

5th Australian Influenza Symposium

Bio21 Institute
Melbourne, Australia

24 – 25 September 2009



Hosted by the WHO Collaborating Centre for Reference and Research on Influenza, VIDRL

with the assistance of the Therapeutic Goods Administration
and the financial support of the Australian Government Department of Health and Ageing

Welcome

The WHO Collaborating Centre for Reference and Research on Influenza and the Therapeutic Goods Administration are delighted to welcome you to the 5th Australian Influenza Symposium.

This Symposium is special for several reasons.

First, it is occurring during the first influenza pandemic since 1968. The Symposium provides a timely opportunity for the Australian influenza community to share experiences in the laboratory, clinical, epidemiological, industry and public health response to the pandemic A(H1N1) virus.

Second, we are staging the Symposium immediately after the *WHO Consultation on the Composition of Influenza Vaccines for the Southern Hemisphere 2010*, which the WHO Collaborating Centre for Reference and Research on Influenza at VIDRL has hosted in Melbourne for the first time. We are fortunate that a number of prominent international scientists and members of the WHO's Global Influenza Programme have stayed on in Melbourne to contribute to the Symposium.

Finally, we are privileged that The Hon. Nicola Roxon MP, Minister for Health and Ageing, will speak in the Symposium. We are grateful for the leadership role that the Australian Government Department of Health and Ageing has played in influenza pandemic preparedness and response and for its continuing support for this Symposium.

The Organising Committee

Dr Ian Barr
Professor Anne Kelso
Katie O'Bryan
WHO Collaborating Centre for Reference and Research on Influenza

Dr Gary Grohmann
Therapeutic Goods Administration

Program

Thursday 24 September

7:30 – 8:30 Registration

8:30 Welcome: Dr. Ian Barr and Dr. Gary Grohmann

Opening comments

8:45 Plenary Session 1: Swine-H1N1pdm influenza

Chair: Prof. Anne Kelso (WHO Collaborating Centre for Reference and Research on Influenza, VIDRL, Victoria)

8:45 Dr. Richard Webby (St. Jude Children's Research Hospital, Memphis, USA)

Swine influenza in pigs

9:10 Dr. Alexander Klimov (WHO Collaborating Centre for Surveillance, Epidemiology and Control of Influenza, Centers for Disease Control and Prevention (CDC), Atlanta, USA)

Emergence and spread of Pandemic H1N1 in North America

9:35 Dr. Wenqing Zhang (Global Influenza Programme, WHO, Geneva, Switzerland)

WHO's role in the H1N1 2009 pandemic

10:00 Morning tea

10:30 Plenary Session 2: Pandemic H1N1: A local Victorian perspective

Chair: Prof. Terry Nolan (Melbourne School of Population Health, The University of Melbourne, Victoria)

10:30 Dr. Rosemary Lester (Department of Health, Victoria)

The Victorian response to Pandemic H1N1

10:55 Dr. James Fielding (Department of Health, Victoria)

An epidemiological perspective of the Victorian outbreak

11:10 A/Prof. Heath Kelly (VIDRL, Victoria)

Epidemiological questions about Pandemic H1N1

11:30 Dr. Mike Catton (VIDRL, Victoria)

Laboratory aspects of Pandemic H1N1

11:45 Dr. Justin Denholm (Royal Melbourne Hospital, Victoria)

Clinical aspects of hospitalised Pandemic H1N1 cases in Victoria

12:00 A/Prof. Paul Johnson (The Austin Hospital, Victoria)

Association between severe pandemic H1N1 2009 influenza infection and immunoglobulin G2 subclass deficiency

12:15 Lunch

12:50 Return to auditorium for the Minister's Address

1:00 Address by The Hon Nicola Roxon MP, Minister for Health and Ageing, introduced by Ms Mary Murnane, Deputy Secretary, Department of Health and Ageing

1:30 Plenary Session 3: National and international aspects of H1N1pdm

Chair: Dr. Lance Jennings (Canterbury Health, Christchurch, New Zealand)

1:30 Prof. Jim Bishop (Chief Medical Officer, Department of Health and Ageing, Canberra, ACT)

Lessons for Australia from the H1N1 pandemic.

1:50 Dr. Sally Roberts (Auckland District Health Board, Auckland, New Zealand)

Pandemic H1N1 in New Zealand

2:10 Dr. Masato Tashiro (WHO Collaborating Centre for Reference and Research on Influenza, National Institute of Infectious Disease, Tokyo, Japan)

Pandemic H1N1 in Japan

2:30 Dr. Rod Daniels (WHO Collaborating Centre for Reference and Research on Influenza, MRC-National Institute for Medical Research, London, UK)

Pandemic H1N1 influenza in Europe

2:50 Dr. Jodie McVernon (Melbourne School of Population Health, The University of Melbourne, Victoria)

The role of modelling in pandemics: Estimating transmission trends and predicting intervention impact

3:10 General discussion on Pandemic H1N1

Panel: Jim Bishop, Heath Kelly, Anne Kelso, Alexander Klimov, Rosemary Lester, Jodie McVernon, Richard Webby

3:30 Afternoon tea

4:00 Workshop 1

(Selected from abstracts)

Chairs: Dr. David Smith (PathWest Laboratory Medicine, Western Australia)

Prof. Robert Booy (Children's Hospital Westmead, The University of Sydney, NSW)

