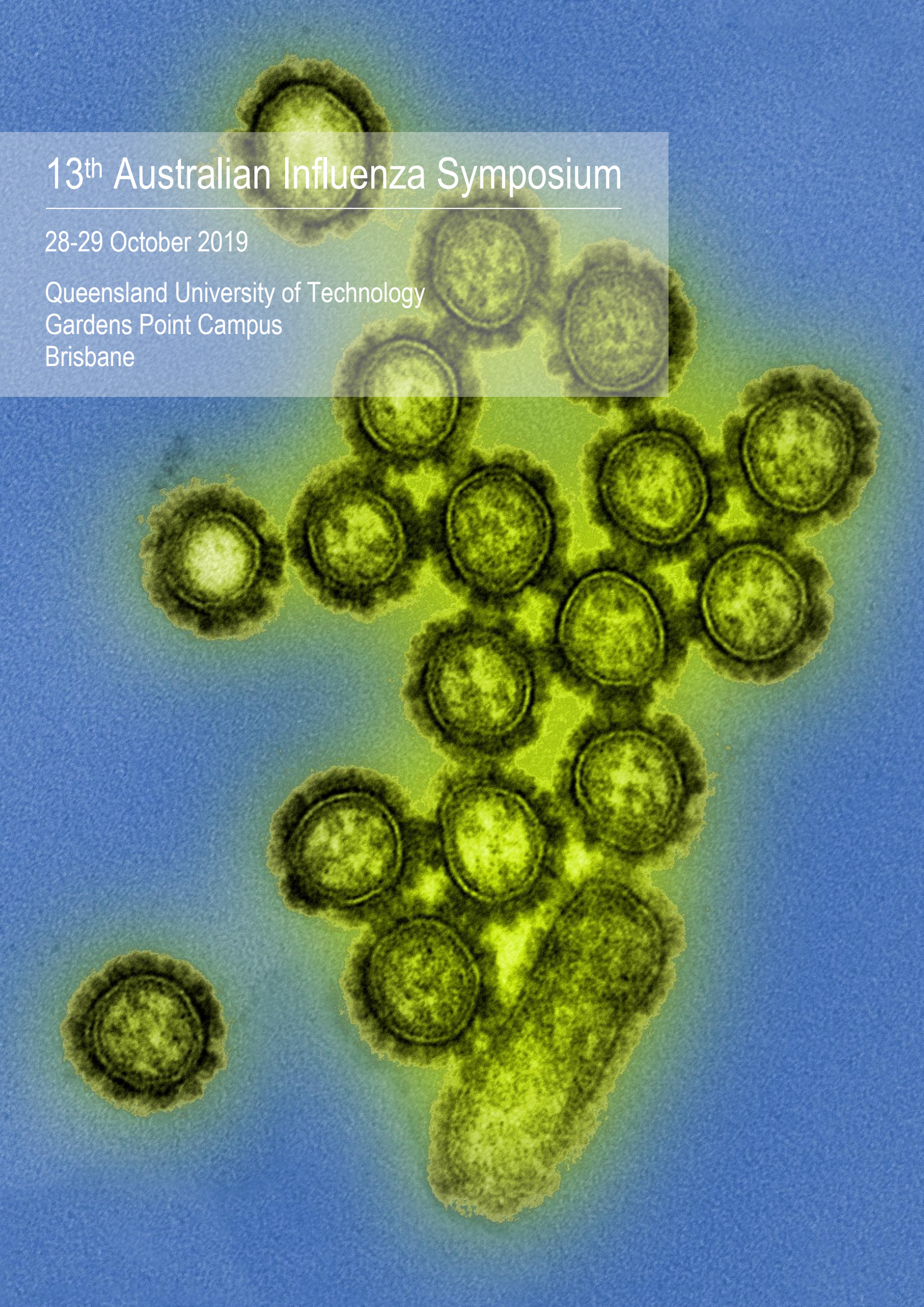
13th Australian Influenza Symposium

28-29 October 2019

Queensland University of Technology
Gardens Point Campus
Brisbane



Welcome

The WHO Collaborating Centre for Reference and Research on Influenza and the Therapeutic Goods Administration are delighted to welcome you to the **13th Australian Influenza Symposium 2019** at Queensland University of Technology, Gardens Point Campus, in Brisbane.

We wish to acknowledge the following:

Symposium Organising Committee:

WHO Collaborating Centre for Reference and Research on Influenza:

- Prof. Ian Barr
- Jayde Simpson

Queensland University of Technology:

- Prof. Kirsten Spann
- Anne-Marie Lacaze

We are especially thankful for the financial support from:

Australian Government
Department of Health

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Cover image source: https://phil.cdc.gov/Details.aspx?pid=18156

Caption: Produced by the National Institute of Allergy and Infectious Diseases (NIAID), this digitally colorized transmission electron microscopic (TEM) image, depicts numbers of H1N1 influenza virus particles. Surface proteins located on the surface of the virus particles are shown in black.

Contents

Social Media

- Social media is permitted at AIS
- WHO CC Melbourne's twitter handle is @WHOFluCCMelb
- AIS event hashtag is #AIS2019Bris
- Please do not include any talks/slides that speakers specifically request NOT be included in social media

Program Day 1

Monday, 28 October 2019		
0800	Registration	
0850	Meeting opening: Ian Barr, WHO CC, Doherty Institute, Melbourne, VIC	
0855	Welcome from local host organiser and meeting logistics: Kirsten Spann, QUT, Brisbane, QLD	
0900	Opening remarks: Jeanette Young, Queensland Chief Health Officer	
Plenary Session 1: Cohort studies | Chair: Kanta Subbarao, WHO CC, Doherty Institute, Melbourne, VIC		
0915	Janet Englund, Fred Hutch Cancer Center, Seattle, WA, USA	Influenza and Other Respiratory Viruses in Childcare Centers
0945	Jonathan Temte, University of Wisconsin, Madison, WI, USA	Use of rapid influenza detection tests for outbreak detection in long-term care facilities
1015	Ben Cowling, The University of Hong Kong, Hong Kong SAR, China	Strategies for the use of enhanced influenza vaccines in older adults
1045	*Morning tea*	
Plenary Session 2: Hot topics | Chair: Philip Britton, The University of Sydney, NSW		
1115	Alan Hampson, Federation University, VIC	Pandemic Influenza in Australia -1919 to 2019
1135	Belinda Herring, WHO Regional Office, Brazzaville, Republic of the Congo, Africa	Influenza surveillance in the WHO African Region
1155	Kirstie Short, University of Queensland, Brisbane, QLD	Chronic medical conditions and severe influenza virus infections
1215	Yi-Mo Deng, WHO CC, Doherty Institute, Melbourne, VIC	Human infection of a locally acquired swine-origin influenza A(H3N2) variant in Australia
1235	Hui-Ling Yen, LKS Faculty of Medicine, The University of Hong Kong, Hong Kong SAR, China	Ferret model of influenza transmission
1255	*Lunch*	
Plenary Session 3: Hot topics – 2019 season | Chair: Chris Blyth, University of Western Australia, Perth, WA		
1345	Ian Barr, WHO CC, Doherty Institute, Melbourne, VIC	Intense interseasonal influenza outbreaks, Australia, 2018/19
1405	Robert Booy, NCIRS, Westmead, NSW	The Reliability of Point of Care Testing in Aged Care Facilities
1425	Aye Moa, University of New South Wales, Sydney, NSW	Flucast - a tool to predict seasonal influenza severity
1445	Rob Moss, University of Melbourne, Melbourne, VIC	Forecasting in an unusual season: what can we learn from 2019?
Roundtable Discussion | Chair: Sheena Sullivan, WHO CC, Doherty Institute, Melbourne, VIC		
1505	**For discussion:** What do we need to do to improve influenza surveillance in Australia and will it make any difference to the outcomes? **Panel Members:** Frances Birrell, Nigel Crawford , Craig Dalton, Janet Englund, Robin Gilmour, Ian Mackay	
1530	*Afternoon tea*	
Workshop 1: Research | Chair: Kirsty Short, UQ, Brisbane, QLD & Kirsten Spann, QUT, Brisbane, QLD		
1600	Patrick Schaeffer, James Cook University, Townsville, QLD	New strategies for influenza nucleoprotein-based diagnostic assays
1615	S. Mark Tompkins, University of Georgia, Athens, GA	Risk assessment of contemporary North American swine influenza viruses in murine models of infection
1630	Louise Rowntree, The University of Melbourne, VIC	Generating Immunity to Severe Influenza Disease in Indigenous Australians
1645	Maryam Shojaei, Westmead Institute for Medical Research, Westmead, NSW	Host directed therapy to regulate neutrophil in an in vitro model of influenza infection
1700	Edin Mifsud, WHO CC, Doherty Institute, Melbourne, VIC	Evaluating the window of susceptibility to secondary bacterial infections post-influenza infection in ferrets
1715	Katina Hulme, The University of Queensland, Brisbane, QLD	Hyperglycaemia induces epithelial-endothelial barrier damage during Influenza A infection
1730	Marcus Tong, The University of Queensland, Brisbane, QLD	Chicken and duck endothelial cells display a markedly different innate immune response to viral challenge
1745	**Day 1 concludes: Drinks and finger food at Owen J Wordsworth Function Rooms, S Block, Room 1215 (12th Floor)**	

Program Day 2

Tuesday, 29 October 2019

Plenary Session 4: Industry – Commercial Updates | Chair: Ian Mackay, Queensland Health, Brisbane, QLD

0830	Philippe Buchy, GSK Vaccines Asia Pacific, Singapore	AS03-adjuvanted pandemic vaccines: clinical updates
0845	Chris Clarke, Seqirus Ltd, Melbourne, VIC	Recent developments in Influenza vaccines manufactured in mammalian cells
0900	Hun Kim, SK Biosciences, Korea	Development of Cell Culture-based Influenza Vaccine, SKYCellflu®
0915	Charles Ross, Vaxxas, Brisbane, QLD	Influenza vaccination by Vaxxas high-density micro-array patch (HD-MAP); results from a phase I clinical trial and real-world applications
0930	Sean Parsons, Ellume Pty Ltd, Brisbane, QLD	Influenza rapid test
0945	Emily Mahon, Roche Diagnostics, Sydney, NSW	Rapid and portable PCR influenza testing - taking point of care to the ED and beyond
1000	Daniela Symons-Troy, Cepheid, Australia	Cepheid C360: Turning Insights into Action
1030	*Morning tea*	

Workshop 2: Epidemiology | Chairs: Marlena Kaczmarek, ACT Health, Canberra, ACT & Jocelynne McRae, NCIRS, Sydney, NSW

1100	Frances Birrell, Queensland Health, Brisbane, QLD	Surveillance of respiratory disease outbreaks in Residential Aged Care Facilities in Queensland: 2012 – 2019
1115	George Milne, University of Western Australia, Perth, WA	Mathematical modelling to inform the national seasonal influenza vaccination policy: findings from our study
1130	Jocelynne McRae, NCIRS, Sydney, NSW	Preventing influenza illness in young infants through maternal vaccination: a multisite, multiyear analysis
1145	Philip Britton, The University of Sydney, NSW	Influenza Associated Myositis: a single-centre, 5-Year retrospective Study
1200	David Muscatello, University of NSW, Sydney, NSW	Rapidly mapping the spread of influenza
1215	Sandra Carlson, Hunter New England Local Health District, Newcastle, NSW	Insights into the burden of influenza in Indigenous Australians, 2019
1230	Robin Gilmour, Health Protection NSW, Sydney, NSW	Drivers of a summer influenza epidemic – New South Wales, 2018-2019
1245	Christopher Blyth, University of Western Australia, Perth, WA	Improving Influenza Vaccination in Children with Comorbidities: A Meta-Analysis
1300	*Lunch*	

Plenary Session 5: | Chair: David Muscatello; UNSW, Sydney, NSW

1345	Benjamin Tang, The University of Sydney, NSW	Predicting risk in severe influenza infection
1415	Jonathan Temte, University of Wisconsin, Madison, WI, USA	Evaluating influenza from a community perspective: insights on School absenteeism, vaccine effectiveness, and molecular epidemiology
1445	Janet Englund, Fred Hutch Cancer Center, Seattle, WA, USA	Influenza in the community: An overview of the Seattle Flu Study
1515	*Afternoon tea*	

Joint RSV session with the Australian Respiratory Virology Meeting | Chair: Kristine Macartney, NCIRS, Westmead, NSW

1545	Welcome	
1550	Nigel Crawford, Murdoch Children's Research Institute, Melbourne, VIC	WHO program phase 1 results and plans for phase 2
1610	Gemma Saravanos, NCIRS, Westmead, NSW	Respiratory syncytial virus-associated hospitalisations in the Australian population
1630	Gary Grohmann, Environmental Pathogens, Canberra, ACT	Influenza – a reality check!
1650	Robert Booy, NCIRS, Westmead, NSW	RSV in the elderly
1710	**Joint session and Australian Influenza Symposium concludes**	

Day 1	Plenary Session 1
0915	**Influenza and Other Respiratory Viruses in Childcare Centers**
Speaker	Janet Englund, Fred Hutch Cancer Center, Seattle, WA, USA

The clinical impact and transmission of respiratory viruses in organized childcare settings has been studied for many years, and high frequencies of illness and viral infection have been documented. The epidemiology of acute respiratory illness and spread of viruses in young children varies depending on the setting and methods used to detect viruses. We have studied respiratory viruses in the childcare setting for over a decade, analyzing the clinical presentation, impact of disease, and molecular patterns of viral transmission. More recent studies performed using molecular sequencing have documented rapid spread of RSV within classrooms, as well as moderate rates of rhinovirus transmission among children in the same classroom. In a 2019 study of RSV and influenza transmission in 102 children attending childcare at two different sites, influenza was detected in 19% of attendees and RSV in 20%. Attack rates for influenza in individual classrooms ranged up to 50%. Both influenza and RSV are important respiratory viral pathogens in young children attending childcare.

