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The Australian Paediatric Surveillance Unit continues to make a significant contribution to the national surveillance of rare childhood diseases to improve the quality of life for Australian children and their families. Over the past 16 years, data collected through the APSU has provided an essential resource to inform national paediatric clinical practice, health service planning, education, the development of national public health policy and screening programs. The long-time success of the APSU has been achieved through its collaborative approach that enables it to collect information each month from about 1,300 paediatricians and other child health physicians, representing 92 per cent of specialist paediatric physicians across Australia.

The diseases that are monitored are chosen for their public health importance and impact on health resources, clinicians and families. They include genetic disorders, child mental health problems, rare injuries, rare infections and vaccine preventable diseases. For many of these rare childhood conditions, the APSU is the only national mechanism for data collection.

In 2007 and 2008, the APSU provided the Australian Government with important surveillance data on severe complications associated with influenza in children, to assist in measuring the severity and epidemiological determinants and to inform the public health response. I congratulate the APSU for its continued success in advancing this important national surveillance system and evidence–based resource for rare childhood diseases.
Dr Jenny Proimos  
President, Paediatrics & Child Health Division, RACP

The Paediatrics & Child Health Division of the Royal Australasian College of Physicians is proud to have an ongoing association with the Australian Paediatric Surveillance Unit. After 2008’s APSU 15th Anniversary Publication 1993-2007, celebrating its 15 years of surveillance, the APSU has continued to accurately collect important surveillance data on rare childhood diseases. These data are used widely by many child and adolescent health professionals to inform their efforts in optimising the quality of life for Australian children, young people and their families.

The APSU continues to be well supported by the majority of Australian paediatricians, with over 90% of them contributing monthly to the APSU’s bank of information about rare diseases. The research studies that have resulted in the past year have provided invaluable information for health care, policy and advocacy, as well as a wonderful mechanism for teaching research to paediatricians and College trainees.

The significant reputation the APSU has built was highlighted at the College’s Congress in 2008, with many researchers from international surveillance units attending to contribute to the APSU’s 15th anniversary and celebrate its many achievements. The Division is very grateful for the regular valuable contribution the APSU makes to the College Congress each year, enabling paediatricians to benefit more directly from the APSU’s surveillance findings.

The road ahead for the APSU will continue to be bright, as the collection of information on numerous uncommon conditions continues, and its reputation and knowledge can be leveraged to help the broader paediatric and child health community drive the establishment of suitable health care practices, resources and supports for children, young people and families affected by these uncommon conditions.

The Paediatrics & Child Health Division of the College congratulates the Director Associate Professor Elizabeth Elliott, the Deputy Director Yvonne Zurynski and all the staff and researchers for their outstanding achievements this year.
Professor Elizabeth Elliott, AM  
Director, Australian Paediatric Surveillance Unit

As indicated by the 2007-8 report, The Australian Paediatric Surveillance Unit continues to thrive as an international leader and innovator in rare disease research.

Several highlight are documented. First is the APSU’s role in establishing and managing – with the National Centre for Immunisation Research and Surveillance – a novel inpatient system for identifying hospitalised children with rare conditions. PAEDS (Paediatric Active Enhanced Disease Surveillance), which is currently running in four tertiary hospitals in four states, complements APSU surveillance and is particularly valuable when detailed or timely data, biological specimens, or follow-up are required.

The second highlight is the leadership role APSU took in convening a National Rare Diseases Working Group and obtaining competitive funds to work towards developing a National Plan for Rare Diseases in Australia. Thirdly, the APSU undertook its second evaluation in 2007. Critical evaluation of our activities is a high priority. We particularly value feedback from paediatricians who so generously give their time and skills to document cases each month. Their opinions of the APSU and suggestions for improvement can be read in recent publications in the Journal of Paediatrics and Child Health. Finally, APSU participated in the 4th Biannual meeting of the International Network of Paediatric Surveillance Units in Germany in 2008.

The extensive publication list indicates APSU’s high, ongoing productivity; the list of new studies indicated continued interest in the unit by researchers; and the many media reports, particularly around Rare Diseases Day, indicate APSU’s willingness to engage with and inform the wider community.

None of APSU’s work could be achieved without dedicated staff, particularly Assistant Director Yvonne Zurynski; the study Investigators; our students; and paediatricians Australia-wide who contribute data each month. Infrastructure support from the NHMRC and the Commonwealth Department of Health and Ageing is crucial to maintain our activities, as is essential support from the Royal Australasian College of Physicians, the University of Sydney, the Children’s Hospital at Westmead where we are housed, and others.

Professor Carol Bower  
Board Chair, Australian Paediatric Surveillance Unit

Whilst not surprising, it is reassuring to see, from evaluations of APSU conducted in 2007 that APSU continues to be an effective surveillance system (meeting criteria established by the Centers for Disease Control and Prevention in the US) and that it continues to be valued by paediatricians in Australia. Not content, however, with this excellent report card, the APSU has added two new methods to its tried and tested armamentarium. The first, a system for delivering rapid response surveillance proved extremely valuable in investigating severe complications following influenza infection in 2007 and again in 2008, and also to monitor the H1N1 influenza epidemic. The second is the augmentation of surveillance in four tertiary hospitals (PAEDS), which has enhanced the information available on conditions such as acute flaccid paralysis and intussusception, by the addition of more detailed clinical data and timely biological specimens. APSU staff are also to be commended on taking a lead role in raising awareness of rare diseases in Australia and convening a National Working Party for rare diseases – allowing Australia to emulate similar initiatives in North America and Europe and which we hope will lead to better provisions for the thousands of children with individually rare diseases and their families.

Congratulations to Professor Elliott, the APSU staff, the contributing paediatricians and APSU researchers, for the excellence of the surveillance system and the uses to which their talents and the data are put.
Patron

Fiona Stanley AC
Professor, School of Paediatrics and Child Health, The University of Western Australia. Director, Telethon Institute for Child Health Research.

Board

Carol Bower* (Chair)
Clinical Professor, Centre for Child Health Research; Head of Epidemiology, Division of Population Sciences, Telethon Institute for Child Health Research, The University of Western Australia. Head and Medical Specialist, Western Australian Birth Defects Registry, Women and Newborn Health Services.

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Senior Lecturer, Department of Preventive and Social Medicine, The University of Otago, New Zealand. Co-Director, New Zealand Paediatric Surveillance Unit.

David Isaacs
Clinical Professor, Discipline of Paediatrics and Child Health, The University of Sydney. Senior Staff Specialist, Department of Immunology & Infectious Diseases, The Children's Hospital at Westmead.

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Jenny Proimos*
President, Paediatrics and Child Health Division, Royal Australasian College of Physicians.

John Ziegler
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Yvonne Zurynski*
Deputy Director, Australian Paediatric Surveillance Unit. Senior Lecturer (Research Only), Discipline of Paediatrics and Child Health, The University of Sydney.

* Board and Scientific Review Panel members

APSU Staff 2007–2008

Professor Elizabeth Elliott, Director (Jan 1993 – )
Dr Yvonne Zurynski, Deputy Director (Feb 2005 – )
Dr Elizabeth Peardon, Medical Education Officer and PhD Student (Jan 2006 – )
Dr Katie Reeve, Research Officer (Oct 2006 – Jun 2008)
Ms Emily Fremantle, Research Assistant (Dec 2006 – Apr 2007)
Ms Nicole McKay, Data Manager (Apr 2006 – )
Ms Karen Pattinson, Office Co-ordinator (Aug 2006 – )
Ms Ingrid Charters, Administration Officer (Oct 2004 – )
Ms Sarah Srikanthan, Publications Officer (Aug 2007 – )

Dr Deepika Mahajan, Research Fellow (Jul 2007– Oct 2008)
Dr David Lester-Smith, Research Officer (Sept 2007–Jan 2009)
Ms Margy Pym, Coordinator, Paediatric Active Enahced Diseases Surveillance System (Jan 2007–June 2009)
Dr Hua Chang, Student, Advanced Research Project for the Fellowship of the Australian College of Emergency Medicine (2007)
Dr Nicola Benwell, Research Officer and Honours Student, Postgraduate Medical Program, The University of Sydney (May 2007–)
Dr Suwen He, Research Officer and Masters in Public Health Student, The University of Sydney (Jul 2007-Oct 2008)
Institutions Collaborating with the APSU 1993-2008

National Organisations
- Australian Maternity Outcomes Surveillance System (AMOSS)
- Association for Genetic Support Australasia (AGSA)
- Association for the Welfare of Children in Hospitals (AWCH)
- Australia and New Zealand Paediatric Nephrology Association
- Australian CHARGE Association
- Australian Enteric Pathogens Surveillance Scheme
- Australian Polio Expert Committee
- Australasian Paediatric Endocrine Group
- Australian Institute of Health and Welfare
- Australian National University Medical School
- Australian Research Alliance for Children and Youth (ARACY)
- Australian Society of Clinical Immunology and Allergy
- Intergovernmental Committee on Drugs: Working party on FASD
- Commonwealth Department of Health and Ageing
- National Births Anomalies Steering Committee
- National Centre in HIV Epidemiology and Clinical Research
- National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases
- National Heart Foundation of Australia
- National Notifiable Diseases Surveillance System
- National Perinatal Statistics Unit
- National Polio Reference Laboratory
- OzFoodNet: Australian Enhanced Foodborne Disease Surveillance
- WHO Regional Commission for the certification of poliomyelitis eradication
- Rett Syndrome Association of Australia & AussieRett
- Royal Australasian College of General Practitioners (RACGP)
- Royal Australasian College of Physicians, Paediatrics and Child Health Division (RACP)
- SMILE Foundation
- Steve Waugh Foundation