4:00 Dr. Gavin Smith (University of Hong Kong, Hong Kong SAR, China)

Origins and evolutionary genomics of the 2009 swine origin H1N1

4:15 Dr. Craig Dalton (Hunter Health, NSW)

Measuring community influenza like illness during a pandemic

4:30 Mr. David Muscatello (Department of Health, NSW)

Harnessing a continuous health survey program to rapidly assess the non-severe population burden of pandemic (H1N1) 2009 influenza

4:45 Dr. James Watson (CSIRO - Australian Animal Health Laboratory, Geelong, Victoria)

Pandemic H1N1 2009 influenza in pigs: the real swine flu

5:00 Dr. Sheena Adamson (Department of Health, NSW)

Lessons from the NSW laboratory response to H1N1 influenza 2009

5:15 Dr. Fahimeh Rahnema (Auckland Hospital/LabPLUS, Auckland, New Zealand)

The H1N1 testing from the first Rangitoto case

5:30 Dr. Geethani Wickramasinghe (WHO National Influenza Centre, Medical Research Institute, Sri Lanka)

Investigation of Pandemic H1N1 in Sri Lanka

5:45 Dr. Rosemary Tan (Veredus Laboratories, Singapore)

An integrated multiplex RT-PCR and microarray assay

6:00 – 7:30 Symposium Reception at Bio 21 Institute (free for all registrants)

Friday 25 September

8:00 Plenary Session 4: Influenza control measures (1)

Chair: Dr. Gary Grohmann (Therapeutic Goods Administration, Canberra, ACT)

8:00 A/Prof Damon Eisen (Victorian Infectious Diseases Service, Victoria)

Should we vaccinate against swine flu? Lessons from 1976

8:20 Dr. Stephen Gardner (GSK Biologicals, Wavre, Belgium)

Vaccine industry's contribution to global pandemic preparation

8:40 Dr. John Wood (National Institute for Biological Standards and Control, Potters Bar, UK)

Pandemic H1N1 vaccine viruses and reagents – learning from H5N1

9:00 Dr. Gary Grohmann (Therapeutic Goods Administration, Canberra, ACT)

Regulatory issues for Pandemic H1N1 vaccines

9:20 Dr. Michael Greenberg (CSL Limited, Victoria)

CSL Pandemic H1N1 influenza human clinical trials

9:40 Dr. James Smith (F. Hoffmann-La Roche Ltd, Basel, Switzerland)

Tamiflu and H1N1 pandemic influenza: Resistance & other issues

10:00 Morning Tea

10:30 Workshop 2

(Selected from abstracts)

Chairs: Dr. Ian Barr (WHO Collaborating Centre for Influenza, VIDRL, Victoria)

A/Prof. Heath Kelly (VIDRL, Victoria)

10:30 Dr. Stephen Lambert (Qpid Laboratory, Royal Children's Hospital, Brisbane, Queensland)

Influenza surveillance in Australia: we need to do more than count

10:45 Mr. Joseph Descallar (Department of Health, NSW)

Near real time estimation of counts of flu/ptn hospitalisations

11:00 Prof. John Mathews (Melbourne School of Population Health, The University of Melbourne, Victoria)

Prior immunity helps to explain wave like behaviour of 1918-19

11:15 A/Prof. Kevin Downard (University of Sydney, NSW)

Weighing in on the surveillance of influenza with mass spec.

11:30 Dr. Othmar Engelhardt (National Institute for Biological Standards and Control, Potters Bar, UK)

Improving H5N1 candidate vaccine viruses

11:45 Mr. Jeff Butler (CSIRO - Australian Animal Health Laboratory/The University of Melbourne, Victoria)

H5N1 evolution in the mammalian host: a ticking time bomb

12:00 Dr. Lumin Xue (RMIT University and CSL Limited, Victoria)

Immunological studies of cold adapted influenza vaccines in mice

12:15 Ms. Joanne Ernest (WHO Collaborating Centre for Reference and Research on Influenza, VIDRL, Victoria)

Use of siRNA against influenza viruses

12:30 Lunch

1:15 Plenary Session 5: Influenza control measures (2)

Chair: Dr. Jodie McVernon (MCRI & Melbourne School of Population Health, The University of Melbourne, Victoria)

1:15 Dr. Klaus Stohr (Novartis Vaccines, Cambridge, USA)

Novartis swH1N1 vaccine development: new data and outlook

1:30 Dr. Lisa Alleva (Australian National University, Canberra, ACT)

Minimising influenza disease using generic medications, and Complementary and Alternative Medicines

1:50 A/Prof. Stephen Turner (The University of Melbourne, Victoria)

A role for CTL immunity to influenza infection: implications for vaccine design.