Day 1	Plenary Session 1
0945	**Use of rapid influenza detection tests for outbreak detection in long-term care facilities**
Speaker	Jonathan Temte, University of Wisconsin, Madison, WI, USA

Jonathan L. Temte MD, PhD

Professor of Family Medicine and Community Health and Associate Dean for Public Health and Community Engagement, University of Wisconsin, School of Medicine and Public Health, Madison, Wisconsin, USA

Introduction

Long-term care facilities (LTCFs) are ideal environments for acquisition and spread of infection: susceptible residents live in crowded institutional settings. Outbreaks of influenza and other acute respiratory infections (ARIs) in LTCFs often occur without identification of pathogens. The Rapid Assessment of and Prophylaxis for Influenza in Dwellers of Long-Term Care Facilities (RAPID-LTCF) study (ClinicalTrials.gov Identifier: NCT02964871) is a randomized controlled trial with the primary objective to evaluate the potential benefits of using rapid influenza diagnostic tests (RIDT) in LTCFs. From interim analyses, we describe a novel approach for respiratory virus surveillance in LTCFs and performance of RIDT for LTCF residents.

Methods

Nursing staff identified LTCF residents with new onset ARI symptoms and collected specimens using a nasal swab. Nursing personnel, using the Quidel Sofia Influenza A+B FIA, tested specimens on-site. Following processing for RIDT, the residual swab was placed into viral transport medium and forwarded to the Wisconsin State Laboratory of Hygiene where it was tested for influenza using RT-PCR (IVD CDC Human Influenza Virus Real-Time RT-PCR Diagnostic Panel) and for 17 other viruses (Luminex NxTAG Respiratory Pathogen Panel [RPP]).

Results

Totals of 160, 215 and 122 specimens were collected during 2016-2017, 2017-2018, and 2018-2019, respectively. RPP identified viruses in 54.8% of tested specimens. The rate of virus detection from specimens was not related to the age of the resident ($X^2=0.917$; $P=0.821$) across four age groups, nor was it dependent on the time between symptom onset and specimen collection ($X^2=1.557$; $P=0.821$). Influenza A (19.2%), FluB (12.6%), RSV (15.9%) and hMPV (20.9%) accounted for 69% of all detections, while CoV (17.2%), R/E (10.5%) and PIV (3.8%) were less common. The distribution of viruses varied significantly across the three years ($X^2 = 71.663$; $df = 12$; $P<0.001$).
The sensitivities of Sofia Influenza A+B FIA for FluA and FluB were 77.8% [95% CI: 62.9 to 88.8] and 70.0% [50.6 to 85.3], respectively. The specificities for FluA and FluB were 98.2% [96.3 to 99.3] and 97.5% [95.4 to 85.3]. Overall, sensitivity and specificity for influenza detection was 74.7% [63.3 to 84.0] and 96.6% [94.1 to 98.2], respectively. Using a low threshold criteria for testing (i.e., new ARI symptoms) coupled with RIDT yielded a likelihood of detecting an outbreak (2 cases or more) of ≥ 84%.

Conclusions

Surveillance of respiratory viruses in LTCFs—using residual nasal swabs collected for RIDT—
is highly feasible. Influenza viruses were common and accounted for 8.9-41.7% of detections. Significant differences in virus distribution occurred across the three study years. Nursing staff-initiated and performed RIDT allowed for rapid identification of influenza with reasonably high sensitivity and specificity. Low threshold criteria for testing allowed for early detection of influenza outbreak within LTCFs.

Day 1	Plenary Session 1
1015	**Strategies for the use of enhanced influenza vaccines in older adults**
Speaker	Ben Cowling, The University of Hong Kong, Hong Kong SAR, China

Benjamin J. Cowling

School of Public Health, The University of Hong Kong

Older adults are recommended to receive annual influenza vaccination. Enhanced influenza vaccines could provide improved protection over standard inactivated influenza vaccines. The objective of our study was to compare immune responses in older adults randomized to receive either one of three enhanced vaccines or standard dose vaccine. We conducted a randomized controlled trial in Hong Kong, enrolling community-dwelling older adults 65-82 years of age. We randomly allocated participants to the 2017/18 and 2018/19 northern hemisphere formulations of: (1) standard dose quadrivalent subunit inactivated influenza vaccine (FluQuadri, Sanofi Pasteur); (2) MF59-adjuvanted inactivated trivalent influenza vaccine (FLUAD, Seqirus); (3) High-dose inactivated trivalent influenza vaccine (Fluzone High-Dose, Sanofi Pasteur); or (4) Recombinant-hemagglutinin high-dose quadrivalent influenza vaccine (Flublok, Protein Sciences). We collected sera before and 30 days after vaccination and tested sera using hemagglutination inhibition (HAI) and virus microneutralization (MN) assays against relevant strains. From 7 October 2017 through 12 January 2018 we randomized 1861 older adults to the four vaccine groups. Mean fold rise (MFR) in HAI titers to egg-propagated A(H1N1) and A(H3N2) and MFR in MN to cell-propagated A(H3N2) were statistically significantly higher in the enhanced vaccine groups compared to standard-dose vaccine. MFR in MN to cell-propagated A(H3N2) was highest among rHA recipients (4.7) followed by high-dose (3.4) and MF59-adjuvanted (2.9) compared to standard-dose recipients (2.3). Similarly, the ratio of post-vaccination MN titers among rHA recipients to cell-propagated A(H3N2) was 2.57-fold higher than standard-dose, which was statistically higher than the high-dose (1.33-fold) and MF59-adjuvanted (1.43-fold) recipient ratio. Enhanced vaccines also resulted in boosting of T cell responses. In summary, older adults receiving enhanced vaccines showed improved humoral and cell-mediated immune responses compared to standard-dose vaccine recipients.

Day 1	Plenary Session 2
1115	**Pandemic Influenza in Australia -1919 to 2019**
Speaker	Alan Hampson, Federation University, VIC

Alan W. Hampson

Adjunct Senior Research Fellow, School of Health & Life Sciences, Gippsland Campus, Federation University, Australia

The centenary of the Spanish Influenza pandemic was widely observed by events conducted in 2018, however, the true centenary of the outbreak in Australia is 2019. Maritime quarantine delayed the entry of the virus into the general population until January 1919 but quarantine of troops returning from WW1 was both controversial and unpopular and its breakdown probably responsible for the eventual spread into the population. Despite forward planning between the States and the Commonwealth, the outbreak precipitated both chaos and tension within the relatively new Australian federation of states. While the pandemic then ran its inevitable course, Australia experienced close to the lowest per capita mortality of any country.

While, in the belief of a bacterial aetiology bacterial vaccines were used in the 1919 outbreak, reportedly with some degree of benefit, the subsequent four pandemics (1957, 1968-9, 1977*, 2009) occurred in the 'virological' era with the potential for intervention with viral vaccines. Although vaccines were prepared in all four cases only in the 1969 Hong Kong pandemic outbreak in Australia, in contrast to the case in other countries, were they successfully delivered ahead of the major outbreak which was due to its unusual course here.

The history of pandemic influenza can be viewed against an interesting historical background of influenza surveillance, vaccine production and formulation in Australia and globally. While much has been learned about pandemics, including their social consequences, there remain unexplained issues including subtype replacement which just might hold a key to more successful intervention.

*the classification of the 1977 'Russian flu' as a pandemic is controversial, however, it does seem to meet the WHO definition updated in 2009.

Day 1	Plenary Session 2
1135	**Influenza surveillance in the WHO African Region**
Speaker	Belinda Herring, WHO Regional Office, Brazzaville, Republic of the Congo, Africa

Belinda L. Herring, Soatiana C. Rajatonirina, Ali Ahmed Yahaya, Mamoudou Harouna Djingarey, and Zabulon Yoti.

WHO Health Emergencies Programme, WHO Regional Office for Africa, Djoue 06, Brazzaville, Congo.

It is estimated that globally, the highest burden of influenza morbidity and mortality is the WHO African Region. Additionally, one fifth of all deaths in children aged less than five years are attributable to pneumonia or respiratory disease. However, the proportion of these deaths resulting from influenza infection in the African region is unknown and a comprehensive picture of the epidemiology of human influenza infection in the WHO African Region (47 Member States) is lacking. There remains a need to accurately present the impact of influenza in the African Region so that health policy can be developed and influenza is prioritized as a potential threat to global health security.
In the region influenza surveillance is conducted as part of the Integrated Disease Surveillance and Response System (IDSR). Additionally, a number of countries have also engaged in sentinel surveillance for influenza which encompasses reporting of virological and epidemiological surveillance data. Currently 34 countries have laboratory capacity to detect influenza using PCR (WHO minimum standard) and a network of 27 laboratories report influenza virological data to the Global Influenza Programmes' data platform FluNet and 20 countries are reporting epidemiological from ILI and/or SARI cases to FluID platform. However, further expansion of the influenza network in the region is required to provide comprehensive influenza data for public health decision making.
Despite the significant gains in the region since the 2009 H1N1pdm09 pandemic challenges still remain, most notably sustainability of systems, competing interests i.e. other infectious outbreaks in countries, a lack of preparedness plans for a potential pandemic and the low priority given to influenza.
WHO/AFRO and partners continue to work building influenza surveillance capacity in the region to strengthen health security and pandemic preparedness.

Day 1	Plenary Session 2
1155	**Chronic medical conditions and severe influenza virus infections**
Speaker	Kirstie Short, University of Queensland, Brisbane, QLD

Kirsty R. Short

School of Chemistry and Molecular Biosciences, The University of Queensland, Australia

The current obesity and type 2 diabetes epidemics represent two of the biggest health crises of the 21st century. Obesity and type 2 diabetes confer susceptibility to numerous different complications, including severe influenza virus infections. Specifically, both conditions significantly increase the risk of being hospitalised with, and dying from, influenza. The mechanisms that underscore this susceptibility remain poorly defined. Similarly, the role of these conditions in viral transmission and impairing an individual's immune response to both vaccination and natural infection are only just beginning to be elucidated. Here, I will discuss our latest research in our understanding of the role of both obesity and diabetes in severe influenza virus infections and what this means for influenza pandemic preparedness. Given that 7 in 10 Australians are overweight/obese and 1 in 11 Australians are living with diabetes this intersection between communicable and non-communicable disease remains of paramount importance.

Day 1	Plenary Session 2
1215	**Human infection of a locally acquired swine-origin influenza A(H3N2) variant in Australia**
Speaker	Yi-Mo Deng, WHO CC, Doherty Institute, Melbourne, VIC

Yi-Mo Deng[1],Frank Y K Wong[2], Natalie Spirason[1], Matthew Kaye[1], Rebecca Beazley[3], Miguel Grau[4], Songhua Shan[2], Vittoria Stevens[2], Kanta Subbarao[1], Sheena Sullivan[1, 5], Ian G Barr[1,5], Dhanasekaran Vijaykrishna[1,4]

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[2] CSIRO Australian Animal Health Laboratory, Geelong, Victoria, Australia
[3] South Australian Department of Health and Wellbeing, Adelaide, South Australia, Australia
[4] Monash University, Melbourne
[5] University of Melbourne, Melbourne

The long-term circulation of influenza A viruses (IAVs) in swine populations poses a potential threat to public health. The 2009 pandemic was caused by a reassortant swine influenza A H1N1 virus with genes that originated from human and avian IAVs that had circulated in swine for several years. Enhanced influenza surveillance globally in swine since this period has shown the continuous introduction of human seasonal influenza viruses into swine, followed by reassortment with influenza A viruses endemic in swine (IAV-S), with many lineages persisting in swine for several decades.

