to emergency departments or medical practices when they had mild illness and should have stayed at home to recover on their own. However, we do need the ability to quickly assess those in risk groups or those whose condition deteriorates. This may require a phone triage system. Health care workers would then only need to directly assess the much smaller numbers of patients who may need antimicrobials or hospital admission or who are severely ill.

Front-line general practitioners and other clinicians faced extreme difficulties because of deficiencies in implementing parts of the pandemic plan.[15] This involved

> resource supply failures, time-consuming administrative burdens, delays in receiving laboratory test results and approval for provision of oseltamivir to patients, and a lack of clear communication about policy changes as the situation progressed.[15]

We could learn to adapt better as circumstances change and improve consultation with front-line clinicians in any future planning.

The core components of current pandemic planning are influenza vaccination and antivirals. This may not be the best approach. Simple infection control measures such as hand hygiene and barrier methods (gloves, masks, isolation) reduce the spread of respiratory viruses.[16] In the 1918–1919 pandemic, the vast majority of deaths were probably from bacterial complications rather than the influenza virus itself.[17] Effective prevention, treatment and vaccines against bacteria are therefore potentially more effective in preventing deaths.

The swine flu outbreak has provided lessons for all of us in the community — clinicians, health officials, politicians and patients. Despite our efforts to contain this virus with pandemic plans, the pandemic (H1N1) 2009 strain behaved like seasonal influenza and spread rapidly throughout the population, and then stopped just as rapidly. We need to devise better ways to decrease the spread of viruses and to identify and treat the small proportion of people infected with influenza who are likely to develop serious disease or complications. Most importantly, we need to establish better trigger points that take virulence as well as virus spread into account before we roll out pandemic plans.

Competing interests

Canberra Hospital, and my microbiology laboratory at the hospital, received public health funds to do additional pandemic (H1N1) 2009 testing.

Author details

Peter J Collignon, FASM, FRACP, FRCPA, Professor,[1] and Director[2]

1 School of Clinical Medicine, Australian National University, Canberra, ACT.

2 Department of Infectious Diseases and Microbiology, Canberra Hospital, Canberra, ACT.

Correspondence: Peter.Collignon@act.gov.au

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