2:10 Dr. Deborah Middleton (CSIRO – Australian Animal Health Laboratory, Geelong, Victoria)

The role of ferrets in evaluating pandemic vaccines

2:30 General discussion on influenza vaccines and control measures

Panel: Lisa Alleva, Michael Greenberg, Gary Grohmann, James Smith, Stephen Turner, John Wood

2:45 Plenary Session 6

Chair: Dr. John Wood (National Institute for Biological Standards and Control, Potters Bar, UK)

2:45 Dr. Alan Hay (WHO Collaborating Centre for Influenza, MRC-National Institute for Medical Research, London, UK)

A life in influenza

3:20 Closing comments

3:30 Workshop concludes

Origins and evolutionary genomics of the 2009 swine origin H1N1

Gavin J.D. Smith¹, Dhanasekaran Vijaykrishna¹, Justin Bahl¹, Samantha J. Lycett², Michael Worobey³, Oliver G. Pybus⁴, Siu Kit Ma¹, Chung Lam Cheung¹, Jayna Raghwan², Samir Bhatt⁴, J.S. Malik Peiris¹, Yi Guan¹, Andrew Rambaut²

¹State Key Laboratory of Emerging Infectious Diseases & Department of Microbiology, The University of Hong Kong, Hong Kong SAR, China. ²Institute of Evolutionary Biology, University of Edinburgh, UK. ³Department of Ecology and Evolutionary Biology, University of Arizona, USA. ⁴Department of Zoology, University of Oxford, UK.

In March and early April 2009, a new swine-origin influenza A (H1N1) virus (S-OIV) emerged in Mexico and the United States. During the first few weeks of surveillance, the virus spread worldwide to 30 countries (as of May 11) by human-to-human transmission, causing the World Health Organization to raise its pandemic alert to level 5 of 6. This virus has the potential to develop into the first influenza pandemic of the twenty-first century. Here we use evolutionary analysis to estimate the timescale of the origins and the early development of the S-OIV epidemic. We show that it was derived from several viruses circulating in swine, and that the initial transmission to humans occurred several months before recognition of the outbreak. A phylogenetic estimate of the gaps in genetic surveillance indicates a long period of unsampled ancestry before the S-OIV outbreak, suggesting that the reassortment of swine lineages may have occurred years before human emergence, and that the multiple genetic ancestry of S-OIV is not indicative of an artificial origin. Furthermore, the unsampled history of the epidemic means that the nature and location of the genetically closest swine viruses reveal little about the immediate origin of the epidemic, despite the fact that we included a panel of closely related and previously unpublished swine influenza isolates. Our results highlight the need for systematic surveillance of influenza in swine, and provide evidence that the mixing of new genetic elements in swine can result in the emergence of viruses with pandemic potential in humans.

Measuring community influenza like illness during a pandemic

Craig Dalton, Sandra Carlson, David Durrheim, John Fesja, and Michelle Butler.

Hunter Health, NSW

Background:

Surveillance for influenza is an important public health function as it allows initiation and evaluation of public health measures. Community-based surveillance of influenza-like illness (ILI) is recommended by the World Health Organisation (WHO) as part of a comprehensive surveillance system during inter-pandemic and pandemic periods. Flutracking is a weekly online survey of ILI completed by community members that integrates participants' ILI symptom information with their influenza vaccination status. Flutracking has been trialled in the 2006-2009 winter influenza seasons, and there are currently among more than 6,000 participants Australia-wide.

Methods:

A weekly email to participants provides a link to an online survey with questions on the previous week's experience of cough, fever and time absent from normal duties. The survey also allowed participants to record their past and current influenza immunisation status, as well as age, postcode of residence and exposure to working with patients. The weekly survey took participants less than 15 seconds to complete. Recruitment occurred through organisations' email systems and media releases. In 2009 participants' symptom rates were analysed over time to assess the incidence and severity of H1N1 Influenza 09 at national and state levels.

Results:

In 2009 there were more than 6,000 weekly participants with over 1,000 regular participants in New South Wales, Victoria, Queensland and Tasmania. The peak week of national influenza activity from May to August 2009 was in mid July. There was an increase in fever and cough rates for both vaccinated and unvaccinated participants during this peak period, as expected given that there appeared to be no cross-protection offered by current seasonal influenza vaccine against H1N1 Influenza 09. The peak 2009 fever and cough rates were lower than 2007 and 2008 (with peak national weekly rates of 5.2% in 2009, 5.7% in 2008, and 10.7% in 2007). The peak 2009 national fever and cough rate so far has also occurred earlier in the year than the peak rate for 2007 and 2008.

Conclusion:

Flutracking 2009 data has continued to demonstrate sustainability, with weekly jurisdiction reports now available to four states. Flutracking data suggest relatively low population attack rates of influenza-like illness in 2009, which were not affected by the increased health seeking behaviour and increased laboratory testing associated with the emergence of swine influenza.

Harnessing a continuous health survey program to rapidly assess the non-severe population burden of pandemic (H1N1) 2009 influenza

Muscatello DM, Barr M, Gibney S, Thackway S

NSW Department of Health

Background

Assessing the population burden of influenza is difficult because most surveillance systems derive their data from health care encounters. This excludes the large portion of the population who are ill but who do not seek medical care and restricts the ability to estimate the attack rate due to biased denominators.¹ Opportunistic, unrepresentative samples bias results and hamper interpretation. Population surveys have been previously used to assess the burden of pandemic influenza.² New South Wales, Australia, has been operating a continuous household health survey program since 2002 which produces representative, population-based, monthly estimates of health status, risk factors, and disease prevalence in adults and children.

Methods

Early in the 2009 pandemic, with rapid ethics approval, we introduced questions aimed at continuously assessing the point prevalence of influenza-like illness. The question asked: "In the last 4 weeks, did you have an illness with any of the following symptoms?". The symptoms were "Fever or high temperature?", "Cough?", "Sore throat?", "Runny nose?", "Fatigue?", "Chills or shakes?", "Body aches and pains?", "Shortness of breath?", "The flu' or flu-like symptoms?". If the respondent answered yes to any question, they were asked if they attended a GP or a hospital for their illness. In this analysis, we used a simple case definition to assess symptoms of influenza-like illness: fever and cough and fatigue.³ We report preliminary, unweighted survey results.