While IAV-Ss are normally limited to transmission amongst pigs, 430 cases of human infection of swine-origin influenza A(H3N2) variant viruses (H3N2v) have been detected in the US since 2010, primarily infecting young people exposed to swine at agricultural fairs. Most of the cases had self-limited influenza-like illnesses. Recent data also suggests that IAV-Ss have been endemic in Australia for many decades, including viruses that were originally derived from human H3N2 viruses as early as 1968, pre-2009 seasonal H1N1 viruses, and H1N1pdm09 viruses

We report a case of a human infected with swine influenza A/H3N2 variant virus in Australia containing H3 and N2 genes derived from 1990's-like human seasonal viruses and internal protein genes from H1N1pdm09. Our results highlight the potential risk of Australian swine influenza A to human health.

Day 1	Plenary Session 2
1230	**Ferret model of influenza transmission**
Speaker	Hui-Ling Yen, LKS Faculty of Medicine, The University of Hong Kong, Hong Kong SAR, China

Hui-Ling Yen

School of Public Health, LKS Faculty of Medicine, The University of Hong Kong

Ferrets have been utilized for influenza research since the first isolation of human influenza virus in 1933. Influenza infection in ferrets cause clinical signs that closely resemble those of humans and may lead to onward influenza transmission to susceptible ferrets. Influenza transmission among ferrets can be mediated by multiple non-mutually exclusive modes, by direct or indirect contact of infectious respiratory secretions or by virus-laden aerosols exhaled by infected animals. Conventionally, influenza transmissibility in ferrets can be studied using the “direct contact” or “respiratory droplet contact” experimental models, where the donor and recipient ferrets are co-housed or housed in separate but adjacent cages, respectively. Under the settings, influenza A viruses that transmit well among humans have been found to transmit efficiently in ferrets under both direct contact or respiratory droplet contact models. On the other hand, zoonotic influenza viruses generally exhibit efficient transmissibility under the “respiratory droplet contact” model. Ferrets have been commonly used for influenza risk assessment studies in the past two decades. Efforts have been made to identify experimental parameters that may influence the transmission outcome with the aim of refining experimental protocols for risk assessment studies. In addition, the application of multidisciplinary technologies has advanced our understanding on influenza transmission mechanisms in this animal model.

Day 1	Plenary Session 3
1345	**Intense interseasonal influenza outbreaks, Australia, 2018/19**
Speaker	Ian Barr, WHO CC, Doherty Institute, Melbourne, VIC

Ian G Barr[1,2], Yi Mo Deng[1], Miguel L Grau[3], Alvin X Han[4,5], Robin Gilmour[6], Melissa Irwin[7], Peter Markey[8], Kevin Freeman[9], Geoff Higgins[10], Mark Turra[10], Naomi Komadina[1], Heidi Peck[1], Robert Booy[11,12], Sebastian Maurer-Stroh[4,5,13], Vijaykrishna Dhanasekaran[1,3], Sheena Sullivan[1,2]

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[7] Rapid Surveillance, Centre for Epidemiology, New South Wales Ministry of Health, St. Leonards,
[8] Centre for Disease Control, Northern Territory Department of Health, Darwin, Northern Territory,
[9] Serology/Molecular Biology Territory Pathology, Royal Darwin Hospital, Northern Territory Government Health, Darwin, Australia
[10] Microbiology and Infectious Disease Directorate, SA Pathology, Adelaide, Australia
[11] National Centre for Immunisation Research and Surveillance (NCIRS), Westmead, Australia
[12] Department of Paediatrics and Adolescent Health, Faculty of Health and Medicine, Uni. Sydney
[13] Department of Biological Sciences, National University of Singapore, Singapore

In 2018, the Australian influenza season was late and progressed with such minimal activity that it barely registered as a season by several surveillance indicators. This was in stark contrast to the 2017 season, when Australia's highest levels of influenza activity were recorded. However, several surveillance indicators suggested that the influenza activity seen in 2018, while low, never really stopped, as it was expected to, at the end of the southern hemisphere spring (November). Instead, Australia experienced an upsurge in influenza cases with a large wet-season outbreak in the tropical north, while southern Australia saw record numbers of laboratory-confirmed influenza notifications, increased hospitalisations and dozens of influenza-related deaths in late summer and early autumn, resulting in an early start to the 2019 influenza season throughout the country.

These outbreaks in Australia reinforce the need for year-round surveillance of influenza even in regions with temperate climates with strong seasonality patterns such as Europe and North and South America. Early identification of major outbreaks can forewarn primary practitioners, aged care institutions and at-risk groups to consider bringing forward vaccination programmes (if feasible) or being alert to respiratory outbreaks and ensuring stocks of antiviral medications are on hand. It would also help alert hospitals and clinics of possible increased attendances and the possible cause of people presenting with ILI.

With high levels of summer tourists coming to Australia each year from the northern hemisphere winter (such as the US, Japan and China) or from tropical regions near Australia where influenza circulates all year (such as Singapore, Indonesia and Malaysia), there will inevitably be further introductions of influenza in the future, as we have noted previously. A better understanding of the reasons why the summer-autumn influenza outbreaks in Australia in 2018/19 were so prevalent may help to mitigate their impact on the population in the future.

Day 1	Plenary Session 3
1405	**The Reliability of Point of Care Testing in Aged Care Facilities**
Speaker	Robert Booy, NCIRS, Westmead, NSW

C Jones[1,2], E Ridgway[2], E Clarke[2], P Clark[2], S Bag[1,2], S Norton[2], J Kok[2,3], R Lindley[1,5], D Dwyer[3,4], R Booy[1,3,6]

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Background
Residents in aged care facilities (ACFs) are at higher risk of influenza and other respiratory pathogens. Early detection of influenza can prompt earlier intervention to reduce morbidity and mortality. Rapid diagnostic polymerase chain reaction (PCR) Point of Care (POC) platforms can screen for influenza within 30-60 minutes.
A study was commenced in the Western Sydney Local Health District (WSLHD) in 2018 to measure the sensitivity and specificity of POC testing in ACF's against the laboratory-based multiplex PCR.

Methods
ACF residents with an influenza like illness routinely notified to the WSLHD Public Health Unit from August 2018 to May 2019, received on-site POC PCR testing of nasopharyngeal swabs using the Cepheid GeneXpert. This is a reverse transcriptase PCR assay that can detect and differentiate between influenza A, B and RSV. The remainder of the swab was delivered to the laboratory for confirmatory testing.

Results
Rapid POC testing was carried out on 73 specimens from 16 ACF's, yielding 20 positive results (19 Flu A, 1 Flu B). Three ILI's were epi-linked and low positive (Ct≥30), all resulting in laboratory false negatives. This provided a sensitivity of 100% and specificity of 100%. Morbidity and mortality will be presented.

Conclusion
The study has found the POC test to be extremely reliable and practical in early identification of influenza, enabling timely use of anti-viral treatment/prophylaxis to decrease transmission and prevent hospitalisations. Improving outbreak management is critical in reducing the burden of respiratory disease in the elderly.

Day 1	Plenary Session 3
1425	**Flucast - a tool to predict seasonal influenza severity**
Speaker	Aye Moa, The Kirby Institute, University of New South Wales, Sydney, NSW

Background

Influenza causes serious illness requiring annual health system surge capacity, yet annual seasonal variation makes it difficult to forecast and plan for the severity of an upcoming season. Research shows that hospital and health system stakeholders indicate a preference for forecasting tools that are easy to use and understand to assist with surge capacity planning for influenza.

Objective

This study aimed to develop a simple risk prediction tool, Flucast, to predict the severity of an emerging influenza season.

Methods

Study data were obtained from the National Notifiable Diseases Surveillance System and Australian Influenza Surveillance Reports, Department of Health, Australia. We tested Flucast using retrospective seasonal data for 11 Australian influenza seasons. We compared five different models using parameters known early in the season that may be associated with the severity of the season. To calibrate the tool, the resulting estimates of seasonal severity were validated against independent reports of influenza-attributable morbidity and mortality. A model with the highest predictive accuracy against retrospective seasonal activity was chosen as a best-fit model to develop the Flucast tool. The tool was prospectively tested against the 2018 and the emerging 2019 influenza season.

Results

The Flucast tool predicted the severity of all retrospectively studied years correctly for influenza seasonal activity in Australia. With the use of real-time data, the tool provided a reasonable early prediction of low to moderate season for the 2018 and severe seasonal activity for the upcoming 2019 season. The tool meets stakeholder preferences for simplicity and ease of use to assist with surge capacity planning.

Conclusions

The Flucast tool may be useful to inform future health system influenza preparedness planning, surge capacity, and intervention programs in real time, and can be adapted for different settings and geographic locations.

Day 1	Plenary Session 3
1445	**Forecasting in an unusual season: what can we learn from 2019?**
Speaker	Rob Moss, The University of Melbourne, Melbourne, VIC

Since 2015, we have been using mathematical models to characterise seasonal influenza epidemics in near-real-time and to generate weekly epidemic forecasts for major Australian cities. In each season, we have encountered new challenges, and from these we have learned how to refine and improve our forecasting methods each year. The 2019 season was no different.

At the start of each season, before there is any evidence of epidemic activity, we begin with a large number of candidate epidemics that we expect might describe the imminent epidemic. These candidates are selected so that, as an ensemble, they are broadly consistent with previous influenza seasons, without requiring that each one is extremely similar to any particular previous season. This is a way of encoding our beliefs that the variety of influenza seasons we have previously seen is indicative of the types of influenza seasons we should expect to see in the future.

Temperate regions of Australia experienced unusual influenza activity 2019. The 2018 influenza season saw very low activity, with a small and late peak. But activity over the 2018-19 inter-seasonal period was unusually high, and epidemic activity began much earlier than was typical of previous seasons. For most of our forecasts, this meant that none of the candidate epidemics bore much resemblance to the surveillance data being reported in May 2019. So what were we to do?

We could have sampled model parameters from broad, uniform distributions — the equivalent of saying "anything is possible" — but instead we selected three different sets of candidates, based on different assumptions about the season:

1. That the season would be "typical" in scale: it had started early, so it would also peak early and end early.
2. That the season would be large in scale ("2017-like"): it had started early, so it would also peak somewhat early and end somewhat early.
3. That the season would be "typical" in terms of peak timing: it had started early, so it would be a much longer and larger season.

We then examined these 3 sets of forecasts at each week of the season, to see which of them best explained the emerging data.

In this talk I will present an overview of how these forecasts performed, before showing some preliminary results of how we can synthesise data from multiple surveillance systems to gain improved insights into influenza activity, which should help us assess current influenza impact ("now-casting") and improve our predictions of future influenza impact (forecasting) in upcoming influenza seasons.

Day 1	Roundtable discussion
1505	**What do we need to do to improve influenza surveillance in Australia and will it make any difference to the outcomes?**
Panel	• Frances Birrell • Nigel Crawford • Craig Dalton • Janet Englund • Robin Gilmour • Ian Mackay
Moderator	Sheena Sullivan, WHO CC Melbourne

Day 1	Workshop 1: Research
1600	**New strategies for influenza nucleoprotein-based diagnostic assays**
Presenter	Patrick Schaeffer, James Cook University, Townsville, QLD

The detection of proteinaceous antigens generally relies on traditional immunoassays and, more recently, on immuno-PCR assays and their derivatives, which do not take advantage of the intrinsic function or binding property of a protein. The RNA-binding nucleoprotein (NP) has been shown to be an excellent target for the development of various influenza A diagnostics due to its high antigenicity and the presence of large numbers in the virus. NP also shows potential as an antiviral drug target [1]. NP binds non-specifically to the sugar-phosphate backbone of RNA as well as to single-stranded DNA (ssDNA) in vitro. We decided to take advantage of this property to develop an ssDNA probe for the detection of nucleoprotein by quantitative PCR (qPCR). We found that recombinant influenza A nucleoprotein from avian H5N1 subtype binds strongest to a 74-base-long ssDNA. Additionally we found that NP could still bind to ssDNA after a urea denaturation step. Taking advantage of the ssDNA-binding property of NP we developed a new immunodiagnostic platform including a qPCR step for sensitive NP detection. Two different formats were successfully tested, one comprising an antibody-based nucleoprotein capture surface and the other based on direct nucleoprotein adsorption under denaturing conditions. Both formats include the replacement of RNA bound to nucleoprotein by a discrete ssDNA probe that is detected by qPCR. This new diagnostic platform afforded low picomolar (pM) detection of influenza nucleoprotein [2]. Here I will present further developments of this system into a point-of-care format for influenza NP detection.