New South Wales
- Anti-Discrimination Commission of New South Wales
- Child and Adolescent Mental Health Statewide Network (CAMSHNET)
- Centre for Kidney Research
- Centre for Mental Health, New South Wales Health
- Gastroenterology and Liver Unit, Prince of Wales Hospital
- Institute for Neuromuscular Research
- Hunter Genetics
- Liverpool Health Service
- Macleay Hastings Health Service
- Millennium Institute of Health Research
- NSW Birth Defects Register
- NSW Centre for Perinatal Health Services Research
- NSW Commission for Children
- NSW Department of Health
- NSW Hospitals: Bankstown, John Hunter, Nepean, Royal Prince Alfred, Royal North Shore, Sydney Children's, Westmead, The Children's Hospital at Westmead
- Paediatric HIV Services Unit, Sydney Children's Hospital
- Prince of Wales Medical Research Institute
- The University of Sydney
- The University of New South Wales
- South Eastern Sydney and Illawarra Area Health Service
- South Eastern Area Laboratory Services
- Sydney South West Area Health Service

Northern Territory
- NT Hospitals: Alice Springs, Royal Darwin
- The Menzies School of Public Health, Darwin
- The Menzies School of Health Research

Victoria
- Australian Mycobacterium Reference Laboratory Network
- Centre for Adolescent Health, Royal Children's Hospital
- Victorian Infectious Diseases Reference Laboratory
- Monash Medical Centre
- Murdoch Children's Research Institute
- Public Health Group, Department of Human Services
- The University of Melbourne
- Victorian Hospitals: Royal Women's, Royal Children's

Queensland
- Queensland Hospitals: Mater Children's, Princess Alexandra, Royal Children's
- Queensland University of Technology
- Tropical Public Health Unit
- The University of Queensland

South Australia
- Finders Medical Centre
- Institute of Medical Veterinary Science
- Mycobacterium Reference Laboratory, Adelaide
- South Australian Health Commission
- Women's and Children's Hospital, Adelaide

Western Australia
- Curtin University
- Disability Services Commission
- Pathcentre, Queen Elizabeth II Medical Centre
- Telethon Institute for Child Health Research
- WA Hospitals: King Edward Memorial, Princess Margaret Hospital for Children, Royal Perth

Tasmania
- Royal Hobart Hospital

International Organisations
- Canadian Immunisation Monitoring Program Active
- European Organisation for Rare Diseases, Paris, France
- Great Ormond St Hospital, London, UK
- Hospital for Sick Children, Toronto, Canada
- New Zealand Organisation for Rare Diseases
- Oakland Children's Hospital, USA
- Orphanet, Paris, France
- Rare Diseases, London, UK
- UK Obstetric Surveillance System
- Westkids, Auckland, NZ

International Network of Paediatric Surveillance Units (INoPSU)
- British Paediatric Surveillance Unit
- Canadian Paediatric Surveillance Programme
- German Paediatric Surveillance Unit
- Greece Paediatric Surveillance Unit
- Latvian Paediatric Association
- Malaysian Paediatric Surveillance Unit
- Netherlands Paediatric Surveillance Unit
- New Zealand Paediatric Surveillance Unit
- Portuguese Paediatric Surveillance Unit
- Swiss Paediatric Surveillance Unit
- Republic of Ireland Paediatric Surveillance Unit
- Welsh Paediatric Surveillance Unit
Acknowledgements

Funding and Sponsorships 2007 – 2008

The National Health and Medical Research Council of Australia supports the APSU with an Enabling Grant entitled “Australian Paediatric Surveillance Unit: A collaborative network for child health research” (Grant No. 402784; Principal Investigators: Elliott EJ, Bower C, Kaldor J, Booy R, Sullivan S) and a Practitioner Fellowship: Elliott EJ (Grant No. 457084).

The Australian Government Department of Health and Ageing, provides infrastructure support for APSU activities that relate to communicable and vaccine-preventable surveillance and for the Paediatric Active Enhanced Diseases system.

The Sydney Medical School, University of Sydney supports the APSU financially. The APSU Director and Assistant Director are members of the Discipline of Paediatrics and Child Health, Faculty of Medicine.

The APSU is a Unit of the Division of Paediatrics and Child Health of the RACP. The RACP provides support for APSU for special projects including production of the bi-annual report.

The Children’s Hospital at Westmead provides office space, IT services and research infrastructure support for the APSU.

Additional financial supporters for individual surveillance studies include:

- Congenital cytomegalovirus infection: Virology Division, Dept of Microbiology, South Eastern Area Laboratory Service, Sydney Children’s Hospital
- HIV/AIDS and perinatal exposure to HIV: National Centre in HIV Epidemiology and Clinical Research
- Hyperinsulinaemic Hypoglycaemia of Infancy: Department of Endocrinology and Diabetes, Mater Children’s Hospital
- Neonatal/Infant Group B Streptococcal Sepsis: Centre for Infectious Diseases and Microbiology, Westmead Millenium Institute
- Neonatal herpes simplex virus infection: Department of Immunology and Infectious Diseases, The Children’s Hospital at Westmead, Herpes Simplex Virus Research Unit
- Neuromuscular Disorders of Childhood: Department of Neurology and Neurosurgery, Royal Children’s Hospital, Melbourne
- Rett syndrome: The Telethon Institute for Child Health Research, USA National Institutes of Health, Rett Syndrome Association of Australia
- Vitamin D deficiency rickets: Roche Products Pty. Ltd, Australia
- Acute intussusception: CSL Biotherapies, GlaxoSmithKline (GSK).
- Mount Majura Wines continues to generously sponsor the APSU wine prize draw.
The Australian Paediatric Surveillance Unit (APSU) is a national research resource, established in 1993 to facilitate active surveillance of uncommon childhood diseases, complications of common diseases or adverse effects of treatment. Conditions are chosen for their public health importance and impact on health resources. To date, a range of infectious, vaccine-preventable, mental health, congenital and genetic conditions, and injuries have been studied (Table 1 and Table 8). For many childhood conditions, the APSU provides the only mechanism for national data collection.

To the end of 2008, the APSU was used by over 200 individual researchers to run 45 surveillance studies. Epidemiological and clinical data collected through the APSU are of direct relevance to clinical and public health policy and resource allocation, and thus impact on the health and welfare of Australian children.¹ The APSU has also been influential in the development of international units.

The APSU is a unit of the Division of Paediatrics and Child Health, Royal Australasian College of Physicians (RACP). It is based at The Children’s Hospital at Westmead. The APSU Board oversees the management and policy directions of the unit, while the APSU Scientific Review Panel (SRP) determines which studies are suitable to run through the APSU mechanism and provides advice on surveillance methods. The activities of the APSU are funded in part by the Australian Government Department of Health and Ageing (Communicable Disease and Health Risk Policy Section), by the Sydney Medical School, The University of Sydney, and by The National Health and Medical Research Council of Australia (NHMRC), and other competitive research funding.

Contributors to the APSU
Contributors to the APSU are clinicians working in paediatrics and child health in Australia. Most (about 80%) are general paediatricians with or without a special interest. In addition, 6% are neonatologists, 4% are surgeons, 3% are geneticists and 2% are emergency physicians. Clinicians are identified through the Division of Paediatrics and Child Health of the RACP, the Australasian Association of Paediatric Surgeons and other paediatric special interest groups. In 2008 an estimated 91% of all paediatricians listed on the RACP list of Fellows and in active clinical practice in Australia participated in APSU surveillance.

Aims of the APSU
1. To provide a national active surveillance mechanism that can be used to:
   • estimate the incidence, epidemiology, clinical features, current management and short-term outcomes of rare childhood conditions in Australia
   • respond to epidemiological emergencies such as outbreaks, emerging or imported diseases.
2. To initiate and facilitate collaborative, national, child health research consistent with national health priorities, including ‘a healthy start in life’ and to fill knowledge gaps.
3. To produce and disseminate evidence that will support development of:
   • the most effective educational resources and clinical guidelines for clinicians
   • the most appropriate prevention strategies and community awareness campaigns.

Operation of the APSU
Individuals or organisations may apply to study a rare condition through the APSU. Applications undergo a process of peer review by the SRP before being listed on the monthly report card. All studies must have the potential to contribute significant new knowledge about rare childhood conditions and to influence policy, clinical practice or resource allocation.

Conditions are usually studied for between one to three years, although provision for ongoing surveillance may be granted for diseases of particular public health significance or with very low incidence (e.g. HIV/AIDS, congenital rubella).

Each month all clinicians participating in APSU surveillance are sent a report card listing up to 17 different conditions under surveillance and asked to return the report card whether they have seen a case or not. All positive reports of cases are then followed up by a brief questionnaire requesting de-identified information about the child’s demographics, details of diagnosis, management and short-term outcome. For more detail on APSU methodology please see the APSU website: www.apsu.org.au.

The APSU celebrated 15 years of surveillance in 2008 with the publication of *APSU: Celebrating 15 years of Surveillance 1993-2007* (ISBN: 978-0-646-49063-2) and by presenting two dedicated sessions at the Annual Scientific Meeting of the Royal Australasian College of Physicians, including a keynote address by Professor Elizabeth Elliott (page 54; ref 26).

In June 2008, Professor Elizabeth Elliott was awarded a Member of the Order of Australia (AM) for “service to paediatrics and child health as an academic, researcher and educator and through establishing the Australian Paediatric Surveillance Unit”.