Results

For interviews between 19 July and 21 August 2009, results were available for 1648 NSW respondents. Of these, 8.3% reported an influenza-like illness with fever, cough and fatigue. These symptoms were more commonly reported for children 0-16 years (15.1%), than for 17-64 year-olds (8.9%) and people aged 65 years or more (3.1%). Of respondents reporting these symptoms, 51.5% attended a GP only, 3.7% attended hospital only and 2.9% attended both. For interviews in July and August, 9.1% and 7.8%, respectively, reported influenza-like illness.

Conclusion

The survey is providing rapid information on the population burden of influenza-like illness during the pandemic. Susceptibility appears to decline with age, with children most susceptible and the elderly least susceptible. More than half of people with influenza-like illness sought medical attention, and almost 7% sought hospital care.

Pandemic H1N1 2009 influenza in pigs: the real swine flu

James Watson¹, Peter Kirkland², Simone Warner³

¹Australian Animal Health Laboratory, Geelong, Vic; ²Elizabeth Macarthur Agricultural Institute, Menangle NSW; ³Primary Industries Victoria, Attwood, Vic

Several countries have reported spillover of the current human influenza pandemic into pig herds, resulting in localised outbreaks of readily transmitted infection within the herds.

To date, there have been two reported incidents in Australia, in New South Wales and Victoria. Both were associated with infection of farm workers, the putative mode of transmission into the herds.

We report on the characterisation of the virus associated with the outbreaks and the performance of laboratory tests used in diagnosing the disease, as well as the clinical and epidemiological features of the disease in swine.

Lessons from the NSW laboratory response to H1N1 influenza 09

Sheena Adamson, Jan Fizzell, Andrew McNamara, Tim Churches, Paul Armstrong

NSW Department of Health

Background

At the onset of the H1N1 influenza 09 pandemic, national and NSW pandemic influenza plans were in place or in final stages of development. The concept of the laboratory plans was for rapid detection of pandemic virus in early phases by molecular testing of respiratory specimens. After community transmission had been established, testing would be limited to assisting clinical management, detecting new outbreaks and monitoring changes in the virus.

The planned laboratory response in NSW was based on the laboratory Pandemic Influenza Management System (PIMS) together with a web-based data collection system, NetEpi, as the single database for cases and test results to inform the public health response. Respiratory specimens for suspected pandemic cases ideally were to be collected in emergency departments (EDs) and Flu Clinics, and not by general practitioners, in order to facilitate rapid testing.

Capability and capacity for pandemic influenza molecular testing in NSW was to be focussed on two primary public laboratories, with additional capability in an additional six, but no testing in the private sector. Laboratory preparedness had been audited in 2008.

Methods

Procedures undertaken by NSW Health in the laboratory response to H1N1 influenza 09 were reviewed to assist planning for a future infectious disease emergency.

Results

In the DELAY phase of the response, the public health aim of testing was to detect the first cases. In the CONTAIN phase, in addition to testing through EDs and Flu Clinics, detection of community transmission included testing by general practitioners for influenza patients outside the case definition. Once community transmission was established, Australia moved to the PROTECT phase, with testing for influenza (including co-circulating A/H3) targeted at hospitalised patients, and community testing discouraged except for where it changed clinical management.

In the DELAY phase, molecular testing was performed solely in the two primary laboratories. In the CONTAIN and PROTECT phases, other secondary public laboratories serially joined the response. Large-scale rapid antigen testing was also performed in the private sector in the PROTECT phase.

Nearly all of the elements of the PIMS were used in the response. During the DELAY and CONTAIN phases, cases and test results were entered into NetEpi, providing a central source for specimen tracking and results irrespective of the testing laboratory.

Conclusions

The PIMS and NetEpi were effective for tracking specimens and results, and enhanced the public health response. The large and sustained volume of community testing had a major impact on timely delivery of results. This high level of testing continued into the PROTECT phase, despite being discouraged, and impacted on priority testing.

The H1N1 testing from the first Rangitoto case

Fahimeh Rahnama, Deborah Williamson, Sally Roberts and Kitty Croxson

Auckland Hospital/LabPLUS, Auckland, New Zealand

Real-time PCR is now the preferred method for rapid detection of influenza virus infection. Choice of primers and probes must accommodate the instability of the influenza genome, particularly the haemagglutinin gene which is commonly targeted for identification of subtype.

Laboratories must therefore regularly reassess primers and probes for their ability to detect emergent variants. This problem is compounded when a totally new influenza virus appears, as occurred with the novel H1N1 swine virus. The concurrent, immediate need for high throughput diagnostic testing obfuscated the traditional orderly progress of primer selection and validation. Testing algorithms had to take into account the initial mix of seasonal and swine influenza and yet be sufficiently streamlined to cope with the high numbers of samples.

We describe the problems encountered following the dramatic introduction of a new virus and the basis for our initial testing algorithms. We discuss the subsequent evolution of these algorithms as experience was gained with the specificity of different primer sets and as the relative contribution of seasonal and swine influenza in the community changed during the first three months of swine flu circulation.