[1] H. Antony, P.M. Schaeffer, A GFP-tagged nucleoprotein-based aggregation assay for anti-influenza drug discovery and antibody development, The Analyst, 138 (2013) 6073-6080.
[2] I. Morin, P.M. Schaeffer, Combining RNA-DNA swapping and quantitative polymerase chain reaction for the detection of influenza A nucleoprotein, Analytical biochemistry, 420 (2012) 121-126.

Day 1	Workshop 1: Research
1615	**Risk assessment of contemporary North American swine influenza viruses in murine models of infection**
Presenter	S. Mark Tompkins, University of Georgia, Athens, GA

Emily F. Beaver[1,2], Shelly Samet[1,2,3], Constantinos S. Kyriakis[1,4], and S. Mark Tompkins[1,2,5]

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[5]Presenting author

Influenza A virus (IAV) is endemic in North American swine. A relative of the 1918 pandemic influenza has circulated in swine since that pandemic and remained relatively stable, with some drift, but no reassortment. However in the 1990's, North American swine IAVs (swIAV) gained an internal gene segment constellation referred to as the triple reassortment internal gene (TRIG) cassette, which quickly became dominant within circulating swIAVs and enabled increased surface protein gene segment reassortment. Subsequent reverse-zoonotic events and antigenic drift in swine resulted in a dramatically increased diversity of HA and NA combinations and today there are nearly a dozen distinct clades of H1N1, H1N2, and H3N2 viruses co-circulating in North American swine. Following the 2009 H1N1 pandemic, reverse zoonosis and continued reassortment resulted in the pdmH1N1 origin matrix gene (pdmM) completely replacing the classical swine origin matrix gene (swM) previously found in the TRIG cassette. The rapid dominance of pdmM gene segment in circulating swIAVs suggested a strong fitness advantage for strains containing the pdmM over the swM gene segment. We hypothesized that the origin of the matrix gene could affect virus replication, pathogenesis, and the subsequent immune responses induced by swIAV infection. To confirm this, we infected BALB/c mice with a panel of H1 and H3 swIAV isolates containing either the pdmM or the swM gene segment. We assessed virus replication, disease, lung pathology, as well as cytokine and chemokine production. Infection of mice with H1 swIAVs containing the pdmM gene resulted in significantly greater morbidity and mortality compared to viruses with the swM segment, while the viruses with the pdmM also consistently replicated at higher levels. The same viruses induced an overall greater proinflammatory response and a greater chemotactic response. While other gene segments such as the NS1 or PB1-F2 traditionally antagonize the immune response, our data suggest that the origin of the matrix gene may contribute to a modified immune response against swIAV infection in mammals.

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Day 1	Workshop 1: Research
1630	**Generating Immunity to Severe Influenza Disease in Indigenous Australians**
Presenter	Louise Rowntree, The University of Melbourne, VIC

Louise Rowntree[a], Patricia Illing[b], Luca Hensen[a], Nicole Mifsud[b], Jennifer Habel[a], Liyen Loh[a], Adrian Miller[c], Steven Tong[a,d], Anthony Purcell[b] and Katherine Kedzierska[a]

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[c]Office of Indigenous Engagement, Central Queensland University, Townsville, Queensland, Australia
[d]Menzies School of Health Research, Casuarina, Northern Territory, Australia

Indigenous populations, including Indigenous Australians, are highly susceptible to severe influenza disease, however, the underlying mechanisms are unknown. While influenza vaccination can prevent infection, current vaccines have varying efficacy between individuals and require annual reformulation due to circulating strain variation. We propose targeting CD8+ T cells capable of recognising conserved influenza peptides presented by human leukocyte antigen (HLA) molecules on the surface of virus-infected cells. We have previously characterised the HLA profiles of Indigenous Australians, finding that although there is some overlap, prominent HLA molecules found in Indigenous Australians (HLA-A*02:01, 11:01, 24:02, 34:01 and HLA-B*13:01, 15:21, 40:01/02, 56:01/02) are rarely identified in the general population (Clemens et al., 2016, ICB). Furthermore, there is a relative lack of known influenza epitopes for the majority of these HLAs, with the exception of HLA-A*02:01 (Koutsakos et al., 2019, Nat Immunol) and these potential epitopes need to be considered for the rational design of universal T cell vaccines. Considering this, we used immunopeptidomics to define peptides naturally processed and presented on the surface of influenza-infected class I reduced (C1R) cells stably expressing high levels of HLA-A*34:01, the most prominent HLA allele in Indigenous Australians. Liquid chromatography-tandem mass spectrometry analyses of peptides isolated from HLA-A*34:01 complexes on the surface of infected cells yielded 80 influenza A and 39 influenza B virus-derived candidate epitopes. To dissect the influenza-specific CD8+ T cells towards these viral peptides, we probed memory CD8+ T cells in HLA-A*34:01-expressing Indigenous individuals. Peripheral blood mononuclear cells were stimulated with influenza-infected C1R.A*34:01 cells for 13 days and then restimulated with pooled or individual peptides in an IFN-□ and TNF-□ intracellular cytokine staining assay. Of the 119 peptides identified, five influenza A and five influenza B peptides were found to be capable of activating CD8+ T cells from HLA-A*34:01+ Indigenous donors. The discovery of peptides presented by high frequency HLA alleles in Indigenous Australians, including HLA-A*34:01, potentiates their use in CD8+ T cell-based vaccines to generate broad immunity against influenza viruses.

Day 1	Workshop 1: Research
1645	**Host directed therapy to regulate neutrophil in an *in vitro* model of influenza infection**
Presenter	Maryam Shojaei, Westmead Institute for Medical Research, Westmead, NSW

Maryam Shojaei[1,2], Ya Wang[1,2], Sally Teoh[1], David Booth[2], Anthony McLean[1] and Benjamin Tang[1,2]

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Objectives

It is increasingly recognized that neutrophils play an important role in host defence to viral infections including influenza. Our previous research found that a neutrophil-specific biomarker, CD177 is the most highly expressed gene in patients who developed respiratory failure and required mechanical ventilation. Host-direct therapy, such as those targeting excessive neutrophils response, could improve host survival. This study aims to provide baseline measurements on the effects of interferon Lambda (IFNλ) on CD177 and neutrophil states in an in vitro model of influenza infection.

Methods

Neutrophils were isolated directly from fresh whole blood collected in EDTA tubes (n=3) using MACSxpress neutrophil isolation kit. Purity of isolated neutrophils was measured by flow cytometry after staining with CD66b, CD11b and CD45. Recovery of untouched isolated neutrophils were also assessed by flow cytometry before and after separation. Isolated neutrophils were pre- stimulated H1N1 followed by treatments with different concentrations of IFNλ for 1,3 and 6 hours. Samples were harvested at the appropriate time points. Expression level of CD177 was measured by qPCR platform.

Results

Flow cytometry result shows that quality of the isolated neutrophils was adequate for carrying out the experiment (purity of neutrophils reached 98% and recovery rates was>80%). Neutrophil viability was very similar between treated and untreated conditions, as assessed by staining of the apoptosis and necrosis markers Annexin V and PI. In treatment groups, IFNλ therapy reduced the capacity of separated neutrophils to express CD177 in response to stimulation with H1N1 by 80% after 1 hour, without impacting adversely on cell survival.

Conclusion

Viral infection stimulates neutrophils and triggers neutrophil- mediated immune responses. Findings from this study demonstrates IFNλ does not cause cell death and could potentially decrease expression of CD177 in activated neutrophils in an in vitro model of influenza infection.

Day 1	Workshop 1: Research
1700	**Evaluating the window of susceptibility to secondary bacterial infections post-influenza infection in ferrets**
Presenter	Edin Mifsud, WHO CC, Doherty Institute, Melbourne, VIC

Edin J Mifsud[1,2], Patrick Reading[1,2], and Aeron Hurt [1,2]

[1] WHO Collaborating Centre for Reference and Research on Influenza, VIDRL, Peter Doherty Institute for Infection and Immunity, Melbourne, Victoria 3000, Australia, 2 University of Melbourne, Department of Microbiology and Immunology, Peter Doherty Institute for Infection and Immunity, Melbourne, Victoria 3000, Australia

Influenza virus infections cause epithelial cell damage and reduced Toll-like receptor responsiveness, which increase susceptibility to secondary bacterial infections (SBI) resulting in increased morbidity and mortality. Streptococcus pneumoniae (SPN) is the most common bacterial pathogen associated with SBIs. We examined interactions between influenza and SPN in ferrets, with the aim of establishing an animal model to evaluate how antiviral treatment may impact SBI progression following influenza infection.

Ferrets were infected intranasally with 103 TCID50 of influenza A(H1N1)pdm09 virus and 5 or 10 days later were infected intranasally with 103 colony forming units of the 19F strain of SPN. Control ferrets were infected with either virus or bacteria alone. Nasal washes were collected daily to enumerate viral and bacterial loads, and clinical signs were monitored.

Compared to ferrets infected with bacteria alone, animals that received SPN 5 days post-influenza showed accelerated disease signs including laboured breathing, lethargy and dehydration. In these ferrets, the bacterial burden in the upper respiratory tract (URT) was approximately 100 to 1000-fold higher than those infected with SPN alone. Conversely, a SBI 10 days after influenza infection resulted in few or no disease signs, although bacterial burden in the URT was approximately 10-fold higher than in animals infected with SPN alone. At both time points, bacteraemia occurred in 1 of ferrets that had been infected with influenza + SPN, but was absent in ferrets infected with SPN alone. Experiments to determine whether treatment with influenza antivirals would reduce the susceptibility to SBI and associated disease severity are ongoing.

Day 1	Workshop 1: Research
1715	**Hyperglycaemia induces epithelial-endothelial barrier damage during Influenza A infection**
Presenter	Katina Hulme, The University of Queensland, Brisbane, QLD

Katina D. Hulme[1], Rebecca J. Marshall[1], Limin Yan[1], Conor J. Bloxham[2], Kyle R. Upton[1], Katharina Ronacher[3], Linda A. Gallo[2,3] & Kirsty R. Short[1,4]

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Introduction

Diabetes mellitus is on the rise globally and is a known susceptibility factor for severe influenza virus infections. However, the mechanism by which diabetes increases the severity of influenza is yet to be fully defined. Here, the effects of high glucose levels on influenza severity were investigated.

Methods

To mimic the pulmonary epithelial-endothelial barrier, human epithelial cells were seeded on the top side of a permeable membrane and primary human pulmonary endothelial cells were seeded on the underside. Once epithelial cells reached confluency, the media in both upper and lower compartments was refreshed every 12 hours. The medium of the lower compartment was refreshed with either 7mmol/L (to represent normal glucose conditions) or 12mmol/L (to represent the hyperglycaemia seen in diabetes). After four days in differential glucose conditions, influenza A/Solomon Islands/03/2006 (H1N1) was added to the upper compartment of the trans-well system. After infection, various assays, qPCR and RNA-Seq was performed to measure barrier integrity and inflammation. Additionally, epithelial cells were fixed and stained using IHC to investigate tight junction integrity.

Results

Using an in vitro co-culture model of the pulmonary epithelial-endothelial barrier, we show that, compared to normal glucose levels, high glucose conditions (a hallmark of diabetes mellitus) prior to influenza A virus infection increases barrier damage. Increased barrier damage was not associated with increased cell death, but rather an increased endothelial cell pro-inflammatory response and subsequent degradation of epithelial cell tight junctions.

Conclusions

This study demonstrated for the first time that hyperglycaemia may increase influenza severity by damaging the pulmonary epithelial-endothelial barrier and increasing pulmonary oedema. This understanding is imperative for the development of therapeutic approaches tailored for vulnerable patient groups infected with influenza virus.