In 2007 the APSU demonstrated the feasibility of using its surveillance system to respond at short notice to monitor epidemiological emergencies by responding to a call from the Department of Health and Ageing for rapid response surveillance for severe complications of influenza. This followed reports of several child deaths during the influenza season. This surveillance was repeated during the 2008 influenza season and is ongoing during the current influenza pandemic caused by influenza A H1N1 09.

In November 2008 APSU convened a National Rare Diseases Working Group comprised of researchers, clinicians and parent support organisations dedicated to developing a coordinated National Plan for Rare Diseases. APSU joined the global effort to raise awareness of rare diseases on International Rare Diseases Day 28th February 2008 (page 48).

APSU has also enhanced the national surveillance effort by contributing to the development and management of two new and innovative surveillance systems:

- **PAEDS**: The Paediatric Active Enhanced Disease Surveillance system was piloted over 18 months from August 2007 in four tertiary paediatric hospitals (Children’s Hospital at Westmead NSW, Royal Children’s Hospital in Victoria, Princess Margaret Hospital in Western Australia, Women’s and Children’s Hospital in South Australia). This project was supported by the Department of Health and Ageing and the initial funding agreement has been extended to June 2010 (page 47).

- **AMOSS**: The Australian Maternity Outcomes Surveillance System (AMOSS) aims to provide detailed, systematically collected data on serious but rare outcomes related to birth and pregnancy. AMOSS is based on the APSU surveillance mechanism and informed by a similar surveillance system operating in the UK, the UK Obstetric Surveillance System (UKOSS).

APSU continues to strengthen its links with policy makers and governments, by informing public health policy and clinical practice. For example, APSU has monitored the incidence of neonatal, congenital and severe complications of varicella after the introduction of the varicella vaccine; data collected on Fetal Alcohol Syndrome informed the new NH&MRC Australian Alcohol Guidelines, and data collected on seatbelt related injuries informed the 7th Amendment to the Australian Road Rules.

APSU has addressed surveillance gaps among Indigenous children and refugees by mounting surveillance for conditions particularly relevant to these groups eg. acute rheumatic fever in Indigenous children (page 20) and Vitamin D deficiency rickets in refugee children (page 35).

APSU is collaborating with the Telethon Institute for Child Health Research in the Alcohol in Pregnancy Project, a highly productive program of research, education, advocacy and policy development related to Fetal Alcohol Spectrum Disorders (FASD). Resources that were developed as part of the project are available for downloading or ordering at [www.ichr.uwa.edu.au/alcoholandpregnancy](http://www.ichr.uwa.edu.au/alcoholandpregnancy).

In 1997 APSU was the first unit of its kind in the world to undergo systematic evaluation according to the Centers for Disease Control and Prevention (CDC) criteria. Our second evaluation in 2007 showed that APSU fulfilled most of its objectives and met CDC criteria for an effective surveillance system: usefulness, simplicity, flexibility, data quality, acceptability, sensitivity, representativeness and timeliness. In addition, a survey of paediatricians who voluntarily report to the APSU showed that most (95%) believe that the work of the APSU is valuable, specifically, for generating knowledge (81%) guiding clinical practice (70%), informing future policy (70%), evaluating current policy (68%), identifying research priorities (78%) and facilitating collaborative research (75%) (page 49).
## Conditions Studied 2007–2008: Key Findings

<table>
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<tr>
<th>Conditions Under Surveillance</th>
<th>Dates of Study</th>
<th>Key findings, implications and publications</th>
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<tbody>
<tr>
<td>Infectious/vaccine preventable conditions</td>
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<tr>
<td>Acute flaccid paralysis (AFP)</td>
<td>Mar 1995–</td>
<td>In 2007–2008, Australia continued to maintain its polio-free status. As in previous years, the most common causes of non-polio AFP were Guillain-Barre syndrome and transverse myelitis. The availability of data through the Paediatric Active Enhanced Disease Surveillance (PAEDS) scheme enabled a greater number of cases to be classified by the Polio Expert Committee in 2008. (1)</td>
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<td>Acute rheumatic fever (ARF)</td>
<td>Oct 2007–</td>
<td>Children with ARF were reported in all states of Australia except the ACT and TAS, with NT and QLD reporting the highest incidence. Only 5% of children had a previous episode of ARF prior to notification to the study. The majority of children with ARF were Aboriginal and Torres Strait Islanders (ATSI) who lived in small towns in remote areas and 43% were living in crowded conditions.</td>
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<tr>
<td>Congenital cytomegalovirus (cCMV) infection</td>
<td>Jan 1999–</td>
<td>Diagnosis of congenital CMV after the newborn period is increasing due to use of molecular techniques to test Newborn Screen Cards and stored cord blood samples. The release of guidelines for use of intravenous immunoglobulin (IVIG) in pregnant women by the Australian Red Cross Blood Transfusion Services has provided a strategy for clinicians treating infected women. A follow-up study of a group of children with cCMV reported to the APSU has been undertaken by the cCMV research group and is pending publication. (2)</td>
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<td>Neonatal and infant Group B streptococcus (GBS) sepsis</td>
<td>Jan 2005–Jun 2008</td>
<td>This study confirmed a significant decrease in the incidence of neonatal GBS infection since the 1980s, when widespread use of intrapartum antibiotics began. This is despite inconsistent antenatal screening and use of intrapartum antibiotics, even for women identified as GBS carriers or with clinical risk factors. The origins of late onset disease remain obscure but the epidemiology is significantly different from that of early onset disease and unaffected by current preventative measures.</td>
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<td>Hepatitis C virus (HCV) infection</td>
<td>Jan 2003–Dec 2007</td>
<td>Perinatal transmission is the main source of HCV infection in Australian children. Infants were born mainly to mothers who used IV drugs, had invasive procedures overseas or had tattoos. Most HCV infected children were clinically asymptomatic with mildly elevated liver function tests at diagnosis; however, HCV induced chronic liver disease and liver failure have been reported among older children. Given that 1-2% of Australian women of childbearing age are infected with HCV, the reported rate of HCV infection is lower than predicted. This may be due to the lack of a consistent approach to identifying children with infection. This study identifies a need for education in regard to investigation, diagnosis and management of HCV infection in children. (3)</td>
</tr>
<tr>
<td>Neonatal herpes simplex virus infection (HSV)</td>
<td>Jan 1997–</td>
<td>Neonatal HSV infection most commonly presents as disease localised to the skin, eye or mouth, with low mortality if treated promptly with antiviral therapy. However, nearly a third of infants present with disseminated multi-organ infection, which carries a high mortality. Presenting features of disseminated infection are often non specific, and cutaneous features may be absent. Therefore this form of neonatal HSV infection is frequently diagnosed post-mortem. Intrauterine infection with HSV is rare and presents with a triad of malformations of the eye, brain (microcephaly or hydrancephaly) and skin (scarring and/or vesicles). (4)</td>
</tr>
<tr>
<td>HIV/AIDS, perinatal exposure to HIV</td>
<td>May 1993–</td>
<td>National surveillance indicates that perinatal exposure to HIV and mother-to-child HIV transmission remains rare among children in Australia. The risk of mother-to-child transmission is minimised among women whose HIV infection is diagnosed antenatally and who make use of interventions: transmission continues amongst children born to women whose HIV infection was diagnosed postnatally. (5) The majority of women acquire HIV through heterosexual contact with partners from high prevalence countries. The APSU was the only source of information in 90% of cases reported in the last 6 years.</td>
</tr>
<tr>
<td>Conditions Under Surveillance</td>
<td>Dates of Study</td>
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<td>Severe complications of influenza</td>
<td>Sep 2007, Jul 2008–Sep 2008</td>
<td>This study confirmed the feasibility of using the APSU for rapid response surveillance of severe complications of seasonal influenza. Surveillance was reactivated during the 2009 H1N1 influenza pandemic. Influenza A was the predominant strain among children reported in 2007 and influenza B in 2008. The majority of complications occurred in NSW, NT, and QLD and Aboriginal Torres Straight Island (ATSI) children were over-represented (29%). CXR-proven pneumonia was the most common complication. Few children were vaccinated or treated with influenza specific antivirals, despite being eligible. Universal influenza vaccination for children from 6 months of age and prompt treatment with oseltamivir may prevent severe complications and reduce the burden on health services and families. (6)</td>
</tr>
<tr>
<td>Intussusception (IS)</td>
<td>May 2007–</td>
<td>After 18 months of data collection, approximately one-fifth of all children reported to APSU with IS had received rotavirus vaccine and 9% had developed IS within two weeks of vaccination. The number of cases of IS has been low suggesting under-reporting, and vaccination details are often incomplete. Additional data collected through the PAEDS system will supplement APSU data and together may inform any potential temporal association between IS and rotavirus vaccination. As there is under-reporting of IS cases and limited vaccination data, these data justify the need for ongoing IS surveillance to further explore any possible relationship between IS cases, the age at vaccination, dose and type of vaccine. (7)</td>
</tr>
<tr>
<td>Non tuberculous mycobacterial infection (NTMI)</td>
<td>July 2004–Sep 2007</td>
<td>The median age of children with NMTI was 2.9 years and most children had no pre-disposing condition. Lymphadenitis was the most frequent presentation and Mycobacterium avium-intracellulare was the most frequently isolated organism. There was significant variation in surgical and medical therapies administered by Australian doctors, with a higher rate of treatment success following complete surgical excision and a lower rate of success following Lentiflavum infection. Despite therapy, recurrence occurred in 23% of children. (8)</td>
</tr>
<tr>
<td>Congenital rubella</td>
<td>May 1993–</td>
<td>Notifications have decreased significantly in recent years and no cases were confirmed in 2007. In 2008 there were three notifications, of which one was confirmed as a case. This was a child born to an immigrant woman from India whose vaccination history could not be confirmed. Serological testing was not performed. The risk of congenital rubella remains, particularly among immigrant women from countries with poorly developed vaccination programs, so continued surveillance is needed. (9)</td>
</tr>
<tr>
<td>Congenital varicella</td>
<td>May 2006–</td>
<td>The rate of congenital varicella remains low with one infant reported in 2007 and none in 2008, suggesting the national varicella immunisation program has had an impact on the incidence of congenital varicella.</td>
</tr>
<tr>
<td>Neonatal varicella</td>
<td>May 2006–</td>
<td>There has been a marked decrease in infants confirmed with neonatal varicella since 2006 with no confirmed cases in 2008. Family members are the most common infecting contact and the majority are unvaccinated. Approximately one third of affected children received treatment with zoster immunoglobulin and/or acyclovir. There is a need for well disseminated guidelines on the management of perinatal exposure to varicella. (10,11)</td>
</tr>
<tr>
<td>Severe complications of varicella</td>
<td>May 2006–</td>
<td>Despite a marked decrease in the number of severe complications of varicella in 2007 compared to 2006, there was an increase in cases in spring and summer, 2008. The median age at diagnosis was 4 years. Approximately half the children presented with secondary bacterial infections including bacteraemia, septic arthritis, osteomyelitis and abscesses, and a third presented with neurological complications. Most children admitted to hospital with severe complications of varicella were previously well children with no risk factors for severe complications. Ninety-six percent of children were unimmunised and other children including siblings were the identified infecting contacts. (12)</td>
</tr>
</tbody>
</table>
## Conditions Studied 2007–2008: Key Findings

