Editorial

Pandemic influenza: inappropriate fear causes inappropriate responses. In healthcare we need good surveillance data to make the best decisions

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In Australia we are feeling the effects of pandemic (H1N1) 2009 influenza A virus (swine flu). By 5 August 2009, there had been 24 114 laboratory confirmed cases, with 74 associated deaths and 2789 hospitalisations.[1] The disproportionate numbers of pregnant women with serious complications has been a surprising element of this influenza outbreak. In the US, their rate of hospital admissions is approximately four times higher than the general population and about 10 times higher than for other women of their age.[2]

This influenza pandemic will undoubtedly cause many more hospital admissions, serious complications and deaths. However, we need to keep this in perspective. Every year influenza viruses infect hundreds of millions of people and in some years even more. As recently as 2007 in Australia we probably had more hospital admissions than swine flu has caused so far.[2,3] It also does not appear to be any worse in regard to hospitalisations and influenza-like illnesses than have been seen in 2003 and more particularly in 1997 when there was a very large outbreak of influenza.[3]

This current H1N1 strain is said to be 'new'. Thus, fear is generated that we will see a pandemic rivalling what occurred with the 1918–19 Spanish H1 influenza. However while novel, swine flu is not a completely new H strain. It is still H1N1. This means it is in the same H group as the H1 strains that have been circulating in various forms since 1918. Thus, many will already have a reasonable amount of immunity as distinct to if a completely new H strain such as H5 (bird flu) were to readily circulate in people. Older people are infected relatively infrequently. Of people with laboratory-confirmed H1N1 influenza, more than 90% are below the age of 50.[1,3,6] This is fortunate as it is in this older group that most of the complications and deaths from seasonal influenza occur.

In Australia there has already been over 70 deaths associated with this H1N1 virus.[1] However, we need to remember that each year it is estimated that between 2000 to 3000 deaths are attributed to seasonal influenza and 18 000 additional hospital admissions.[4] Thus, it seems so far that we are having a less severe flu than what occurred in recent winters. Comparisons to previous years can be found at excellent websites such as that of the Victorian Infectious Diseases Reference Laboratory (VIDRL).[3] A highly innovative way of looking at flu outbreaks, and which also gives us much more timely information, is available from Google (Flu Trends).[5] This and other sites[3] show we now have low levels of flu activity in most of Australia, with activity having peaked in early July.

In the US after there were 20 000 laboratory confirmed cases, the Centers for Disease Control and Prevention (CDC) estimated that there had already been over 1 million infections there.[6,7] Thus, by the end of July in Australia there were likely also over a million people infected. If we use these US and Australian estimates along with other data, it would appear that for every 20 000 infected people that ~40 were admitted to hospital and approximately four go into Intensive Care Units. There was also approximately one associated death (or 0.005% mortality). Estimates are always problematic but it does appear that this H1N1 virus is less virulent than seasonal influenza. However, it may spread more easily and thus infect more people. Seasonal influenza is estimated to affect between 5 and 15% of the population each year. Already over 5% of Australians have likely been infected. If by the end of this year 20% are infected with swine flu, then this implies that in these 4 million infected people there will be ~200 deaths, 8000 hospital admissions and 800 ICU admissions. This is obviously a serious problem, but nowhere near the catastrophic estimates promulgated by some in the media of 20 000 or more deaths. These estimates are based on inappropriate analogies with the 1918 Spanish flu where 1 to 2% of people infected died. The reason such comparisons are unreasonable is that 95% of deaths associated with 1918–19 influenza pandemic were due to bacterial complications, especially pneumonia.[8,9] The predominant organisms causing disease were Pneumococcus, *Streptococcus pyogenes*, *Staphylococcus aureus* and *Haemophilus influenzae*. In Australia we still have relatively low levels of resistance in respiratory pathogens. We also have antibiotics readily available to treat all likely bacterial complications and so we are very unlikely to see such high death rates. Indeed if one reviews the

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death rates in the US since 1900 attributed to influenza, one can see there were quite high death rates up until the end of World War II. It was only after that time that death rates seemed to markedly decrease.[10] There are undoubtedly multiple factors involved including better housing and nutrition, but the availability of antibiotics since that time has likely made a marked contribution.

Knowing why people die in these epidemics is very important. So far in pandemic plans the main thrust has been quarantine type measures, influenza virus vaccination plans and anti-virals. If most people are dying from bacterial infections, this needs a much better focus, including looking at the use of better pneumococcal vaccines. This also means making sure that appropriate antibiotics are available and will be delivered in a timely fashion for the small percentage who get pneumonia. It is also very important that we do much more to stop antibiotic resistant bacteria developing and spreading. This is a particular problem in the developing world. While the death rates here may only be 0.005%, in developing countries where there is both very high levels of resistance and problems with appropriate antibiotics being available, death rates are likely to be much higher.