Day 1	Workshop 1: Research
1730	**Chicken and duck endothelial cells display a markedly different innate immune response to viral challenge**
Presenter	Marcus Tong, The University of Queensland, Brisbane, QLD

Zhen Wei Marcus Tong[1], Anjana C. Karawita[1], Arjun Challagulla[2], Lee Trinidad[2], Sue Lowther[2], Mathilde Richard[3], Tim J. Doran[2], Michelle L. Baker[2] & Kirsty R. Short[1,4]

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[4]Australian Infectious Diseases Research Centre, The University of Queensland, Brisbane Australia

Introduction

Highly pathogenic avian influenza viruses (HPAIVs) represent an ongoing threat to the poultry industry, impacting animal health and causing major economic losses worldwide. Chickens (Gallus gallus) are highly susceptible to HPAI whilst ducks (Anas platyrhynchos) are typically resistant. In chickens, HPAIVs primarily infect the endothelium leading to cell death, oedema, haemorrhaging, microthrombosis, disseminated intravascular coagulation and disruption of the innate immune response. Together, these features help account for the rapid and high mortality rates of HPAI in gallinaceous species. In contrast, HPAIVs rarely infect the duck endothelium and this is likely to account for the reduced disease severity seen in these birds. Here, we seek to characterize species dependent differences in the innate immune response of avian endothelial cells in order to understand their differential susceptibility to HPAI.

Methods

Primary chicken and duck endothelial cells were cultured from the aorta and bone marrow of embryonated eggs. The identity and purity of these cell cultures was confirmed by RT-PCR, uptake of Ac-LDL and tube formation assays. Cells were subsequently challenged with poly (I:C) (a TLR3 agonist and mimic of viral infection) or HPAIV (A/chicken/Vietnam/0008/2004 (H5N1)) and the transcriptome was characterized.

Results

Chicken endothelial cells from both the bone marrow and aorta expressed significantly more pro-inflammatory cytokines than duck endothelial cells after challenge with poly (I:C). In contrast, no difference was observed in IFNα expression. To further understand these species dependent differences, RNA-Seq is currently being performed on duck and chicken endothelial cells infected with A/Vietnam/04(H5N1).

Conclusions

Here, we provide the first evidence that chicken and duck endothelial cells have a differential innate immune response to viral challenge. These data represent an important first step towards understanding species dependent differences in the pathogenesis of HPAIVs.

Day 2	Plenary Session 4
0830	**AS03-adjuvanted pandemic vaccines: clinical updates**
Speaker	Philippe Buchy, GSK Vaccines Asia Pacific, Singapore

The provision of a sufficient number of doses of a pandemic influenza vaccine in a timely manner is necessary to control the spread of the disease. One way to increase the availability of pandemic vaccines is to use an adjuvant with antigen sparing propriety, decreasing the amount of antigen needed per dose. GSK's proprietary adjuvant system AS03 has been shown to be well tolerated when combined with antigens including H5N1 and H7N9, and allows a substantial reduction in the amount of antigen needed to elicit a satisfactory immune response. Clinical trials have shown that AS03-adjuvanted pandemic influenza vaccines have an acceptable safety profile. As well as improving vaccine immunogenicity, there is some evidence that the addition of AS03 to H5N1 influenza vaccine may induce some cross-clade antibody responses to heterologous H5N1 strains. Alternative H5N1 vaccination schedules were evaluated in study Q-H5N1-009 (two doses 3.75 µg HA/AS03A given 0, 7, 14 or 21 days apart and study D-H5N1-012 (two doses 3.75 µg HA/AS03A given 6 months or 12 months apart. These studies indicated that an accelerated dosing schedule could be used to reduce the time to effective immunization during a pandemic, despite lower HI antibody responses. Populations at risk could therefore be primed with a single vaccine dose in the pre-pandemic setting or on the announcement of a pandemic, with the second dose administered up to 12 months later without compromising the immune response.

In children, two doses of 1.9 µg HA/AS03B, i.e. half the adult dose of antigen and adjuvant, given 21 days apart was selected based on the results of the dose-ranging study. Primary vaccination with H5N1 vaccines elicited strong immune responses against the vaccine strain in children 6 months to 17 years of age (HI antibodies and neutralizing antibodies), exceeding European and US immunogenicity criteria for the HI antibody responses 21 days after the second dose. D-Pan H5N1 vaccine also induced notable cross-reactive immune responses against drifted H5N1 strains, including both antigenically distant strains and strains within the same clade as the vaccine strain.
Two doses of Q-Pan AS03-adjuvanted H7N9 vaccine (Day 0, 21) induced strong HI immune responses to the vaccine strain in adults (18–64 years of age). All groups who received adjuvanted vaccine exceeded the European and US immunogenicity criteria for the vaccine strain 21 days after the second dose. The vaccine responses against homologous and heterologous strains persisted for at least 12 months after the first vaccine dose. As for H5N1, H7 antigens required adjuvantation, as unadjuvanted vaccine elicited much lower immune responses than their adjuvanted equivalent. In study Q-H9N2-001, adults (18–64 years of age) were randomised to receive two doses (Day 0, 21) of one of five formulations of Q-Pan H9N2 vaccine (1.9 µg or 3.75 µg HA with AS03A or AS03B or 15 µg HA unadjuvanted) followed by a booster dose or saline at Month 6. All European and US immunogenicity criteria were met for the vaccine strain 21 days after the first and second doses and at Month 6.

Day 2	Plenary Session 4
0845	**Recent developments in Influenza vaccines manufactured in mammalian cells**
Speaker	Chris Clarke, Seqirus Ltd, Melbourne, VIC

Christopher Clarke, Jonathan Anderson (Seqirus Australia Pty Ptd)

The first trivalent influenza vaccine manufactured in mammalian cells (TIVc) was licensed in Europe and the United States more than 10 years ago. An extensive clinical development program, demonstrated that efficacy, immunogenicity and safety of this vaccine manufactured in mammalian cells using a viral seed derived in eggs was similar to vaccines manufactured in eggs.[1]

Since that time, significant advances in the production of cell-based influenza vaccines have occurred. Firstly, the cell-based vaccine is now quadrivalent (QIVc) including two A and two B strains.1 Secondly, the vaccine is manufactured in a state-of-the–art-facility, constructed in collaboration with Biomedical Advanced Research and Development Authority (BARDA), using optimised techniques that have allowed larger production volumes and consequently much wider availability of the vaccine.2 Lastly, the vaccine is now not only manufactured in mammalian cells, but also from seed viruses derived from mammalian cells. This simple step mitigates the risk of antigenic mismatches that can occur during egg adaption and are known to impact on vaccine effectiveness, particularly for A/H3N2 strains.[1]

In 2017/18 northern hemisphere (NH) season, the only vaccine strain for which a cell seed was used was A/H3N2, but from 2019/20 NH season all four vaccine strains in QIVc will be derived from cell seeds. There are early signs suggest the use of cell seeds may offer a clinical benefit over vaccines manufactured in eggs. Point estimates for relative vaccine effectiveness from the 2017/18 season in United States consistently favoured QIVc though results varied depending on study population and design and confidence intervals spanned zero in some cases[1,3-5].

Cell based production of influenza now not only offers improved manufacturing flexibility and scalability but may also bring additional clinical benefit over vaccines produced using egg-based technology. The magnitude of such a benefit is likely to vary annually depending on the extent of egg adaption and the nature of circulating strains in a given season.

1. Lamb Drugs. 2019 Aug;79(12):1337-1348
2. https://globalbiodefense.com/2017/08/29/pandemic-preparedness-seqirus-quadruples-manufacturing-output-for-flu-vaccine/
3. Izurieta J Infect Dis. 2018 Dec 18
4. LeMarcus Vaccine. 2019 Jul 9;37(30):4015-4021
5. Bruxoort Vaccine. 2019 Sep 16;37(39):5807-5811

Day 2	Plenary Session 4
0900	**Development of Cell Culture-based Influenza Vaccine, SKYCellflu®**
Speaker	Hun Kim, SK Biosciences, Korea

It is known that cell culture-based influenza vaccine has several advantages compared with egg-based influenza vaccine. Therefore, SK bioscience had introduced cell culture system for manufacturing influenza vaccine and got approval of SKYCellflu®, cell culture-based seasonal trivalent influenza vaccine, from Ministry of Food and Drug Safety (MFDS, formerly Korean FDA) in December 2014. The advantages of cell culture production system used by SK bioscience are as follows; It allows the production of a greater amount of influenza vaccine in a shorter amount of time and it also has lower risk of safety issues such as allergic reactions to egg components. These advantages of cell culture production system allow enhanced preparedness against pandemic influenza as well as the shortage of seasonal influenza vaccine. For the production of SKYCellflu®, SK bioscience has developed a unique Madin-Darby Canine Kidney (MDCK)-derived cell line, named MDCK-Sky that can be grown in suspension cultures with serum free media, thereby obtaining a reliable production yield with a relatively simple and highly efficient manufacturing process. Furthermore, SK bioscience has adopted single use bioreactor system for cultivation of influenza virus and this closed system has enabled to produce influenza vaccine with reduced opportunities of contamination, production time and costs.

Day 2	Plenary Session 4
0915	**Influenza vaccination by Vaxxas high-density micro-array patch (HD-MAP); results from a phase I clinical trial and real-world applications**
Speaker	Charles Ross, Vaxxas, Brisbane, QLD

Background

The HD-MAP is a high-density micro-array patch for vaccine delivery into the skin. We have conducted a phase I trial using the HD-MAP to deliver a monovalent influenza vaccine. This is the first clinical evaluation of the vaccine dosesparing potential of a MAP.

Method

HD-MAPs were coated with a split inactivated influenza virus vaccine (A/Singapore/GP1908/2015 [H1N1]) (A/Sing). Healthy volunteers were vaccinated with doses of 15, 10, 5, or 2.5 µg of A/Sing haemagglutinin (HA) via HD-MAP applied to the forearm (FA), or 15 µg HA via HD-MAP applied to the upper arm (UA). Control groups received uncoated HD-MAPs applied to the FA ('placebo control') or commercially available Afluria® quadrivalent influenza vaccine (QIV) delivered intramuscularly (IM) to the deltoid.

Results

The A/Sing vaccine coated onto HD-MAPs was stable when stored at 40°C for at least 12 months. HD-MAP vaccination was safe and well-tolerated; any AEs were mild or moderate. 2.5 µg HA administered by HD-MAP induced haemagglutination inhibition (HAI) and microneutralization (MN) titres that were not significantly different to those induced by 15 µg HA injected IM. HD-MAP delivery of 15 µg (FA and UA) and 10 µg (FA) HA resulted in a faster increase in HAI responses than IM injection, with 83%, 95% and 90% subjects respectively seroconverting at day 8, compared with 68% for the IM QIV group. The results indicated that overall, HD-MAP delivery induced a range of responses that were similar or potentially superior to those seen with IM injection of QIV.

Summary

Vaccination using the HD-MAP that can be stored outside the cold-chain, was safe and well-tolerated and resulted in immune responses that were equivalent to or enhanced compared with IM injection. Using the HD-MAP, a 2.5 µg dose (1/6 of the standard dose), induced HAI and MN titres equivalent to those seen with 15 µg HA injected IM. Removal of cold-chain, simplicity of use and the potential enhanced immunogenetic advantages of dose sparing and early onset kinetics makes the HDMAP ideal for real world applications such as pandemic influenza and developing countries.

Day 2	Plenary Session 4
0930	**Influenza rapid test**
Speaker	Sean Parsons, Ellume Pty Ltd, Brisbane, QLD

Day 2	Plenary Session 4
0945	**Rapid and portable PCR influenza testing - taking point of care to the ED and beyond**
Speaker	Emily Mahon, Roche Diagnostics, Sydney, NSW

The Roche cobas® Liat® instrument is a molecular point of care instrument for real-time polymerase chain reactions (PCR) testing. As it is a small, easy to use and portable analyser, it enables rapid and on-demand molecular testing at the point of care. With only one minute sample handling, Influenza A/B and RSV results can then be obtained within 20 minutes. The performance and flexibility of the system offers the opportunity to change the way we test influenza, reaching beyond the Emergency Department and into the community.

Day 2	Plenary Session 4
1000	**Cepheid C360: Turning Insights into Action**
Speaker	Daniela Symons-Troy, Cepheid, Australia

In today's information age data rules and it represents an expanding set of powerful opportunities. As much as 70% of clinical decisions may be informed by data from the medical laboratory and the vast amount of data being created is growing at an exponential pace. Managing this significant volume of information quickly, correctly and securely is increasingly more difficult and complex. However, the challenge isn't simply making the data available. The ability to access and share meaningful insights from the wealth of information the laboratory generates is a necessity. Cepheid is responding to this opportunity by introducing Cepheid c360, a powerful innovative connectivity solution. Cepheid c360 is a secure, hosted platform that securely collects and aggregates in dashboard format real-time information from any GeneXpert system.