<table>
<thead>
<tr>
<th>Conditions Under Surveillance</th>
<th>Dates of Study</th>
<th>Key findings, implications and publications</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Congenital/genetic disorders</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hyperinsulinaemic hypoglycaemia of infancy (HI)</td>
<td>Jan 2005–Mar 2007</td>
<td>A definitive cause for HI was specified in 7% of infants recruited to this study, highlighting the difficulties in diagnosing this condition and the need for increased awareness and improved diagnostic services. The majority of infants with HI were small for gestational age and did not have a genetic explanation for their hyperinsulinism. A high proportion was discharged on diazoxide which in the long term may have significant side effects. One quarter of infants had a seizure at presentation, highlighting the need for clinician awareness of the possibility of complications of hypoglycaemia. (13)</td>
</tr>
<tr>
<td>Neuromuscular disorders of childhood (NMD)</td>
<td>Jan 2007–</td>
<td>Children with neuromuscular disorders commonly presented with delayed motor development. The majority were diagnosed at 2–5 years of age using targeted genetic testing. Muscular dystrophies, followed by congenital myopathies, inherited neuropathies and spinal muscular atrophy were the key diagnosis. A report and recommendation paper for a National NMD Register is being developed and Australasia is now represented on two major international clinical trial networks for paediatric neuromuscular disorders. (14)</td>
</tr>
<tr>
<td>Rett syndrome</td>
<td>May 1993–Apr 1995; Jan 2000–</td>
<td>The APSU continues to contribute new cases of Rett syndrome to the Rett syndrome cohort, which now consists of 355 cases. Differences in enteral nutrition support, gross motor skills, fracture incidence, and an initial diagnosis of autism, are related to the type of mutation carried. The APSU study reaffirmed that large MECP2 deletions are an important cause of Rett syndrome. Guidelines for best practice in the management of scoliosis in Rett syndrome will be disseminated in 2010. (15)</td>
</tr>
<tr>
<td><strong>Other injury/illness</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Serious seatbelt injuries</td>
<td>Jan 2006–Dec 2007</td>
<td>Eighty percent of 4–8 year olds with serious seatbelt injuries were travelling in age-inappropriate restraints. The burden of seatbelt related injuries, particularly among children aged 6–8 years, is significant in terms of deaths, sustained injuries and length of hospital stay. Study results informed the new recommendations for child restraints under the 7th Amendment to Australian Road Rules as recommended by the National Transport Commission. (16)</td>
</tr>
<tr>
<td>Simple Vitamin D deficiency rickets</td>
<td>Jan 2006–Aug 2007</td>
<td>The APSU study highlighted the resurgence of vitamin D deficiency rickets in Australia. 400 children were confirmed with the diagnosis. Risk factors for vitamin D deficiency rickets are dark skin colour, veiling of mother or child and being a recent migrant. Although screening programs detected 72% of affected children, public health campaigns are required to address this resurgence of vitamin D deficiency rickets. (17)</td>
</tr>
<tr>
<td>Vitamin K deficiency bleeding (VKDB)</td>
<td>May 1993–</td>
<td>The majority of children with VKDB have a late onset versus an early/classical onset disease. Of those with a late onset, more than half have liver disease. Most children with VKDB did not receive vitamin K at birth or received insufficient Vitamin K. Two of three infants who died from VKDB were without liver disease and did not receive Vitamin K at birth. Implications of a change in the mode of vitamin K administration in newborns (introduction of oral preparations) are being analysed. (18)</td>
</tr>
</tbody>
</table>
Conditions Studied 2007–2008: Key Findings

References


18. National Health and Medical Research Council, Paediatric Division of the Royal Australasian College of Physicians, Royal Australian and New Zealand College of Obstetrics and Gynaecology, Royal Australian College of General Practitioners, Australian College of Midwives Inc. *Joint statement and recommendations on vitamin K administration to newborn infants to prevent vitamin K deficiency bleeding in infancy*. 2000. Canberra: NH&MRC.
Response Rates
In 2007, 1277 clinicians participated in the monthly surveillance of 17 conditions, with an overall response rate of 96% (Figure 1). In 2008, 1318 clinicians participated in the monthly surveillance of 15 conditions and the overall response rate was 94% (Figure 1). This maintains the excellent participation level by contributing clinicians since APSU’s inception in 1993. In 2007, 65% of clinicians reported by e-mail and this increased to 68% in 2008.

Figure 1. APSU annual response rate (%) 1993-2008

New South Wales (NSW) has the greatest proportion of the national population of children aged less than 15yrs (32.3%), Victoria has 24.2% and Queensland 21%. Correspondingly, NSW has the greatest number of participating clinicians. Response rates to the monthly report card have remained high in all states, with Queensland and Tasmania recording the highest rates in 2007 and NSW and Tasmania recording the highest rates in 2008 (Table 2).

Table 2. Response rates to monthly report card, number of clinicians reporting to the APSU and proportion of all children <15yrs of age for each state for 2007 and 2008.

<table>
<thead>
<tr>
<th>STATE</th>
<th>RESPONSE RATE (%)</th>
<th>CLINICIANS REPORTING N (%)</th>
<th>NUMBERS AND PROPORTION OF AUSTRALIAN CHILDREN &lt;15yrs N (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACT</td>
<td>94</td>
<td>91</td>
<td>20 (1.6)</td>
</tr>
<tr>
<td>NSW</td>
<td>97</td>
<td>95</td>
<td>514 (40.3)</td>
</tr>
<tr>
<td>NT</td>
<td>93</td>
<td>86</td>
<td>14 (1.1)</td>
</tr>
<tr>
<td>QLD</td>
<td>98</td>
<td>94</td>
<td>208 (16.3)</td>
</tr>
<tr>
<td>SA</td>
<td>95</td>
<td>93</td>
<td>99 (7.8)</td>
</tr>
<tr>
<td>TAS</td>
<td>98</td>
<td>99</td>
<td>23 (1.8)</td>
</tr>
<tr>
<td>VIC</td>
<td>94</td>
<td>92</td>
<td>285 (22.3)</td>
</tr>
<tr>
<td>WA</td>
<td>96</td>
<td>91</td>
<td>114 (8.9)</td>
</tr>
<tr>
<td>Australia</td>
<td>96</td>
<td>94</td>
<td>1277 (100)</td>
</tr>
</tbody>
</table>

Respondent Workload
Workload in completing questionnaires is low. During 2007 the majority of clinicians (79%) had no cases to report; 13.8% reported one case, 5.1% reported two and 2% reported three or more cases. During 2008, 79.5% of clinicians had no cases to report; 12.0% reported one case, 4.6% reported two and 4% reported three or more cases.

Summary of surveillance study results 2007-2008
A summary of the classification of all reports received for the period 2007-2008 is presented in Table 3. Duplicate reports are identified according to the child’s date of birth, first two letters of the first name and first two letters of the surname. After duplicates are identified, all data are completely deidentified. Errors include cases that do not meet case definition criteria or administrative errors including ‘report made by mistake’.