Investigation of Pandemic H1N1 in Sri Lanka

Dr. Geethani Wickramasinghe

Introduction

As part of the pandemic preparedness plan, a decision was taken to screen all passengers entering Sri Lanka in order to prevent pandemic H1N1 entering into the country. It was also decided to admit all passengers with influenza like illness (ILI), to the Infectious Disease Hospital (IDH) for management. This paper presents the epidemiological, clinical and public health aspects of 71 patients with laboratory confirmed Pandemic Influenza (H1N1) 2009, detected during a two month period, from 16.6.09

Methodology

All passengers entering Sri Lanka were screened and those who were suspected of having ILI were admitted to IDH. Nasopharyngeal aspirates (NPA) were collected and transported to the National Influenza Center (NIC) at Medical Research Institute (MRI). These samples were processed, and the Direct Fluorescent Test (DFT) for Influenza A & B and real time RT-PCR for pandemic H1N1 (CDC protocol) were carried out. Clinical, epidemiological & demographic data were analysed. The first ten NPAs that were positive by the PCR test were sent to the WHO H5 Reference Laboratory, University of Hong Kong for confirmation of test results.

Results

Of the 229 samples tested 71 were positive for Pandemic H1N1 2009 virus infection by the real time RT PCR test. Of these 71 samples only 10 were positive for Influenza A virus, by DFT. Of the 71 positive cases, 62 arrived from other countries: 28 from Australia, 8 from UK, 4 from USA, 9 from Middle Eastern countries, 1 from Canada, 2 each from India, Singapore, Malaysia and Thailand and 4 transit passengers, 2 from Phillipines and 2 from Indonesia. Nine patients had acquired the infection locally and they were either family contacts or very close contacts of positive patients. There was no hospital acquired infection and no community transmission was reported.

Both sexes were equally affected. The predominant age group affected was 11 to 20 yrs followed by 21-31yrs & 31 to 40 yrs. Most patients presented with sign & symptoms of mild to moderate ILI. Diarrhoea & vomiting was seen 14% of them after about 3-4 days of admission. Complications like Pneumonia or RDS were not encountered. All patients were treated with Oseltamivir and there were no deaths. The University of Hong Kong reported that all 10 samples that were sent to them were positive for Pandemic Influenza H1N1 2009; all were sensitive to Oseltamivir and there were no mutation in 274 gene.

Conclusions:

1. Sensitivity of real time RT PCR was much higher than DFT in the diagnosis of pandemic H1N1.
2. Highest number of positives arrived from Australia.
3. Children & young adults were predominantly affected & both sexes were equally affected.
4. Most of the patients had mild to moderate clinical illness and responded well to Oseltamivir.
5. There was no community transmission due to good public health interventions ie. early detection and isolation of patients, early confirmation of the diagnosis, appropriate treatment and management were key to the containment of infection.

An integrated multiplex RT-PCR and microarray assay

Patrizia Di Pietro¹, Floriana San Biagio¹, Monica Capozzoli¹, Kian Leong Ong, Mitsuharu Sato, Rosemary Tan²

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Introduction

The VereFlu™ Lab-on-Chip Influenza Assay is a multiplex RT-PCR/microarray-based device intended for the simultaneous and qualitative detection and identification of commonly known human influenza A subtypes H1N1 (seasonal and H1N1-2009), H3N2, H5N1, H7 (type detection only), H9N2 and Influenza B from viral RNA in nasopharyngeal and/or nasal swab specimens. The oligonucleotide primers and probes have been designed to specifically target unique regions of the HA gene of the selected human influenza subtypes and additional primers and probes targeted on the NA gene for the H1N1-2009 and H5N1 subtype.

VereFlu™ Lab-on-Chip is developed around a silicon chip that integrates both the processes to perform multiplex nucleic acid amplification with an ultra-fast miniaturized PCR reactor and detection of the amplicons with a high grade quality customized microarray. VereFlu™ interfaces with the VereID Biosystem which monitors and control parameters to optimize the reaction. The detection of the amplicons on the microarray is performed by an optical reader and the image is processed and analyzed by software.

Assay analytical performance characteristics

The assay limit of detection (LoD) and Cutoff were determined using serial dilutions of quantified (TCID50/ml) virus culture. The analytical specificity (cross-reactivity) of the VereFlu assay was evaluated by testing a panel of 10 common non-influenza respiratory viruses as well as by cross reactivity amongst influenza viruses. The assay reproducibility was determined using quantified (TCID50/ml) culture at two different viral RNA concentrations (LoD and Cutoff concentration) under different conditions that represent the most common source of variability.

Results

The analytical sensitivity (LoD) ranged from 2×10^2 to 60 TCID50 and from 2×10^2 to 10^2 RNA copies/reaction. The analytical specificity study demonstrates that the VereFlu assay reagents do not cross-react with non-influenza respiratory viruses tested, the overall analytical specificity for the VereFlu assay was 96%.

A total of 76 positive (31 H1N1-2009, 21 H3N2, 13 seasonal H1N1 and 11 FluB) and 8 negative specimens were tested. Clinical sensitivity for H1N1-2009 was 97% (95% CI: 84%-99%). We are performing more tests on clinical samples currently and will present them at the symposium.