Whether you function in a centralised or decentralised testing environment, Cepheid c360 empowers healthcare professionals with actionable insights to drive better outcomes from disease surveillance and reporting to system performance monitoring, without compromising data integrity or demanding significant resources to deploy and manage. By having a comprehensive 360-degree view of your testing data accompanied with seamless streamlined communication you can strengthen your institutions disease surveillance, monitoring and reporting capabilities in a secure, compliant and sustainable manner.

Day 2	Workshop 2: Epidemiology
1100	**Surveillance of respiratory disease outbreaks in Residential Aged Care Facilities in Queensland: 2012 – 2019**
Presenter	Frances Birrell, Queensland Health, Brisbane, QLD

Frances Birrell, John Marquess, Angela Wakefield, Karen Heel, Paul Van Buynder, Bonnie Macfarlane, Penny Hutchinson

Background

Passive surveillance of laboratory confirmed influenza limits the ability to identify cases associated with Residential Aged Care Facility (RACF) outbreaks in Queensland. Routine influenza surveillance involves direct electronic transfer between laboratory information systems and the Queensland Health Notifiable Conditions Register (NoCS).

During 2012, formal state-wide surveillance of RACF respiratory outbreaks was introduced. The primary objective was to establish a central data repository to enable estimation of the magnitude, frequency, distribution and duration of outbreaks; with the aim of informing, and potentially improving, outbreak prevention and control strategies.

There are approximately 600 RACFs in Queensland with 70 percent in the south east of the state. Most other facilities are located near the larger regional population centres.

Methods

Public Health Units (PHUs) play an integral role in supporting RACFs with the confirmation, investigation and management of outbreaks. The Queensland Health Communicable Diseases Branch (CDB) hosts a central database to record aggregated outbreak level data collected by PHUs. This includes facility details, pathogen(s), number of confirmed cases, number of individuals at risk, outbreak duration, vaccination coverage and use of antiviral prophylaxis.

PHUs access individual case data during outbreak management and, where the pathogen is influenza, can assign a system-generated unique identifier in NoCS for each outbreak. Outbreak identifiers are used to link the institutional outbreak data to individual laboratory confirmed cases for further analysis.

Results

The outbreaks database includes summaries for approximately 800 RACF outbreaks of respiratory disease, to 30/09/2019, with approximately 70 percent identifying influenza as the only, or main, aetiological agent. Summary analysis, utilising these routinely collected data, will be presented to outline the characteristics of RACF respiratory outbreaks in Queensland.

Conclusions and Recommendations

RACF outbreak surveillance in Queensland has evolved since its introduction in 2012. However, despite improvements in data quality over this period, some data are consistently difficult to obtain. Linkage of RACF outbreak and NoCS data provides an opportunity to better characterise the epidemiology of influenza outbreaks among these vulnerable populations. The future and sustainability of this surveillance activity in Queensland will be discussed.

Day 2	Workshop 2: Epidemiology
1115	**Mathematical modelling to inform the national seasonal influenza vaccination policy: findings from our study**
Presenter	George Milne, University of Western Australia, Perth, WA

George Milne, Joel Kelso, Simon Xie, University of Western Australia; Sheena Sullivan, Vivian Leung, WHO Influenza Centre; Hannah Moore, Rosie Barnes, Tom Snelling, Telethon Kids Institute; Jessica Wong, Ben Cowling, University of Hong Kong

In 2017 the Commonwealth Department of Health commissioned a consortia from the WHO Influenza Centre Melbourne, Telethon Kids Institute, University of Hong Kong and the University of Western Australia to (1) determine the influenza health burden in Australia and (2) using mathematical modelling, determine the effectiveness of alternative vaccination strategies which may significantly reduce the burden. The Phase 1 epi study used data from most states and territories averaged over 10 years (2007 to 2016), giving the relationship between case numbers and hospitalisation and mortality rates. This phase also determined current vaccination coverage by age class, giving a *baseline* vaccination scenario.

The modelling study involved developing individual-based (*c.f.* agent based) models for Albany, Newcastle and Cairns, with an overall population of ~400,000, following methods developed previously at UWA. The models were applied separately to determine the effectiveness of potential changes to Australia's current influenza vaccination "profile". These included increased vaccination coverage to at-risk groups, to school-age children, use of enhanced vaccines to those aged 65 and above, and LAIV replacing QIV for those aged 3 to 17 years; a total of 60 scenarios were evaluated. These results were then scaled to an Australian population of ~24.7 million.

The effectiveness of each alternative vaccination strategy was determined by the reduction in health burden, between the current vaccination baseline and the new strategy. As we model each individual in each of the 3 communities, we also determined the direct *vs* indirect protection afforded by vaccination, the herd immunity effect.

This talk will present the key study results.

Day 2	Workshop 2: Epidemiology
1130	**Preventing influenza illness in young infants through maternal vaccination: a multisite, multiyear analysis**
Presenter	Jocelynne McRae, National Centre for Immunisation Research and Surveillance, Sydney, NSW

McRae J, Blyth C, Cheng A, Quinn H, Wood N and Macartney K.

Background & Aims

Infants <6 months of age are at high risk of severe hospitalised influenza. As they are unable to be vaccinated, protection is provided through influenza-specific antibody transfer during pregnancy. This study explored risk factors for hospitalisation and predictors for disease severity. Maternal vaccine coverage and vaccine effectiveness (VE) in Australian infants were assessed for a three-year period.

Methods

Data were captured using two active prospective Australian sentinel hospital surveillance networks: The Influenza Complications Alert Network (FluCAN) and the Paediatric Active Enhanced Disease Surveillance (PAEDS) network. Burden of disease for infants aged <6 months hospitalised with laboratory-confirmed influenza (LCI) was performed for infants captured 2011-2018. Multivariate regression analyses were performed for factors predicting severity outcomes. Using a test-negative design case-control study, maternal VE against early infant hospitalisation was estimated using multivariable logistic regression. Control infants provided an estimate of maternal vaccine coverage.

Results

Of 455 LCI cases from 2011-18, 15.5% were Indigenous Australians, 30.0% had an underlying medical condition (including prematurity), and 39.6% were aged <2 months. Influenza A accounted for 78.5% and influenza B for 20.7%. The median LOS was 2 days. Factors associated with intensive care unit (ICU) admission (14%) were oseltamivir use (OR 9.9, 95% CI: 4.34, 22.68), medical comorbidities (OR 8.76, 95%CI: 3.57, 21.48), age <1 month (OR 6.75, 95%CI: 1.76, 25.87), prematurity (OR 3.69, 95%CI: 1.48, 9.18) and influenza B (OR 2.27, 95%CI: 1.03, 4.99). Factors associated with increased hospital length of stay (LOS) included medical comorbidities (IRR 4.24, 95%CI: 3.29, 5.46) and ICU admission (IRR 3.98 95%CI: 3.00, 5.28). Overall maternal vaccine uptake 2016-2018 among cases and controls was 34.1% and 38.9% respectively. In 2018 when influenza A/H1N1 predominated, maternal VE against infant hospitalisation up to age 6 months was 77% (OR 0.23, 95% CI: 0.05, 0.99); VE was much lower in 2017 (OR -8%, 95% CI: -142%, 52%) when influenza A/H3N2 predominated, and consistent with all-age effectiveness.

Conclusions

Infants aged <6 months experience severe influenza. Most have no risk factors other than their age. Maternal vaccination is effective at reducing infant disease, but varies by season. Improved uptake of maternal influenza vaccine to better infant health outcomes is a public health priority.

Day 2	Workshop 2: Epidemiology
1145	**Influenza Associated Myositis: a single-centre, 5-Year retrospective Study**
Presenter	Philip Britton, University of Sydney, NSW

James Kerr[1], Kristine Macartney[2,3], Philip N Britton[3,4]

1. School of Public Health, University of Sydney;
2. National Centre for Immunisation Research and Surveillance, the Children's Hospital at Westmead;
3. Discipline of Child and Adolescent Health, Sydney Medical School, University of Sydney;
4. Department of Infectious Diseases and Microbiology, the Children's Hospital at Westmead, Sydney, Australia.

Background

Myositis is a known but under-recognised uncommon complication of influenza. This was evident when a cluster of influenza-associated (IAM) presented to our hospital in 2015. We aimed to describe the clinical features and epidemiology of IAM at a single centre over a 5 year period.

Methods

We identified cases of hospitalised myositis retrospectively from 2011 to 2015 using ICD-10 codes. We performed chart reviews to collect demographic, clinical, laboratory and outcome data. We excluded myositis with non-viral causes, and cross referenced ICD-10 coded cases with a laboratory record of influenza-positive hospitalised children to identify cases of confirmed IAM. We defined probable IAM as viral myositis occurring during the influenza season. We described epidemiological and clinical features of viral myositis and IAM and compared IAM (confirmed and probable) with all hospitalised influenza.

Results

We identified 283 cases of ICD-coded presumptive viral myositis with seasonal peaks in cases from May to October (85% of cases); 69 were tested for influenza and 52 (78% tested) were positive. Given the strong seasonality, concurrent with the influenza season, we estimated the proportion of viral myositis attributable to IAM as 80% (95% CI 76-85) annually. Overall, 226 cases of IAM (confirmed and probable) were identified of which 21% (n=49) were laboratory confirmed and the remaining probable. IAM was significantly associated with being male (82%), aged 5-9 (73%), and having influenza B (86%). The majority of children presented with bilateral calf pain or refusal to weight bear; the mean creatinine kinase (CK) value was 3579u/L and no cases had renal impairment.

Conclusions

Childhood viral myositis shows strong seasonality concurrent with influenza season. In this retrospective, longitudinal analysis, we confirmed the typical clinical syndrome of IAM, its age predilection, benign course in most children and strong association with Influenza B. Early clinical recognition is important to avoid unnecessary treatment and testing.

Day 2	Workshop 2: Epidemiology
1200	**Rapidly mapping the spread of influenza**
Presenter	David Muscatello, University of New South Wales, Sydney, NSW

D. J. Muscatello1, R. N. Leong[1], R. M. Turner[1,2], A. T. Newall[1]

[1]University of New South Wales, [2]University of Otago

Introduction

Surveillance of influenza epidemics is a priority for risk assessment and pandemic preparedness. Mapping epidemics can be challenging because influenza infections are incompletely ascertained, ascertainment can vary spatially, and often a denominator is not available. Rapid, more refined geographic or spatial intelligence could facilitate better preparedness and response. Using the epidemic of influenza type A in 2016 in Australia, we demonstrated a simple adaptive method of automatically representing the spatial intensity and evolution of an influenza epidemic.

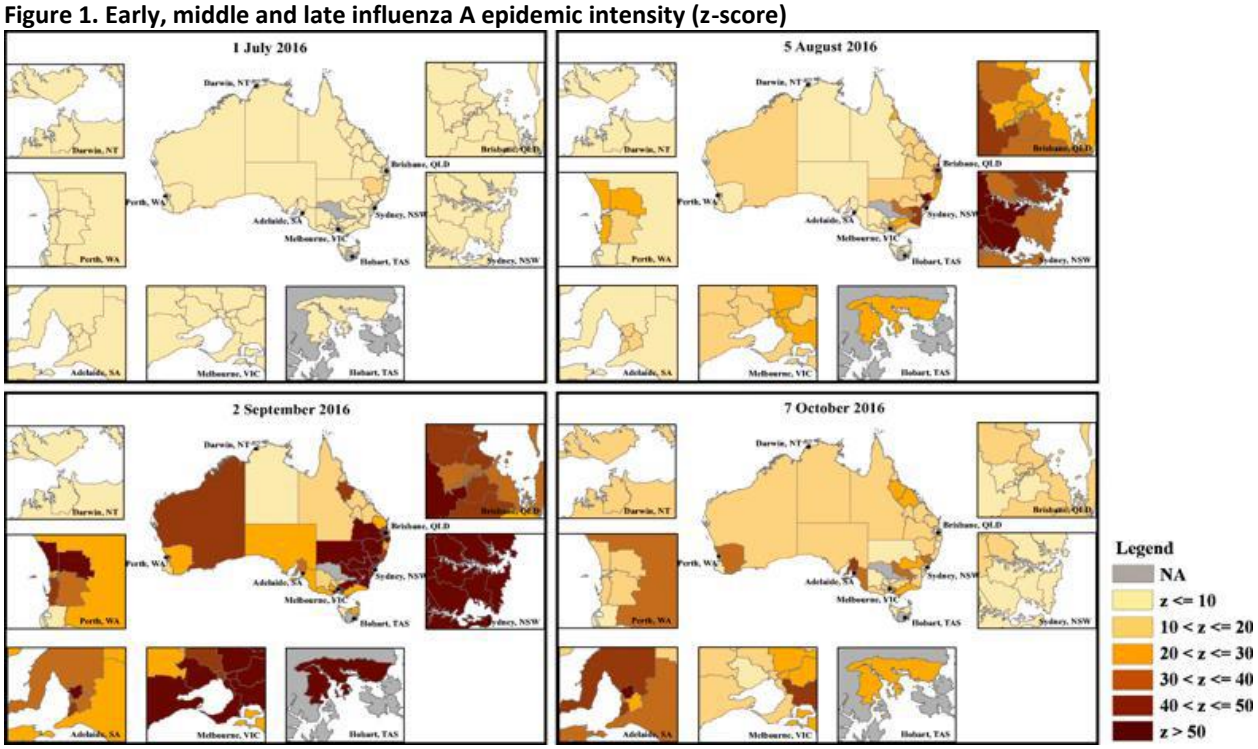
Methods

Weekly counts of persons with laboratory confirmed influenza type A infections in Australia in 2016 were analysed by 86 sub-state geographical areas. Weekly standardised epidemic intensity was represented by a z-score calculated using the standard deviation of below-median counts in the previous 52 weeks. A geographic information system was used to map the epidemic.