All data are provided after review by the expert investigators responsible for each surveillance study and are accurate as at December 2009. However, it is possible that some notifications may be reclassified at a later date as additional clinical data for existing notifications, or additional notifications, are received.
Table 3. Summary of results for studies conducted during 2007-2008

<table>
<thead>
<tr>
<th>Conditions Under Surveillance</th>
<th>Year</th>
<th>Total notifications</th>
<th>Questionnaires returned, n (%)</th>
<th>Duplicate cases</th>
<th>Errors</th>
<th>Probable/unknown cases</th>
<th>Total confirmed cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute flaccid paralysis *</td>
<td>2007</td>
<td>57</td>
<td>57 (100)</td>
<td>5</td>
<td>17</td>
<td>0</td>
<td>35</td>
</tr>
<tr>
<td></td>
<td>2008</td>
<td>90</td>
<td>83 (92)</td>
<td>11</td>
<td>9</td>
<td>0</td>
<td>63</td>
</tr>
<tr>
<td>Acute rheumatic fever *</td>
<td>2007</td>
<td>32</td>
<td>29 (91)</td>
<td>2</td>
<td>11</td>
<td>0</td>
<td>16</td>
</tr>
<tr>
<td></td>
<td>2008</td>
<td>82</td>
<td>71 (87)</td>
<td>10</td>
<td>10</td>
<td>3</td>
<td>48</td>
</tr>
<tr>
<td>Congenital cytomegalovirus infection *</td>
<td>2007</td>
<td>33</td>
<td>23 (70)</td>
<td>0</td>
<td>4</td>
<td>4</td>
<td>15</td>
</tr>
<tr>
<td></td>
<td>2008</td>
<td>54</td>
<td>46 (85)</td>
<td>3</td>
<td>5</td>
<td>4</td>
<td>34</td>
</tr>
<tr>
<td>Neonatal Group B streptococcus sepsis **</td>
<td>2007</td>
<td>77</td>
<td>60 (78)</td>
<td>2</td>
<td>12</td>
<td>0</td>
<td>46</td>
</tr>
<tr>
<td></td>
<td>2008</td>
<td>37</td>
<td>31 (84)</td>
<td>1</td>
<td>12</td>
<td>0</td>
<td>18</td>
</tr>
<tr>
<td>Hepatitis C virus infection</td>
<td>2007</td>
<td>6</td>
<td>6 (100)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>6</td>
</tr>
<tr>
<td>Neonatal herpes simplex virus infection</td>
<td>2007</td>
<td>12</td>
<td>11 (92)</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>9</td>
</tr>
<tr>
<td></td>
<td>2008</td>
<td>20</td>
<td>19 (95)</td>
<td>9</td>
<td>1</td>
<td>0</td>
<td>9</td>
</tr>
<tr>
<td>Perinatal exposure to HIV *</td>
<td>2007</td>
<td>44</td>
<td>33 (75)</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>32</td>
</tr>
<tr>
<td>Perinatal HIV infection</td>
<td>2007</td>
<td>44</td>
<td>33 (75)</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>32</td>
</tr>
<tr>
<td>Perinatal exposure to HIV *</td>
<td>2008</td>
<td>42</td>
<td>34 (81)</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>32</td>
</tr>
<tr>
<td>Hyperinsulinaemic hypoglycaemia</td>
<td>2007</td>
<td>8</td>
<td>6 (75)</td>
<td>2</td>
<td>1</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>Severe complications of influenza</td>
<td>2007</td>
<td>58</td>
<td>51 (88)</td>
<td>0</td>
<td>36</td>
<td>0</td>
<td>15</td>
</tr>
<tr>
<td>Severe complications of influenza</td>
<td>2008</td>
<td>104</td>
<td>104 (100)</td>
<td>8</td>
<td>37</td>
<td>0</td>
<td>59</td>
</tr>
<tr>
<td>Acute intussusception</td>
<td>2007</td>
<td>97</td>
<td>78 (80)</td>
<td>6</td>
<td>27</td>
<td>0</td>
<td>45</td>
</tr>
<tr>
<td></td>
<td>2008</td>
<td>116</td>
<td>91 (78)</td>
<td>9</td>
<td>13</td>
<td>0</td>
<td>69</td>
</tr>
<tr>
<td>Neuromuscular disorders</td>
<td>2007</td>
<td>167</td>
<td>139 (83)</td>
<td>21</td>
<td>38</td>
<td>1</td>
<td>79</td>
</tr>
<tr>
<td></td>
<td>2008</td>
<td>107</td>
<td>88 (82)</td>
<td>9</td>
<td>12</td>
<td>0</td>
<td>67</td>
</tr>
<tr>
<td>Non tuberculous mycobacterial infection *</td>
<td>2007</td>
<td>26</td>
<td>21 (81)</td>
<td>0</td>
<td>7</td>
<td>7</td>
<td>7</td>
</tr>
<tr>
<td>Rett syndrome *</td>
<td>2007</td>
<td>32</td>
<td>31 (97)</td>
<td>7</td>
<td>0</td>
<td>1</td>
<td>23</td>
</tr>
<tr>
<td></td>
<td>2008</td>
<td>21</td>
<td>20 (95)</td>
<td>3</td>
<td>1</td>
<td>1</td>
<td>15</td>
</tr>
<tr>
<td>Congenital rubella (with defects)</td>
<td>2007</td>
<td>1</td>
<td>1 (100)</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>2008</td>
<td>3</td>
<td>3 (100)</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Serious seatbelt injuries</td>
<td>2007</td>
<td>25</td>
<td>22 (88)</td>
<td>4</td>
<td>4</td>
<td>0</td>
<td>14</td>
</tr>
<tr>
<td>Congenital varicella</td>
<td>2007</td>
<td>4</td>
<td>4 (100)</td>
<td>1</td>
<td>2</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>2008</td>
<td>1</td>
<td>1 (100)</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Neonatal varicella</td>
<td>2007</td>
<td>8</td>
<td>7 (88)</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>2008</td>
<td>1</td>
<td>1 (100)</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Severe complications of varicella infection</td>
<td>2007</td>
<td>9</td>
<td>7 (78)</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>2008</td>
<td>10</td>
<td>9 (90)</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>7</td>
</tr>
<tr>
<td>Simple vitamin D deficiency rickets</td>
<td>2007</td>
<td>140</td>
<td>128 (91)</td>
<td>1</td>
<td>28§</td>
<td>0</td>
<td>99</td>
</tr>
<tr>
<td>Vitamin K deficiency bleeding</td>
<td>2007</td>
<td>6</td>
<td>6 (100)</td>
<td>2</td>
<td>1</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>2008</td>
<td>8</td>
<td>7 (88)</td>
<td>3</td>
<td>0</td>
<td>1</td>
<td>3</td>
</tr>
</tbody>
</table>

* Include notifications from APSU and other sources (e.g. laboratory). § Includes errors and unclassified cases
# Includes one case imported from a high prevalence country

An estimate of incidence is calculated using the reported number of newly diagnosed cases of disease in a defined population seen by paediatricians in a defined period of time. As 100% case ascertainment is unlikely to be achieved by any one surveillance scheme, ‘reported rate’ is used in this report to represent estimates of minimum incidence. The reported rate of each condition is expressed either as the number of new cases per 100,000 live births per annum (for conditions diagnosed before 12 months of age) or per 100,000 children in the age range specified in the study protocol (Tables 4a and 4b). Population figures for the denominator are obtained from the Australian Bureau of Statistics.2

Tables 4a and 4b shows the reported rate of disease for conditions studied through the APSU during 2007-2008. For conditions where cases were ascertained through additional complementary sources e.g. mandatory reporting systems and laboratory surveillance (including perinatal exposure to HIV, acute flaccid paralysis and Rett syndrome), cases from more than one source have been included to estimate the rate of disease.