Influenza surveillance in Australia: we need to do more than count

Stephen Lambert¹, David Smith², Heath Kelly³

¹Qpid Laboratory, Royal Children's Hospital, Brisbane; ²PathWest Laboratory Medicine WA, QEII Medical Centre, Perth; ³Victorian Infectious Diseases Reference Laboratory, Melbourne

Background

Seasonal surveillance is important in gauging the impact, timing, and spread of influenza, as well as being an important pandemic preparedness activity. Based on national surveillance data, Queensland has been cited as having the most severe influenza seasons in recent years. Given there are no epidemiological reasons why Queensland should disproportionately suffer the effects of influenza season compared to other states and territories, it is likely such findings are related to information bias.

Methods

We compared the month-by-month values from 2004 to 2008 of influenza A and B laboratory tests performed, number positive, and the proportion of tests positive for major laboratories in three Australian states: Queensland, Victoria, and Western Australia. The participating laboratories were Queensland Health laboratory network (QH), Victorian Infectious Diseases Reference Laboratory (VIDRL), and PathWest.

Results

There were year-on-year positive increases in influenza tests performed in each laboratory, with growth between 2004 and 2008 being QH: 216%; VIDRL: 85%; and PathWest: 196%. The rise in testing and positives at all three laboratories was matched by a rise in influenza notifications in each state.

Comparing the month-by-month figures shows a remarkable correlation between the timing and peak value of the proportion of all tests positive for influenza in each of the states. This finding is independent of the variation in number of tests performed. Further, during the severe influenza season in 2007, all laboratories had increased numbers of tests performed, positive, and the proportion positive.

Discussion

The proportion of laboratory tests that are positive for influenza appears to be an important, readily calculated metric for monitoring the temporal and geographical extent, and potentially the severity, of the seasonal influenza epidemic. Unlike the number of tests positive, this value is unbiased by the recent growth in laboratory testing in the three states examined. Consideration should be given to a trial period of following the proportion of laboratory tests positive for influenza, and comparing its timeliness and consistency with other measures used to monitor the epidemiology of influenza in Australia.

Near real-time estimation of counts of influenza or pneumonia hospitalisations from near real-time ED data during pandemic (H1N1) 2009 influenza circulation

Descallar J, Muscatello DJ, Cretikos M, Zheng W

NSW Department of Health

Background:

In NSW, hospital admissions data takes at weeks to months to be available for analysis. NSW Health's near real time emergency department (ED) surveillance system allows rapid monitoring of hospital activity, although not all hospitals are included. For 1996-2007, comparison of time series of ED visits assigned a pneumonia or influenza (P/I) diagnosis and resulting in admission with time series of actual hospital P/I admissions showed similar trends.

Aim:

Using near real time ED admissions data, to rapidly estimate P/I hospitalisations during circulation of pandemic (H1N1) 2009 influenza, and to compare it with admissions forecast using FluSurge software.

Methods:

For a baseline period 1 July 2004 to 30 June 2008, influenza and pneumonia admissions from 43 NSW EDs were compared with hospital admissions for the same period to determine a simple multiplicative factor to estimate statewide admissions. The factor was then applied to weekly ED visits assigned an ED diagnosis of pneumonia or influenza to estimate total NSW hospital admissions by week. These were overlaid on a time series of admissions forecast using FluSurge software that were added to a modelled background seasonal admissions baseline.

Results:

In NSW during the 2004-2008 baseline period, there were 144,359 influenza or pneumonia hospitalisations, and 46,886 equivalent admissions from ED for the hospitals included in ED surveillance, giving a multiplicative factor of 3.08. Estimated admissions peaked at 1650 in the week ending 16 July and subsequently declined to baseline levels by the week ending 13 Aug. With an assumed attack rate of 20% and hospitalisation rate of 1%, FluSurge forecast a much larger and later peak in activity (Figure 1). Nevertheless, the early gradients of the ED and FluSurge curves were remarkably similar.

Conclusion:

The initial pandemic wave led to a lower peak in admissions and an earlier decline than forecast. The method provided rapid and valuable assessment of the impact of the pandemic on hospital admissions.

Prior immunity helps to explain wave like behaviour of 1918-19

John D Mathews⁴, Emma S McBryde⁵, Jodie McVernon¹, Paul K Pallaghy¹, James M McCaw¹

¹Vaccine and Immunisation Research Group, Melbourne School of Population Health, University of Melbourne, 3010, Victoria, Australia.

²Victorian Infectious Disease Service, Royal Melbourne Hospital, 3050, Victoria, Australia.

The ecology of influenza may be more complex than is usually assumed. For example, despite multiple waves in the influenza pandemic of 1918-19, many people were apparently unaffected. Were they unexposed, or protected by pre-existing cross-immunity in the first wave, by acquired immunity in later waves, or were their infections asymptomatic? We model all these possibilities to explain patterns of repeat attacks in 24,706 individuals potentially exposed to summer, autumn and winter waves in 12 English populations in 1918-9. Before the summer wave, only 52% of persons (95% credibility estimates 41-66%) were apparently susceptible. Virus transmissibility was high with credibility estimates for R_0 of 3.10-6.74; prior immunity reduced the estimates for R to 1.57-3.96. However, only 25-66% of exposed and susceptible persons reported symptoms. After each wave, 33-65% of protected persons became susceptible again before the next wave through waning immunity or antigenic drift. Spread of the 2009 H1N1v pandemic may also be limited by immunity from prior exposure to, or vaccination against, seasonal influenza. Such immunity may be short-lived, and not well correlated with levels of HI antibody. It is too soon to say whether any recurrent pandemic waves in 2009-10 will have higher mortality rates, as seen in the second and third waves in 1918-19.