Results

There were 79,628 notifications of influenza A infections included. Of these, 79,218 (99.5%) were allocated to a geographical area. The maps indicated areas of elevated epidemic intensity across Australia by week and area that were consistent with the start, peak and decline of the epidemic when compared with counts aggregated at the state and territory level.

Figure 1. Early, middle and late influenza A epidemic intensity (z-score)



Conclusions

The methods could be automated to rapidly generate spatially varying epidemic intensity maps using a surveillance data stream. This could improve local level epidemic intelligence in a variety of settings and for other diseases. It may also increase our understanding of geographic epidemic dynamics.

Day 2	Workshop 2: Epidemiology
1215	**Insights into the burden of influenza in Indigenous Australians, 2019**
Presenter	Sandra Carlson, Hunter New England Local Health District, Newcastle, NSW

Sandra Carlson, Kristy Crooks, Craig Dalton, David N Durrheim

Background
Indigenous Australians have an increased risk of adverse outcomes due to influenza, however national influenza surveillance does not comprehensively monitor influenza activity and severity by Indigenous status. Flutracking, an Australian and New Zealand community level online influenza-like illness (ILI) surveillance system, has collected Indigenous status data since 2012. We provide insights into the community burden of influenza in Indigenous Australians, using Flutracking ILI data.

Methods
We report on 2019 vaccination coverage, cumulative ILI results, and health seeking behaviour, stratified by Indigenous status, and compared to prior years. All analyses were age-standardised to the relevant Indigenous and non-Indigenous Australia populations. Only data from epidemiological weeks 17 to 38 were included.

Results
Total ILI incidence for Indigenous participants up to end of September was at similar high levels for 2017 (35.3%), 2018 (37.8%) and 2019 (33.5%). However, for non-Indigenous participants, total ILI incidence levels were much lower in 2018 (26.5%) and 2019 (29.0%), as compared to 2017 (35.7%) (Figure). The percentage of Indigenous participants seeking health advice for influenza was higher than non-Indigenous participants most years from 2015 to 2019, with approximately half of Indigenous participants with ILI seeking health advice each year from 2017 to 2019. The percentage of Indigenous Flutracking participants reporting being vaccinated against influenza was consistently lower than non-Indigenous participants every year from 2012 to 2019.

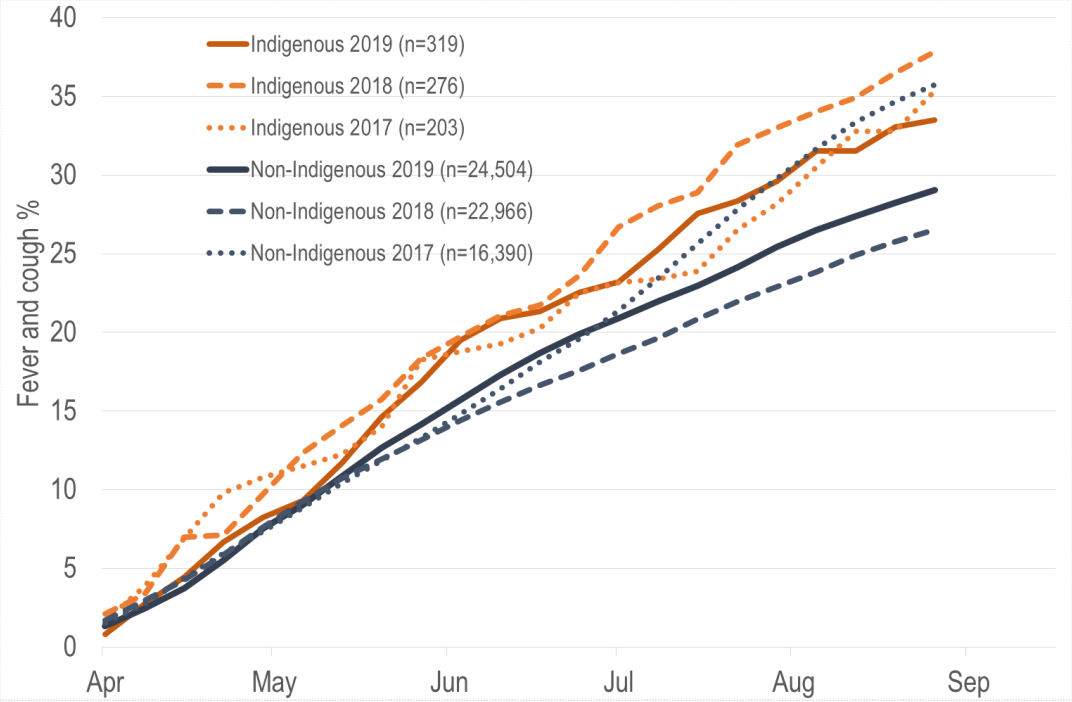


Figure. Cumulative incidence of ILI by Indigenous Status, Australia, April to September, 2017 - 2019, by week (age-standardised)

Conclusion
At the national level, 2018 was considered an exceptionally mild influenza year. However, Indigenous Flutracking participants experienced high ILI activity and health seeking behaviour, similar to 2017 and 2019.

Day 2	Workshop 2: Epidemiology
1230	**Drivers of a summer influenza epidemic – New South Wales, 2018-2019**
Presenter	Robin Gilmour, Health Protection NSW, Sydney, NSW

Celeste Marsh, Ross Andrews, Sean Tobin, Robin Gilmour (presenting), Vicky Sheppeard

Background

Influenza incidence in NSW typically follows a seasonal pattern with an epidemic peak in the cooler months and generally low transmission otherwise. An unusually high number of influenza cases (n=5469) were notified in NSW during the summer months of 2018/2019, almost double that of the previous summer. High activity continued in to March. We aimed to assess the extent to which this summer influenza epidemic in NSW was linked to overseas and/or domestic travel and the degree to which prior vaccination played a role using novel technology to distribute online surveys from within NSW's notifiable disease database.

Methods

We undertook a case-control study with a case defined as laboratory-confirmed influenza with illness-onset between 1 December 2018 and 21 March 2019. Pertussis notifications over the same time-frame served as the control group. Individuals with listed mobile phone numbers were eligible for participation; 2824 cases and 808 controls were invited to complete a short online survey. Logistic regression analyses were used to derive odds ratios for the various exposures and to control for potential confounders.

Results

Responses were obtained from 649 (23%) of cases and 260 (32%) of controls. Respondents were reasonably representative of their source populations but the peak age-group was older in the influenza cases (20-40 years) compared to the pertussis controls (5-19 years). From December to January, influenza cases were seven times more likely to have travelled overseas or come into contact with an ill overseas visitor in the week before falling ill compared to controls (AOR=7.09, p=<0.007). Cases most commonly reported travel to countries in SE Asia and northern Europe. The association of cases with overseas travel or contact with an overseas traveller was weaker in the latter months (1 Feb-21 Mar). Gender, contact with an ill local visitor and prior vaccination were not found to be associated with influenza.

Conclusions

Our study suggests that overseas travel was an important early driver of the 2018/2019 summer influenza epidemic in NSW, with local transmission continuing despite warmer conditions. The incidence of summer influenza appears to be increasing in Australia. Understanding the role that overseas travel plays in driving this increase is important to inform public health action and guide pre-travel influenza vaccination advice.

Day 2	Workshop 2: Epidemiology
1245	**Improving Influenza Vaccination in Children with Comorbidities: A Meta-Analysis**
Presenter	Christopher Blyth, University of Western Australia, Perth, WA

Christopher C Blyth[1,2,3,4], Allen C Cheng[5,6], Nigel W Crawford[7,8], Julia E Clark[9], Jim P Buttery[10,11], Helen S Marshall[12], Joshua R Francis[13], Jocelynne McRae[14,15] ,Tom Kotsimbos[16], Paul M Kelly[17], Kristine K Macartney[14,15,18] on behalf of the Paediatric Active Enhanced Disease Surveillance (PAEDS) and Influenza Complications Alert Network (FluCAN) Collaboration

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Background and aims: Jurisdictionally-based vaccination programs were established providing free quadrivalent influenza vaccine (QIV) for preschool Australian children in 2018. This was in addition to the National Immunisation Program (NIP)-funded QIV for Indigenous children and children with comorbid medical conditions. We assessed the impact of this policy on disease burden and vaccine coverage, as well as report on 2018 vaccine effectiveness.

Method: Subjects were recruited prospectively from twelve hospitals. Children aged ≤16 years hospitalised with acute respiratory infection (ARI) and influenza were considered cases. Hospitalised children with ARI testing negative for influenza were considered controls.

Results: A total of 458 children were hospitalised with influenza: 31.7% were <2 years, 5.0% were Indigenous, and 40.6% had medical comorbidities. Influenza A was detected in 90.6% of children (A/H1N1: 38.0%; A/H3N2: 3.1%; A/unsubtyped 48.6%). The median length of stay was 2 days (IQR: 1,3) and 8.1% were admitted to ICU. Oseltamivir use was infrequent (16.6%). Two in-hospital deaths occurred (0.45%). In test-negative-controls, 36.0% were vaccinated including 50.7% of children with comorbid conditions and 35.0% of Indigenous children. Vaccine effectiveness of QIV for preventing influenza hospitalisation was estimated at 78.8% (95%CI: 66.9; 86.4). 2019 data is currently being analysed.

Conclusion: Compared with 2017, a significant reduction in severe influenza was observed in 2018, possibly contributed to by improved vaccine coverage and high vaccine effectiveness. Despite introduction of jurisdictionally-funded preschool programs and NIP-funded vaccine for children with risk factors, improved coverage is required to ensure protection against paediatric influenza morbidity and mortality. Updated 2019 results will be presented.

Day 2	Plenary Session 5
1345	**Predicting risk in severe influenza infection**
Speaker	Benjamin Tang, The University of Sydney, NSW

Severe influenza infection has no effective treatment available. One of the key barriers to developing host-directed therapy is a lack of reliable prognostic factors needed to guide such therapy. Here, we use a network analysis approach to identify host factors associated with severe influenza and fatal outcome. In influenza patients with moderate-to-severe diseases, we uncover a complex landscape of immunological pathways, with the main changes occurring in pathways related to circulating neutrophils. Patients with severe disease display excessive neutrophil extracellular traps formation, neutrophil-inflammation and delayed apoptosis, all of which have been associated with fatal outcome in animal models. Excessive neutrophil activation correlates with worsening oxygenation impairment and predicted fatal outcome (AUROC 0.817–0.898). These findings provide new evidence that neutrophil-dominated host response is associated with poor outcomes. Measuring neutrophil-related changes may improve risk stratification and patient selection, a critical first step in developing host-directed immune therapy.

Day 2	Plenary Session 5
1415	**Evaluating influenza from a community perspective: insights on School absenteeism, vaccine effectiveness, and moleculr epidemiology**
Speaker	Jonathan Temte, University of Wisconsin, Madison, WI, USA

Jonathan L. Temte MD, PhD

Professor of Family Medicine and Community Health and Associate Dean for Public Health and Community Engagement, University of Wisconsin School of Medicine and Public Health, Madison, Wisconsin USA

Background
Schools are purported to be primary venues of influenza transmission and amplification with secondary spread to communities. We assessed K—12 student absenteeism monitoring as a means for early detection of influenza activity in the community. In the process, we have gained insights on influenza vaccine effectiveness and molecular epidemiology of influenza in a small community.