Table 4a. Reported rate for each condition studied to December 2007

<table>
<thead>
<tr>
<th>Conditions Under Surveillance</th>
<th>Study period</th>
<th>Duration of study (years)</th>
<th>Total notifications</th>
<th>Questionnaires returned, n (%)</th>
<th>Total confirmed cases for duration of study</th>
<th>Reported Rate for duration of study (per 10^5 per annum)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Infectious/vaccine preventable conditions</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acute flaccid paralysis</td>
<td>Mar 1995 ongoing</td>
<td>13</td>
<td>723</td>
<td>665 (92)</td>
<td>447</td>
<td>0.87^a</td>
</tr>
<tr>
<td>Acute rheumatic fever</td>
<td>Oct 2007 ongoing</td>
<td>0.5</td>
<td>32</td>
<td>29 (91)</td>
<td>16</td>
<td>0.78^a</td>
</tr>
<tr>
<td>Congenital cytomegalovirus infection</td>
<td>Jan 1999 ongoing</td>
<td>9</td>
<td>292</td>
<td>206 (71)</td>
<td>93</td>
<td>4.02^a</td>
</tr>
<tr>
<td>Neonatal Group B streptococcus sepsis</td>
<td>Jul 2005 ongoing</td>
<td>2.5</td>
<td>241</td>
<td>196 (81)</td>
<td>132</td>
<td>19.31^a</td>
</tr>
<tr>
<td>Neonatal herpes simplex virus infection</td>
<td>Jan 1997 ongoing</td>
<td>11</td>
<td>194</td>
<td>188 (97)</td>
<td>98</td>
<td>3.48^a</td>
</tr>
<tr>
<td>Hepatitis C virus infection</td>
<td>Jan 2003 – Dec 2007</td>
<td>5</td>
<td>92</td>
<td>80 (87)</td>
<td>47</td>
<td>0.23^3</td>
</tr>
<tr>
<td>Perinatal exposure to HIV infection</td>
<td>May 1993 ongoing</td>
<td>14.5</td>
<td>513</td>
<td>455 (89)</td>
<td>301</td>
<td>8.1^a</td>
</tr>
<tr>
<td><strong>Serious complications of seasonal influenza</strong></td>
<td>Sep 2007</td>
<td>0.1</td>
<td>58</td>
<td>51 (88)</td>
<td>15</td>
<td>†</td>
</tr>
<tr>
<td>Intussusception</td>
<td>May 2007 ongoing</td>
<td>0.5</td>
<td>97</td>
<td>78 (80)</td>
<td>45</td>
<td>16.40^2</td>
</tr>
<tr>
<td><strong>Non tuberculosis mycobacteria infection</strong></td>
<td>Jul 2004 – Sep 2007</td>
<td>3.25</td>
<td>192</td>
<td>153 (80)</td>
<td>44</td>
<td>0.34^b</td>
</tr>
<tr>
<td>Congenital rubella (with defects)</td>
<td>May 1993 ongoing</td>
<td>14.5</td>
<td>107</td>
<td>103 (96)</td>
<td>50</td>
<td>0.08^a</td>
</tr>
<tr>
<td>Congenital varicella</td>
<td>May 2006 ongoing</td>
<td>1.5</td>
<td>8</td>
<td>8 (100)</td>
<td>2</td>
<td>0.48^a</td>
</tr>
<tr>
<td>Neonatal varicella</td>
<td>May 2006 ongoing</td>
<td>1.5</td>
<td>20</td>
<td>18 (90)</td>
<td>14</td>
<td>3.33^a</td>
</tr>
<tr>
<td><strong>Severe complications of varicella infection</strong></td>
<td>May 2006 ongoing</td>
<td>1.5</td>
<td>34</td>
<td>25 (74)</td>
<td>19</td>
<td>0.31^a</td>
</tr>
<tr>
<td><strong>Congenital/genetic disorders</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hyperinsulinaemic hypoglycaemia</td>
<td>Jan 2005 – Mar 2007</td>
<td>2.25</td>
<td>116</td>
<td>101 (87)</td>
<td>43</td>
<td>0.72^a</td>
</tr>
<tr>
<td>Neuromuscular disorders</td>
<td>Jan 2007 ongoing</td>
<td>1</td>
<td>167</td>
<td>139 (83)</td>
<td>79</td>
<td>1.81^d</td>
</tr>
<tr>
<td>Rett syndrome</td>
<td>Jan 2000 ongoing</td>
<td>8</td>
<td>245</td>
<td>239 (98)</td>
<td>129</td>
<td>0.77</td>
</tr>
<tr>
<td><strong>Other injury/illness</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Serious seatbelt injuries</td>
<td>Jan 2006 – Dec 2007</td>
<td>2</td>
<td>85</td>
<td>78 (92)</td>
<td>47</td>
<td>0.67^a</td>
</tr>
<tr>
<td>Simple vitamin D deficiency rickets</td>
<td>Jan 2006 – Aug 2007</td>
<td>1.7</td>
<td>851</td>
<td>805 (95)</td>
<td>400</td>
<td>5.91^a</td>
</tr>
<tr>
<td>Vitamin K deficiency bleeding</td>
<td>May 1993 ongoing</td>
<td>14.5</td>
<td>132</td>
<td>128 (98)</td>
<td>29</td>
<td>0.78^3</td>
</tr>
</tbody>
</table>

* Total confirmed cases according to case definition in the study protocol
  a. Reported incidence per 100,000 live births
  b. Reported incidence per 100,000 children < 15 years
  c. Reported incidence per 100,000 children ≤ 24 months
  d. Reported incidence per 100,000 children < 16 years
  e. Reported incidence per 100,000 children < 10 years
  f. Reported incidence per 100,000 females < 16 years
  g. Reported incidence per 100,000 children < 13 years
  † Due to the limited surveillance period a reported rate cannot be calculated
Table 4b. Reported rate for each condition studied to December 2008

<table>
<thead>
<tr>
<th>Conditions Under Surveillance</th>
<th>Study period</th>
<th>Duration of study (years)</th>
<th>Total notifications</th>
<th>Questionnaires returned, n (%)</th>
<th>Total confirmed cases for duration of study*</th>
<th>Reported Rate for duration of study (per 10^5 per annum)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infectious/vaccine preventable conditions</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acute flaccid paralysis</td>
<td>Mar 1995 ongoing</td>
<td>14</td>
<td>813</td>
<td>748 (92)</td>
<td>510</td>
<td>0.91^b</td>
</tr>
<tr>
<td>Acute rheumatic fever</td>
<td>Oct 2007 ongoing</td>
<td>1.5</td>
<td>114</td>
<td>100 (88)</td>
<td>64</td>
<td>1.04^a</td>
</tr>
<tr>
<td>Congenital cytomegalovirus (cCMV) infection</td>
<td>Jan 1999 ongoing</td>
<td>10</td>
<td>346</td>
<td>252 (73)</td>
<td>127</td>
<td>4.86^a</td>
</tr>
<tr>
<td>Neonatal Group B streptococcus sepsis</td>
<td>Jul 2005 – June 2008</td>
<td>3</td>
<td>278</td>
<td>227 (82)</td>
<td>150</td>
<td>18.03^a</td>
</tr>
<tr>
<td>Neonatal herpes simplex virus infection</td>
<td>Jan 1997 ongoing</td>
<td>12</td>
<td>214</td>
<td>207 (97)</td>
<td>107</td>
<td>3.44^a</td>
</tr>
<tr>
<td>Perinatal exposure to HIV infection</td>
<td>May 1993 ongoing</td>
<td>15.5</td>
<td>555</td>
<td>489 (88)</td>
<td>333</td>
<td>8.3^a</td>
</tr>
<tr>
<td>Neonatal herpes simplex virus infection</td>
<td>May 1993 ongoing</td>
<td>15.5</td>
<td>555</td>
<td>489 (88)</td>
<td>333</td>
<td>8.3^a</td>
</tr>
<tr>
<td>Serious complications of seasonal influenza</td>
<td>Jul 2008 – Sep 2008</td>
<td>0.25</td>
<td>104</td>
<td>104 (100)</td>
<td>59</td>
<td>†</td>
</tr>
<tr>
<td>Intussusception</td>
<td>May 2007 ongoing</td>
<td>1.5</td>
<td>213</td>
<td>169 (79)</td>
<td>114</td>
<td>13.56^c</td>
</tr>
<tr>
<td>Congenital rubella (with defects)</td>
<td>May 1993 ongoing</td>
<td>15.5</td>
<td>110</td>
<td>106 (96)</td>
<td>51</td>
<td>0.08^d</td>
</tr>
<tr>
<td>Congenital varicella</td>
<td>May 2006 ongoing</td>
<td>2.5</td>
<td>9</td>
<td>9 (100)</td>
<td>2</td>
<td>0.28^b</td>
</tr>
<tr>
<td>Neonatal varicella</td>
<td>May 2006 ongoing</td>
<td>2.5</td>
<td>21</td>
<td>19 (90)</td>
<td>14</td>
<td>1.95^e</td>
</tr>
<tr>
<td>Severe complications of varicella infection</td>
<td>May 2006 ongoing</td>
<td>2.5</td>
<td>44</td>
<td>34 (77)</td>
<td>26</td>
<td>0.25^c</td>
</tr>
<tr>
<td>Congenital/genetic disorders</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neuromuscular disorders</td>
<td>Jan 2007 ongoing</td>
<td>2</td>
<td>274</td>
<td>227 (83)</td>
<td>146</td>
<td>1.66^d</td>
</tr>
<tr>
<td>Rett syndrome</td>
<td>Jan 2000 ongoing</td>
<td>9</td>
<td>266</td>
<td>259 (97)</td>
<td>144</td>
<td>0.76</td>
</tr>
<tr>
<td>Other injury/illness</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vitamin K deficiency bleeding</td>
<td>May 1993 ongoing</td>
<td>15.5</td>
<td>140</td>
<td>137 (98)</td>
<td>32</td>
<td>0.80^e</td>
</tr>
</tbody>
</table>

* Total confirmed cases according to case definition in the study protocol
  a. Reported incidence per 100,000 live births
  b. Reported incidence per 100,000 children < 15 years
  c. Reported incidence per 100,000 children ≤ 24 months
  d. Reported incidence per 100,000 children < 16 years
  † Reported incidence per 100,000 females < 16 years
  †† Due to the limited surveillance period a reported rate cannot be calculated
Acute Flaccid Paralysis (AFP)
BR Thorley, HE Kelly, KA Brussen, M Ryan, J Antony, E Elliott

**Case Definition:** Any child less than 15 years of age newly diagnosed with acute flaccid paralysis in one or more limbs or acute onset of bulbar paralysis. All cases are reviewed by the National Polio Expert Committee (PEC) and classified as: confirmed poliomyelitis, non-polio AFP, polio-compatible AFP or non-AFP.

**Background:** For complete study protocol including rationale and objectives please see [www.apsu.org.au](http://www.apsu.org.au)

**Results:** Although no case of poliomyelitis was reported in children for 2007–2008, in July 2007 a case of suspected poliomyelitis in a 22 year old adult was reported by the attending clinicians. The National Polio Reference Laboratory subsequently identified a wild poliovirus from a clinical specimen that provided an epidemiological link to the patient’s recent travel to Pakistan. A case of poliomyelitis had not been reported in Australia since an importation from Turkey in 1977. This event highlights the need to maintain surveillance for AFP in children aged less than 15 years.

A total of 98 children aged less than 15 years were classified as having non-polio AFP by the PEC in 2007–2008. The most common diagnosis was Guillain Barre syndrome in 35 (36%) and transverse myelitis in 14 (14%).