This pandemic also has highlighted the importance of infection control for all suspected respiratory infections and how poorly this has been complied with in the past.[11] There is no reason why seasonal influenza viruses should be treated differently to the swine flu virus. Infection control measures used appropriately can decrease transmission of infection, not only in hospitals but also in the general community.[11] During the SARS outbreak in Hong Kong when the general population adopted many infection control practices, including personal distancing, masks and hand hygiene, laboratory-confirmed respiratory virus rates decreased by over 80%.[11] The problem we have in our hospitals is that proper respiratory precautions are often not followed, particularly with seasonal influenza. We need to make sure people who are infected are kept separated from those without infections, including in waiting rooms. We also need to make sure suitable precautions are taken but without going overboard. That means we need to insist on hand hygiene, droplet precautions and the wearing of masks. The main effect of masks, however, may be to stop people touching their own mouth and nose and transmitting infection by their hands rather than via the respiratory route. Doing more to define the main routes of infection is important as it is impossible to have negative pressure rooms for all of the people who may have respiratory illnesses. Also the widespread availability of N95 masks has logistic and financial implications if they become the standard for all respiratory tract infections.

Vaccines are going to be important in controlling this infection. However fear can drive us to make hasty decisions that are not necessarily based on good and robust data. This can include rushing into a mass population vaccination program before we have adequate safety and efficacy data. Also we might try to deliver the vaccine in ways that put people at increased risk for other infections (e.g. by using multi-dose vials). In 1976 in the US, when a swine flu (H1N1) epidemic was thought to be imminent after an outbreak in an army camp, a vaccine was fast-tracked and given to over 40 million people. Unfortunately approximately one in 100 000 developed the Guillain-Barré syndrome (ascending paralysis) and the swine flu virus did not spread to the general community.[12] Obviously if one has a virus that has caused a death rate similar to 1918 (1%), then obviously the most appropriate response to take is to vaccinate even if rare side-effects occur. However, we need good data, surveillance and other information from multiple groups so that we can target our vaccine and other intervention strategies to the groups where we know that the benefits are going to far outweigh any likely or potential side effects. Currently the efficacy of killed influenza vaccines is much lower than many other vaccines (e.g. conjugated pneumococcal). It may be as low as 50% in healthy adults.[13] While this is still better than having no vaccine at all, we need to look at ways of developing better vaccines than give much better efficacy and also ongoing benefit from year to year compared with the types of vaccines we have now. Live vaccines may be one possibility, but this requires a lot more development and research.

Fear, especially early on in this pandemic, meant many people inappropriately went to see their GPs or to emergency departments.[14,15] Most people who get swine flu have a mild infection. They should have stayed at home, got better by themselves and not mixed with other people and risked spreading infection. With influenza we need to reserve access to our medical facilities for those who are in the risk groups, for example, pregnant women, people with moderate to severe asthma, etc. This means they then can be seen promptly, tested and given anti-virals if needed and most importantly, promptly treated if they develop bacterial pneumonia. If we have our system inundated with people because of fear or inappropriate public health concerns, then the very people who need to be seen will be at the end of the queue and may not be given appropriate therapy in time.

We also need to question when we trigger pandemic plans. We have pandemics every few years when new variations of seasonal viruses develop somewhere and then circulate around the world in the flowing winters. Pandemic just means the virus is widespread and goes from continent to continent. It leaves an impression of high virulence but that is often not the case. The US has the more appropriate sub-classification that looks at virulence (Category 1 to 5 depending on the associated death rate).[6] While it was appropriate for us to launch our pandemic plan in Australia when initial data from Mexico suggested an associated mortality of a few percent, it was quickly clear from Canadian and US data that the mortality was much less than 0.1% and thus, less than seasonal influenza.

Appropriately, the Australian, State and Territory Health Departments have now all adopted a new designation that was not part of the original pandemic plan. This is called 'PROTECT'[1] and it is designed to protect those who are most vulnerable to complications from influenza and thus make sure we deliver antiviral therapy, and if necessary, antibiotics as a priority.[14]

In this issue of the Journal several articles show us how we can better approach essential areas of surveillance for infections, including bloodstream and surgical site infections. We need good surveillance in place not only for swine flu but for seasonal influenza and the bacteria that complicate influenza. Examples are bloodstream infection surveillance, rates of resistance in *Staphylococcus aureus* and *pneumococcus*. These are needed so we can learn from outbreaks and make more appropriate actions (e.g. the correct antibiotics used empirically for bacterial complication of influenza). We need decisions to be based on data rather than fear. We also need rapid and reliable ways of detecting infections that are important. This includes the rapid detection of methicillin-resistant *Staphylococcus aureus* as well as viruses such as swine flu.

The work of Graves *et al*.[16] show that improvements in and resourcing of infection control will lead to major savings in the form of increased bed numbers becoming available in our overcrowded hospitals. These articles all highlight the need for surveillance, to not only help stop the spread of viruses with our healthcare facilities but also bacteria, especially *Staphylococcus aureus*. We need good and robust ways of measuring what is happening in the community and in our hospitals not only for influenza but all infections that cause serious complications.

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