Weighing in on the surveillance of influenza with mass spec

Kevin M. Downard¹, Alexander B. Schwahn¹ and Jason W.H. Wong²

¹School of Molecular & Microbial Biosciences, University of Sydney, Sydney NSW

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The recent declaration of a new influenza pandemic, the first in 40 years, has raised a heightened awareness of the threat the influenza virus poses. Influenza is a leading cause of death in Australia that is responsible for the loss of some 3000 lives each year, usually in the young and aged. Much higher rates of infection exist in the general population that, while not life threatening, inflict illness and suffering that cause significant economic and healthcare burdens. With today's widespread international travel, future pandemics can develop more rapidly providing a relatively short window with which to survey and assess the nature of this antigenically-variable virus and administer effective treatments.

To characterize the virus at the molecular genetic level, the reverse transcriptase polymerase chain reaction (RT-PCR) is employed. Isolation of viral RNA is followed by its reverse transcription to cDNA prior to PCR amplification and sequencing. The design and annealing of suitable primers to targeted gene sequences is critical to the success of the approach and is complicated by the potential for mutations within the primer target sequence. Subtyping of the virus uses sets of primers to conserved sequences of the hemagglutinin and neuraminidase antigens for particular virus subtypes. We have developed a more direct approach with which to type and subtype the virus, and also establish its lineage and similarity to other strains, through the analysis of whole virus digests by high resolution mass spectrometry [1]. Conserved signature peptides have been identified for the hemagglutinin and other antigens across all forms of human influenza that are unique for a particular type, subtype and lineage. The detection of one or more of these peptides by mass spectrometry enables viral strains to be unequivocally assigned based upon their mass alone. The method complements the approach we developed to establish the antigenicity of influenza with mass spectrometry [2,3,4]. Furthermore, we have recently investigated the ability of the signature peptide approach to distinguish between human, animal and recent reassortment strains. This presentation will illustrate the approach with results from the analysis of seasonal and pandemic strains of the influenza virus.

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Improving H5N1 candidate vaccine viruses

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Influenza A virus vaccines are usually made using reassortant viruses that are generated by either classical reassortment or reverse genetics; for pre-pandemic and pandemic viruses, the use of reverse genetics has been extensively used in recent years. However, for avian H5N1 viruses, yields obtained by manufacturers from first generation candidate vaccine viruses were sometimes lower than expected, raising concerns about global production capacity. We were able to demonstrate, for the candidate vaccine virus NIBRG-14, that the low yield was, at least partially, due to a lower than usual content of haemagglutinin (HA) antigen in virus preparations (Harvey et al, Vaccine 26 (2008) 6550-6554). We therefore generated a number of mutant candidate vaccine viruses in attempts to increase the proportion of HA to total viral protein. We now report the generation and characterisation of improved H5N1 candidate vaccine viruses, the antigen content of which is similar to that obtained by seasonal reassortant viruses.

H5N1 evolution in the mammalian host: a ticking time bomb

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We investigated the pathogenic determinants of H5N1 viruses by infecting ferrets with two H5N1 avian influenza viruses that were highly pathogenic in poultry. Ferrets infected with A/Vietnam/1203/2004 showed rapid systemic spread of virus leading inevitably to death. In contrast, A/Chicken/Laos-Xaythiani/26/2006 was highly attenuated in ferrets. Only two of ten ferrets infected with this virus exhibited weight loss and evidence of infection beyond the respiratory tract and neither was euthanased on humane grounds. However, reisolation of virus from the liver and nasal washes of these two ferrets and a single subsequent passage into further ferrets was followed by a dramatic increase in pathogenicity. Enhanced pathogenicity, manifest as a greater rate of disease onset, disease severity and the degree of systemic spread and was associated with the acquisition of Lys at position 627 of the polymerase subunit PB2. Isolates that maintained Glu at PB2 627 remained attenuated. Other researchers have shown that influenza viruses with Glu at PB2 627 cannot efficiently form viral ribonucleoprotein complexes in mammalian cells. The mechanism underlying selection of Lys at PB2 627 in mammalian species is unknown yet expected to be associated with overcoming this defect. Our results imply that acquisition of lysine at PB2 627 may evolve in humans rather than the avian infection source, it may contribute to increased pathogenicity of H5N1 in humans, and screening for this mutation in patients may be a useful predictor of infection severity.

Immunological studies of cold adapted influenza vaccines in mice

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Cold-adapted (*ca*) live attenuated influenza vaccines (LAIVs) have been introduced as alternatives to existing inactivated influenza vaccines. The influenza A components of the FDA-approved *ca* LAIVs (*Flumist*®; Medimmune) have common internal genes derived from the donor strain A/Ann Arbor/6/60 *ca* and surface genes derived from current wild-type (*wt*) epidemic strains. Previous studies had indicated that the immunogenicity of LAIVs are largely related to their surface antigens (HA and NA). However, the relationship between the humoral and cellular mediated immune (CMI) arms of adaptive responses to LAIVs is unclear.