The PEC determines if a case is compatible with poliovirus infection after review of a clinical questionnaire, completed by the paediatrician who notified the case, and laboratory results from virus culture from stool specimens for each case. The availability of clinical data through a new surveillance system, Paediatric Active Enhanced Disease Surveillance (PAEDS) enabled a greater number of cases to be classified by the PEC in 2008. The reporting rate for non-polio AFP in 2008 was 1.51 per 10^5 children aged <15 years, well above the WHO AFP surveillance performance indicator and the highest rate ever reported by Australia. PAEDS is a collaboration between the APSU and the National Centre for Immunisation Research and Surveillance, and funded by the Department of Health and Ageing to enable surveillance of rare diseases in hospitalised children and timely collection of clinical data and biological specimens.

For case classification details and reported rates please see Tables 3 and 4, pages 16–18.

**Study Highlights and Impacts:**
- The data generated via the AFP surveillance program is a critical component of Australia’s reporting obligations to the WHO as evidence of the maintenance of polio-free status. In 2008, the AFP surveillance program reported a non-polio AFP rate of 1.51 per 10^5 children aged <15 years. This is the fifth time that Australia has exceeded the WHO AFP surveillance performance indicator rate of one case of non-polio AFP per 10^5 children aged <15 years since the scheme was established in 1994.
- As in previous years, the most common diagnosis of non-polio AFP was Guillain-Barre Syndrome and transverse myelitis.
- The availability of clinical data through a new inpatient surveillance system operating in four hospitals, PAEDS*, complements APSU data and enhanced case identification in 2008.

*For more information on the PAEDS system please refer to page 47.

**Correspondence to:** Dr Bruce Thorley, Victorian Infectious Diseases Reference Laboratory, Locked Bag 815, Carlton South, Vic 3053. Email: Bruce.Thorley@mh.org.au Ph: 03 9342 2608 Fax: 03 9342 2665.

**Original Articles:**

**Abstracts:** None

**Presentations:**
Acute Rheumatic Fever (ARF)
J Carapetis, S Noonan, E Elliott, Y Zurynski, B Currie, M McDonald, G Wheaton, D Isaacs, J Ramsay, P Richmond, N Curtis, M Nissen

Case Definition: Any new episode of Acute Rheumatic Fever (even if there is a history of previous episodes) in any child <15 years of age and diagnosed according to the criteria provided by the National Heart Foundation Guidelines for Diagnosis and Management of ARF and RHD (NHFA & CSANZ (2006) Diagnosis and management of acute rheumatic fever and rheumatic heart disease in Australia: An evidence-based review. http://www.heartfoundation.org.au/document/NHF/PP-590_Diagnosis-Management_ARF-RHD_Evidence-Based%20Review_Sep06_Update_FINAL.pdf)

Background: For complete study protocol including rationale and objectives please see www.apsu.org.au

Results: During 2007–2008, there were 114 notifications and the questionnaire return rate was 88% (n=100). There were 64 confirmed cases, three probable cases, 12 duplicate reports, 19 reporting errors and two cases that could not be classified due to missing data. Almost all 64 children confirmed with ARF were born in Australia; 1 was born in New Zealand. The majority were reported in Queensland and Northern Territory where internal surveillance systems for ARF are already in place and the majority were Aboriginal or Torres Strait Islanders (Figure 2). Estimates of annual incidence for each state are shown in Table 5. Median age at diagnosis was 10 years (range 4–14 years) and 44% were male. Five children are known to have had previous ARF prior to the commencement of this study; reason for recurrence in all 5 was failure to present for regular secondary antibiotic prophylaxis with penicillin. At the time of diagnosis 73% of children resided in small towns/remote areas and 22% in large town/inner-city areas (3% unknown). Limited information was provided about social determinant risk factors and this may reflect the lack of routine collection of such information during medical consultations. Of the 28 children who had this information recorded, 93% were reported to live in a house with more than five residents in the house; and 68% of these lived in houses with 3–4 bedrooms.

Table 5. State and national estimated annual incidence

<table>
<thead>
<tr>
<th>State</th>
<th>N</th>
<th>Reported rate per 10^5 children &lt; 15 years per annum (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>NSW</td>
<td>9</td>
<td>0.3 (0.2–0.6)</td>
</tr>
<tr>
<td>NT</td>
<td>18</td>
<td>17.3 (10.2–27.3)</td>
</tr>
<tr>
<td>QLD</td>
<td>28</td>
<td>1.6 (1.1–2.4)</td>
</tr>
<tr>
<td>SA</td>
<td>1</td>
<td>0.2 (0–1.0)</td>
</tr>
<tr>
<td>VIC</td>
<td>1</td>
<td>0.1 (0–0.3)</td>
</tr>
<tr>
<td>WA</td>
<td>7</td>
<td>0.8 (0.3–1.7)</td>
</tr>
<tr>
<td>Total</td>
<td>64</td>
<td>0.8 (0.6–1.0)</td>
</tr>
</tbody>
</table>

The major manifestations in children diagnosed with ARF were carditis (45%), chorea (25%), poly-arthralgia* (25%), carditis (subclinical) (19%), poly-arthritis (21%), aseptic mono-arthritis* (19%), erythema marginatum (6%) and carditis and chorea (8%). Minor manifestations included raised ESR (72%), fever (61%), raised CRP (56%) and prolonged PR interval (20%). Prior to symptom onset, 30% reported a sore throat (58% received treatment), and 14% reported skin sores. Commonly affected joints were knees (59%) and ankles (42%). Long term secondary prophylaxis to prevent recurrent ARF was commenced in 62 of the 64 confirmed cases.

*Poly-arthralgia and aseptic mono-arthritis are considered major manifestation in known high risk groups including ATSI and Pacific Islander communities

Study Highlights and impacts:
- APSU provides a national picture of the estimated annual incidence of ARF as well as estimates of annual incidence for each state. Children with ARF were reported in all states of Australia except for the ACT and Tasmania. Only 9% (5/64) of the children had documented evidence of previous ARF.
- The majority of children with ARF were Aboriginal and Torres Strait Islanders who lived in small towns in remote areas. Children with information provided on their living conditions, 43% lived in crowded conditions.

Correspondence to: Ms Sara Noonan, Menzies School of Health Research, C/- 8 Denham Drive, Valley View, SA, 5093.
Email: sara.noonan@menzies.edu.au Ph: 08 88943 5015.

Original Articles: None
Abstracts: None
Presentations: None


**Congenital Cytomegalovirus Infection (cCMV)**

W Rawlinson, G Scott, P Palasanthiran, M Ferson, D Smith, G Higgins, M Catton, A McGregor, D Dwyer, A Kesson

**Case Definition:** Definite cCMV: Any child from whom CMV is isolated in the first three weeks of life from urine, blood, saliva or any tissue taken at biopsy. Suspected cCMV: Any child up to 12 months of age, in whom CMV is isolated from urine, blood, saliva or any tissue taken at biopsy **and/or** a positive serum IgM is found **and** in whom clinical features exist that may be due to intrauterine CMV infection.

Clinical features associated with cCMV infection include: prematurity, low birth weight, sensorineural deafness, other neurological abnormalities (encephalitis, microcephaly, developmental delay), seizures, microphthalmia, chorioretinitis, cataracts), hepatitis, hepatosplenomegaly, thrombocytopenia, pneumonitis or myocarditis.

**Background:** For complete study protocol including rationale and objectives please see www.apsu.org.au

**Results:** In 2007–2008 there were 87 notifications of cCMV and the questionnaire return rate was 79% (n=69). There were 49 confirmed cases, eight probable cases, three duplicate reports and nine errors. It appears that diagnosis of cCMV after the newborn period is increasing due to use of PCR techniques to test Newborn Screen Cards and stored cord blood samples. This retrospective testing identified newly diagnosed cases of cCMV infections that had occurred in previous years and were not previously diagnosed or reported. Additional cases may be identified by testing for CMV all babies who have an abnormality on newborn hearing screening tests. Three cases were confirmed in 2008 for notifications received in 2004 and 2005. The total of 49 confirmed cases includes these additional three cases.

For case classification details and reported rates please see Tables 3 and 4, pages 16–18.

**Study Highlights and Impacts:**
- The study provides baseline data about cCMV infections in Australia, monitors symptoms and improves early diagnosis through better understanding of CMV clinical epidemiology.
- Diagnosis of cCMV after the newborn period is increasing due to use of PCR techniques to test Newborn Screen Cards and stored cord blood samples.
- The release of guidelines for use of intravenous immunoglobulin (IVIG) in pregnant women by the Australian Red Cross Blood Transfusion Services has provided a strategy for clinicians treating infected pregnant women.
- A follow up study of a group of congenitally infected children reported to APSU at birth and seen by APSU investigators in paediatric infectious disease practice has been undertaken by the cCMV research group, including Brendan McMullan, Cheryl Jones, Pamela Palasanthiran and William Rawlinson. The results of this study are being prepared for publication.

**Correspondence to:**
Professor William Rawlinson, Virology Division, Department of Microbiology, Prince of Wales Hospitals, High Street, Randwick NSW 2031. Email: w.rawlinson@unsw.edu.au Ph: 02 9382 9113 Fax: 02 9398 4275c

**Original Articles:**
- None

**Abstracts:**

**Presentations:**
Neonatal Group B Streptococcal (GBS) Sepsis - Final Report
L Gilbert, S Garland, H Gidding, D Isaacs, A Daley, D Burgner, A Keil, J Faoagali, C Cooper

Case definition: Any infant with group B streptococcal (GBS) disease confirmed by isolation of GBS from a normally sterile site eg. blood, cerebrospinal fluid, joint fluid etc. All incident cases should be reported, in infants aged 0–7 days (early onset) or 8 days to 12 months (late onset) of age irrespective of symptoms. GBS may present clinically as:

- Early onset neonatal sepsis (EOD) (birth to 7 days) with symptoms and signs varying in severity from overwhelming multi-organ system disease with shock, respiratory failure, meningitis, DIC or acute tubular necrosis (especially in preterm infants) to non-specific signs such as fever, lethargy and poor feeding, localised infection e.g. pneumonia, or even apparently asymptomatic bacteraemia (more likely in full-term infants) OR
- Late onset sepsis (LOD) (8 days to 12 months) with evidence of fever, lethargy, poor feeding, with or without signs of focal infection such as meningitis, bone or joint infection or urinary tract infection. Occasionally late onset infection presents as overwhelming sepsis with shock.