Methods
Since 2014, we have been conducting a prospective observational study of all-cause (a-TOT), illness-associated (a-I), and influenza-like illness-associated (a-ILI) absenteeism within the Oregon School District, Oregon, WI (OSD: enrollment = 3,900 students). Automated processes within OSD's electronic student information system facilitated absenteeism reporting. Students are screened for ILI, and visited at home, where pharyngeal specimens are collected for influenza RT-PCR and multipathogen testing. Surveillance of medically attended laboratory-confirmed influenza (MAI) occurs in five primary care clinics in and adjoining OSD as part of the Wisconsin Influenza Incidence Surveillance Project using the same laboratory testing.
Poisson general additive log linear regression models of daily counts of absenteeism and MAI from the initial 5 years were compared using correlation analysis. Influenza vaccine receipt status was verified using the Wisconsin Immunization Registry. Vaccine effectiveness (VE) was estimated using a test-negative design. Finally, whole genome sequencing (WGS) was performed for specimens from the 2017/2018 influenza season.

Results
From 1/06/2015—6/30/2019, influenza A and B viruses were detected in 301 and 98 of 1,717 visited students, respectively. Of MAI patients, 742 had influenza A and 253 had influenza B. Over the 5 years, a-I was significantly correlated with MAI in the community ($r=0.495$; $P<0.001$) with a 9-day lead-time, a-ILI was significantly correlated with MAI in the community ($r=0.519$; $P<0.001$) with no lead-time, and a-TOT performed poorly ($r=0.300$; $P<0.001$), following MAI by 23 days.
Overall VE was estimated at 7% [95% CI: -23 to 30]. Significant vaccine protection was found in only one season (2014/2015) for only influenza B Yamagata (81% [37 to 94]; $P=0.007$). VE was higher for medically attended children (30%; [-37 to 64]; $P=0.298$) than for those not seeking care (1%; [-36 to 28]; $P=0.938$), although this difference was not significant.
During 2017/2018, we observed three peaks in the incidence of A(H3N2), where each peak consisted of different viruses at the consensus level. The first peak consisted primarily of 3C2.A2 viruses that had largely identical hemagglutinin (HA) sequences. The second peak had more diversity, ranging from 0 to 12 single nucleotide variations in HA sequences. The third peak consisted primarily of 3C3.A viruses, indicating a shift in the consensus population. Interestingly, the peaks and changes in the influenza population occurred near breaks in the school year, suggesting the shifts in the influenza population may have been due to the introduction of influenza viruses from outside the community.

Conclusions
Surveillance using cause-specific absenteeism was feasible to implement in OSD and has performed well over a 5-year period marked by diverse presentations of seasonal influenza. Monitoring a-I and a-ILI can detect influenza outbreaks in the community, providing early warning in time for community mitigation efforts for seasonal and pandemic influenza. A community-based sampling frame leads to significantly lower estimates of influenza VE. Some of this difference may be due to care-seeking behavior. Finally, viruses isolated from school-aged children and their families in a small community represented wide diversity within a single influenza season and demonstrated successive waves of genetically distinct viruses.

Day 2	Plenary Session 5
1445	**Influenza in the community: An overview of the Seattle Flu Study**
Speaker	Janet Englund, Fred Hutch Cancer Center, Seattle, WA, USA

Janet A. Englund, Helen Y. Chu, MD, MPH, Michael Boeckh, MD, PhD, Michael Famulare, PhD, Barry R. Lutz, PhD, Deborah A. Nickerson, PhD, Mark Rieder, PhD, Lea M. Starita, PhD , Matthew Thompson, MD, MPH, DPhil, Jay Shendure, MD, PhD, Trevor Bedford, PhD

Co-investigators:
Amanda Adler, MS, Jesse Bloom, PhD, Elisabeth Brandstetter, MPH, Jeris Bosua, BA, Chris D. Frazar, MS, Peter D. Han, MS, James Hadfield, PhD, ShiChu Huang, PhD, Michael L. Jackson, PhD, MPH, Anahita Kiavand, MS, Louise E. Kimball, PhD, Enos Kline, BS, Kirsten Lacombe, RN, MSN, Jennifer Logue, B S, Victoria Lyon, MPH, Thomas R. Sibley, BA, Monica L. Zigman Suchsland, MPH, Caitlin Wolf, BS

Influenza epidemics and pandemics are important causes of morbidity and mortality worldwide. We initiated a community-wide surveillance study to gather data from community, clinics, and hospital sites to develop a model of how flu enters and spreads in an urban population. We recruited individuals with symptomatic respiratory disease in the Seattle metropolitan area of the USA during our winter 2018-2019 viral respiratory season. Individuals were enrolled from medical clinics, college campuses, urgent care centers, emergency departments, childcare facilities, homeless shelters, workplaces, and homes. Clinical and epidemiological information and mid-nasal swabs were collected from individuals of all ages with at least two acute symptoms that had started within the past week. We also collected residual nasal swabs and data from persons seeking care at four hospitals, including a large children's hospital. Swabs were tested using a multiplex molecular assay, and influenza whole genome sequencing was performed. Geospatial mapping and computational modeling platforms were used to describe the spread of influenza. We instituted a community-wide influenza surveillance system in Seattle and observed the introduction of influenza A/H3N2 into the community. Evidence of transmission of influenza into and within the Seattle area was demonstrated. Our approach provides the background for future community-based surveillance systems.

Note: This was presented in part at IDWeek 2019, Washington DC, by Helen Chu, MD.

Day 2	Joint session with the Australian Respiratory Virology Meeting
1550	**WHO program phase 1 results and plans for phase 2**
Speaker	Nigel Crawford, Murdoch Children's Research Institute, Melbourne, VIC

Since 2017, the RCH has been a clinical site for the WHO RSV surveillance network, based on the Global Influenza Surveillance and Response System (GISRS) platform https://www.who.int/influenza/rsv/en/, with the laboratory aspects co-ordinated by the WHO Collaborating Centre at Doherty, the University of Melbourne.

In this presentation we will review some of the clinical data over the past three seasons at RCH and place it in the international context. There are also emerging RSV vaccine and monoclonal antibody (mAb) candidates, so expanding surveillance in Australia and providing a health economics perspective are some of the important next steps.

Day 2	Joint session with the Australian Respiratory Virology Meeting
1610	**Respiratory syncytial virus-associated hospitalisations in the Australian population**
Speaker	Gemma Saravanos, NCIRS, Westmead, NSW

Saravanos G[1,2], Sheel M[1,2], Homaira, N[3,4], Quinn, H[1,2], Dey A[1,2], Brown E[1,2], Wang H[1], Britton, P[2,5,6], Macartney K[1,2,5], Wood N[1,2,5]

[1]National Centre for Immunisation Research and Surveillance, [2]University of Sydney, [3]University of New South Wales, [4]Sydney Children's Hospital Randwick, [5]The Children's Hospital at Westmead, [6]Mary Bashir Institute of Infectious Diseases and Biosecurity

Background

Respiratory Syncytial Virus (RSV) disease is a leading cause of hospitalisation in young children and severe disease in older adults is increasingly recognised. Vaccine candidates targeting disease prevention in these groups are in active development. In Australia there are no national data on RSV-associated hospitalisations across the age spectrum. We aimed to estimate rates of RSV-associated hospitalisation, and to identify groups at high-risk of severe RSV disease to inform future prevention strategies.

Methods

We carried out a retrospective review of National Hospital Morbidity Database data for all RSV-associated hospitalisations in Australia, 2006–2015. We described RSV-coded hospitalisation rates by age, sex, Indigenous status, jurisdiction, and seasonality (month and year); hospital length of stay; and in-hospital deaths. In addition, a clinical chart review was undertaken on RSV-test positive child deaths at a single paediatric hospital over a 20 year period (1998-2018).

Results

During 2006–2015, there were 63,814 hospitalisations with an RSV-specific principal diagnostic code; 60,551 (94.9%) were of children under 5 years of age. The hospitalisation rate for children under 5 years was 418 per 100,000 population; for children under 6 months of age it was 2,224 per 100,000 population; the highest rate was for infants aged 0–2 months (2,778 per 100,000 population). RSV-coded hospitalisation rates were higher for adults aged 65 or more than for people aged 5–64 years (incidence rate ratio [IRR], 6.6; 95% CI, 6.2–7.1), and were also higher for Indigenous Australians than other Australians (IRR, 3.3; 95% CI, 3.2–3.5). A total of 138 in-hospital deaths were recorded, including 82 of adults aged 65 years or more (59%) and 21 of children aged under 5 years (15%). During 1998-2018, there were 32 RSV-test positive child deaths at a single paediatric hospital all of whom had a significant comorbidity. Results from the clinical chart review of these cases will be presented.

Conclusions

Prevention strategies targeting infants, such as maternal or early infant vaccination, would likely have the greatest impact in reducing severe RSV disease. Further characterisation of RSV disease epidemiology, particularly in older adults and Indigenous Australians, is needed to inform future health care strategies.

Day 2	Joint session with the Australian Respiratory Virology Meeting
1630	**Influenza – a reality check!**
Speaker	Gary Grohmann, Environmental Pathogens, Canberra, ACT

G Grohmann[1] and J Herz[2]

[1]Environmental Pathogens, Canberra, [2]BioIntellect, Sydney

The current use and production of influenza vaccines for seasonal and pandemic purposes has hardly changed in 30 years; and while the use of quadrivalent and adjuvanted vaccines has emerged for some seasonal campaigns the world is still reliant on traditional egg- or cell-based vaccines for a pandemic threat. Recombinant vaccines have also been licensed but hardly used compared to the traditional vaccines, and many new technologies are in development. Progress in pandemic preparedness is also questionable: there is little in the way of stockpiles and no guarantee that stockpiles will be effective or sufficient; there are only a few laboratories that can produce candidate vaccine viruses (CVVs); determining the yield and safety of CVVs will take time; the use and production of reagents to standardise vaccine has not changed and takes some 3-4 months; some regulators may require a clinical trial before the vaccine is used adding significant time to the production/release process, moreover, there is still no regulatory harmonisation for pandemic influenza vaccines; the time to produce vaccine is still around 4-6 months and even then the volume available will be minimal compared to the needs of the world – it may take a full year for large volumes to be produced meaning that no vaccine will be available for the 1st wave of a pandemic; courses of antiviral drugs and antibiotics will not be available globally and may be compromised by resistant organisms; the effect of the WHO global action plan (GAP) to secure local production in Low and Middle income countries (LMICs) has been useful but adds little to the global capacity of influenza vaccine production unless regular seasonal vaccines are made and used; the WHO Pandemic Influenza Preparedness Framework (PIP) which is based in virus and benefit sharing to secure vaccine (and AVs) for LMICs has essentially failed as virus sharing is not optimal and there is no guarantee that vaccine and AVs will be available to LMICs at the start of a pandemic – indeed vaccine won't be available to any country for around 6 months, and then only small volumes; logistical issues are still a bottleneck in the process of vaccine production; finally, local immunisation policies for seasonal influenza vaccination have hardly changed in the last 10 years leaving vulnerable risk groups such as children unvaccinated in many countries as well as a lack of infrastructure to cope with implementation. The only conclusion is that we are not well prepared and have hardly advanced in practical terms for the production of pandemic vaccines, since 1968. In research and surveillance areas there has been a great deal of activity; novel vaccine approaches (non egg/cell) and delivery systems have shown promise; the global effort on universal vaccines is encouraging but unlikely to yield results for many years; WHOs Global Influenza Surveillance and Response System (GISRS) has expanded globally resulting in greater antigenic and genetic analyses for vaccine virus selection purposes which is also supported by GISAID and other public data bases. To secure enough pandemic vaccine globally all production platforms will be needed but the current capacity is only around 2 billion doses in the 1st year after a pandemic is declared; and this depends on yield, dose(s) required, antigen concentration and formulation, all of which is unknown. Vaccine manufacturing capacity is largely in Northern Hemisphere where existing supply obligations to North American and European authorities are unlikely to leave sufficient capacity for the rest of the world. Moreover, there may still be some need for seasonal vaccine production and use in some areas and this needs to be carefully considered. The current traditional approaches are conservative and will produce some vaccine within 12 months of a pandemic but new thinking is needed and a fresh approach if the world is to be ready for the next pandemic with enough vaccine for all.

Day 2	Joint session with the Australian Respiratory Virology Meeting
1650	**RSV in the elderly**
Speaker	Robert Booy, NCIRS, Westmead, NSW

2019 Delegates

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