In current study tetramer-based flow cytometry assays and ELISPOT assays were used to measure viral specific-T cell and B cell responses in the respiratory tract, in addition to specific serum antibody responses. Results demonstrated that both humoral and local CMI immune responses were induced by *ca* LAIVs. These responses were variable and were influenced by both viral and host factors. Type I (CTL) responses were induced by low-yielding *ca* reassortants with reduced growth characteristics, such as CR35 (H1N1). Viruses with enhanced growth characteristics, such as CR6 (H3N2), produced higher Type II (HA-specific Ab) responses. In addition host factors, such as the MHC type, were found to play important roles in responses to the same viruses. Susceptible mouse strains, such as C57BL/6 showed higher CTL but lower serum Ab responses than more resistant strains, such as BALB/c. The efficiency of viral replication at the site of inoculation appears to be a key factor in determining the relative contributions of local or systemic and humoral or cellular immune responses to *ca* LAIVs

In summary, a fine balance between the humoral and CMI, local and systemic immune responses induced by *ca* LAIVs was demonstrated. The need to assess local immune responses, in addition to serum antibody levels, for the evaluation of vaccine efficacy was an important conclusion of this study. Further studies of regulation between the humoral and CMI responses to *ca* LAIVs and of cytokines secreted by T_C and T_H cells, such as IL-2, IL-4, IL-6, IL-12 and INF- γ , would allow the selection of better candidate vaccine strains.

Use of siRNA against influenza viruses

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Use of RNAi for targeting of viral disease remains an attractive potential therapeutic. For influenza, prone to antigenic drift and, as seen this year, shift, targeting of internal genes may control viral load, enabling the immune response to clear the infection. Design and testing of siRNAs against laboratory strains of influenza may identify potentially useful target sequences. However the effectiveness of siRNA against seasonal circulating influenza and the recently emerging 2009 'swine-origin' virus is yet to be demonstrated. A panel of both novel and published siRNAs were tested against seasonal influenza A(H1N1) and A(H3N2) viruses. One siRNA was found to be broadly effective, inhibiting growth of the majority of viruses. There was no absolute correlation between sequence conservation, sequence homology and siRNA effectiveness. Thus, use of siRNA to target a highly mutable viral disease, such as influenza, requires testing against a panel of emerging strains to ensure therapeutic benefit.

Novartis swH1N1 vaccine development: new data and outlook

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Despite intensive pandemic vaccine preparations leading to mock-up licensed vaccines in several countries in the past year, large scale clinical trials had to be prepared to establish the immunogenicity of swH1N1 pandemic vaccines. Additional public health requests for data included: immunization schedule to reduce logistical challenges of large scale immunization campaigns, low-dosing to allow for antigen sparing, cross-reactivity to address likely strain variation, anti-body persistence as timing of waves is uncertain and boostability to capitalize on pre-existing cross-reactive immunity from previous seasonal strain infection.

In less than 4 months, several of these questions could be answered through results from clinical trials with cell-culture (CC) and egg-derived swH1N1 pandemic vaccines. E.g. MF59 adjuvanted antigen-sparing CC swH1 vaccine elicits after one dose antibody levels reaching CHMP criteria with two dose providing even better protection. More data is expected in the coming weeks on: comparative immunogenicity in adults, elderly and children, effect of seasonal priming on swH1N1 immunization, immunogenicity of adjuvanted and non-adjuvanted vaccines, effect of different immunization schedules. Considerable impact of the swH1 pandemic in the Southern Hemisphere is likely to trigger widescale immunization programs and mid-term vaccine development focus should be on establishing cross-reactivity of low-dose adjuvanted versus non-adjuvanted vaccines as well as persistence of antibodies and boostability.

Minimising influenza disease using generic medications, and Complementary and Alternative Medicines

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Fibrates and statins are used to lower blood lipid levels in humans, and their use in the population has been widely studied. David Fedson's suggestion that statins could be used to limit influenza disease in a pandemic first captured our attention in 2006, and since then we have concentrated on experiments using statins, fibrates and other agents, to attempt limit influenza disease. The simplicity of this approach lies with accepting that highly pathogenic influenza viruses induce a non-homeostatic host response, including excessive production of cytokines. Hence the use of agents to limit this response should improve disease outcomes. Our group has shown that the fibrate gemfibrozil halved the mortality associated with severe influenza in mice. We have since expanded our research focus to include natural agents and traditional Chinese medicines with similar immunomodulatory properties to fibrates and statins. Some of these CAM inhibit the release and/or biological activity of the pro-inflammatory cytokine high mobility group box 1 (HMGB1). Elevated levels of HMGB1 in serum have been linked with mortality in sepsis and malaria, and HMGB1 was recently shown to be elevated in the bronchoalveolar lavage fluid of mice with influenza. Downstream of excessive pro-inflammatory cytokine production, we are interested in immunosuppression, one of the consequences of excessive cytokine production. It is acknowledged that if the primary, excessive host response to highly pathogenic influenza virus does not directly kill the patient, the ensuing immunosuppression might increase the chance that death will occur, by increasing the patient's susceptibility to secondary infection. We suggest that by treating patients with generic medications such as statins or fibrates, or with CAM such as traditional Chinese medicines, a reduction in levels of circulating pro-inflammatory cytokines should be achievable. Aside from the possibility that these treatments could directly reduce influenza morbidity and mortality, we propose that surviving patients might also demonstrate a more robust immune response to secondary infection, should it eventuate.

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