Background: For complete study protocol including rationale and objectives please see www.apsu.org.au

Results: From July 2005–June 2008, there were 278 notifications of GBS sepsis. Clinical information was received for 227 (82%) notifications and 150 infants met case definition criteria for GBS sepsis. Eighty-eight infants presented with EOD (0.11 per 1,000 births) and 61 presented with LOD (0.08 per 1,000 births). In one infant the details of presentation were unknown and the child could not be classified as EOD or LOD. Pneumonia was more common among EOD presentations and meningitis was associated with LOD. Intrapartum fever was more common in mothers of infants with EOD (30.9%) than LOD (0%) but preterm labour was more common in mothers of infants with LOD (57.9%) than EOD (28.4%). Approximately half the mothers (50% of mothers of infants with EOD and 58.3% of mothers of infants with LOD) had been screened for GBS carriage prior to delivery and, of these, 50% and 39.3%, respectively, were positive. Intrapartum antibiotics were given to 15.4% of mothers of infants with EOD and 8.2% of infants with LOD. The distribution of serotypes and serotype III subtypes was significantly different among isolates from infants with late onset disease, compared with those from early onset disease or vaginal isolates from pregnant women. Specifically, an uncommon subtype of serotype III, which generally corresponds to the virulent sequence type 17, was found in 30% of invasive isolates from infants with late onset disease compared with 7% of those from infants with early onset disease and 4% of those from pregnant women. The proportions of serotypes and serotype III subsets among isolates from neonates with early onset disease were similar to those of isolates from vaginal swabs.

For case classification details and reported rates please see Tables 3 and 4, pages 16–18.

Study Highlights and Impacts:

- This study has confirmed a significant decrease in the incidence of neonatal GBS infection since the 1980s, when widespread use of intrapartum antibiotics began. Though the true incidence is double that recorded in this study (as found by a similar study by the German PSU, using capture/recapture methodology) the disease is now uncommon, despite inconsistent antenatal screening and use of intrapartum antibiotics, even for women identified as GBS carriers or with clinical risk factors. The origins of LOD remain obscure but the epidemiology appears to be significantly different from that of EOD and largely unaffected by current preventative measures.

Correspondence to: Professor Lyn Gilbert Centre for Infectious Diseases and Microbiology, Level 3, Institute of Clinical Pathology and Medical Research, Westmead Hospital, Westmead NSW 2145. Email: lyng@icpmr.wsha.ses.gov.au Ph: 02 9845 6255 Fax: 02 9893 8659.

Original Articles: None

Abstracts: None

Presentations:
Hepatitis C Virus (HCV) Infection – Final Report
C Jones, W Hardikar, E Elliott, S Polis, A Kesson, C Mews, J Kaldor

Case definition: Any child aged <15 years with newly diagnosed hepatitis C virus infection defined as:
- at least one confirmed positive anti-HCV antibody test performed at age greater than or equal to 18 months, OR
- a positive anti-HCV antibody test on a single occasion AND a positive test for HCV RNA (PCR or RT-PCR) on single occasion at any age > 1 month of age, OR
- a positive HCV RNA test (PCR or RT-PCR) on two separate occasions.

Background: For complete study protocol including rationale and objectives please see www.apsu.org.au

Results: Over the study period (2003–2008) there have been 92 notifications of HCV infection and the questionnaire response rate was 87% (n=80). There were 49 confirmed or probable cases of childhood HCV infection. Forty six (94%) infected children were born in Australia and three were born overseas (one each from Russia, Japan and Pakistan). Eighty one percent of children were born to mothers who were reported to be hepatitis C antibody positive and 6.1% of children had a history of their own intravenous drug use. The median age at diagnosis was 3 years 11 months (range 1 month to 14 years). The sex distribution was equal. The majority of children (90%) were asymptomatic at diagnosis; hepatomegaly was reported in 4% of cases and 47% of infected children showed minor elevation of liver transaminases (AST and ALT). Thus, HCV infected children are predominantly offspring of HCV infected women. As these children are mostly asymptomatic through childhood, clinical signs can not be used to identify HCV infected children. Consideration should be made of the cost benefit of screening all hepatitis C exposed children for this chronically and potentially debilitating infection.

For case classification details and reported rates please see Tables 3 and 4, pages 16–18.

Study Highlights and Impacts:
- Perinatal transmission is the main source of HCV infection in Australian children.
- Infants at risk were born to mothers who used IV drugs, had invasive procedures overseas or had tattoos.
- Most HCV infected children were clinically asymptomatic with mildly elevated liver function tests at diagnosis, however, HCV induced chronic liver disease and liver failure have been reported among older children.
- Given that 1–2% of Australian women of childbearing age are infected with HCV, the reported rate of infected children is lower than predicted. This may be due to the lack of a consistent approach to identifying children with HCV infection.
- This study has defined the local epidemiology of hepatitis C virus infection in childhood.
- The fact that HCV infected children were predominantly offspring of HCV-infected women identifies a need for education to improve investigation, diagnosis and management of HCV infection in children.
- We have identified a population of children at long term risk of sequelae of HCV in early adulthood.

Correspondence to: A/Professor Cheryl Jones, Head, Centre for Perinatal Infection, The Children’s Hospital at Westmead, Locked Bag 4001, Westmead NSW 2145. Email: CherylJ@chw.edu.au Ph: 02 9845 3382 Fax: 02 9845 3389

Original Articles: None

Abstracts: None

Presentations:
Neonatal Herpes Simplex Virus (HSV)

C Jones, D Isaacs, P McIntyre, T Cunningham, S Garland

Case definition: Any child aged ≤28 days (regardless of gestation) with clinical evidence of HSV infection* and either:
- HSV isolated from the baby OR
- HSV detected in CSF by PCR in association with CSF pleocytosis or other evidence of HSV encephalitis OR
- specific HSV-IgM detected in baby’s serum OR
- mother seroconverted or IgM positive and baby has typical clinical manifestations OR
- HSV isolated from mother around delivery and baby has typical clinical manifestations.

* Clinical manifestations may be localised (herpetic lesions of the skin, eye or mouth) or disseminated including encephalitis, pneumonitis, or hepatitis (manifest by coagulopathy, jaundice, hepatosplenomegaly)

Background: For complete study protocol including rationale and objectives please see www.apsu.org.au

Results: A total of 214 reports of neonatal HSV disease were made to the APSU from January 1997–December 2008. Clinical data were provided for 207 notifications (97%), of which 111 met the criteria of a confirmed or probable case of neonatal HSV infection. There was a predominance of females (55% female versus 45% male) which is in contrast to the more even gender proportions reported in the general population. The most common type of presentation of HSV infection in the newborn was with skin, eye, and mouth disease (49%). Disseminated HSV infection was reported in 27%, including 18/30 infants who also had encephalitis, and 10/30 infants who presented with pneumonitis. 21% presented with CNS infection alone. Three infants (2.7%) had features present at birth consistent with intrauterine infection. Of the 22 infants (21.6%) in this series who had died by the time of notification, 21 had died from complications of the HSV infection and 20 had presented with disseminated infection, including nine infants whose diagnosis was made at post mortem.

For case classification details and reported rates please see Tables 3 and 4, pages 16–18.

Study Highlights and Impacts:
- Neonatal HSV infection most commonly presents as disease localised to the skin, eye or mouth, which has a low mortality if treated promptly with antiviral therapy.
- However, nearly a third of infants present with disseminated multi-organ infection, which carries a high mortality. Presenting features of disseminated infection are often non specific, and cutaneous features may be absent, so this form of neonatal HSV infection is frequently not diagnosed until post-mortem.
- Intrauterine infection with HSV is rare and presents with a triad of malformations of the eye, brain (microcephaly or hydranencephaly) and skin (scarring and/or vesicles).

Correspondence to: A/Prof. Cheryl Anne Jones, Head, Centre for Perinatal Infection Research, The Children's Hospital at Westmead, Locked Bag 4001, Westmead NSW 2145 Email: CherylJ@chw.edu.au Ph: 02 9845 3382 Fax: 02 9845 3389.

Publications:
HIV Infection, AIDS and Perinatal Exposure to HIV
A McDonald, J Kaldor, K Nadew, J Ziegler, E Elliott

Case definition: Any child under 16 years of age who is found to be HIV antibody positive or have positive virus culture, polymerase chain reaction (PCR) or antigen. It is important that these reports include children born with maternal antibodies even if they are subsequently found not to have HIV infection.

Please report any neonate or child who meets the case definition who you have seen in the last month and have not previously reported to the APSU. This includes both old and new patients, even if they have been reported to the national HIV registry or the national AIDS register.

Background: For complete study protocol including rationale and objectives please see www.apsu.org.au

Results: During 2007 and 2008, there were 86 notifications of perinatal exposure to HIV and 67 (77%) questionnaires were returned. Of these reports, 63 infants met the criteria of perinatal exposure to HIV and one infant came from a high prevalence country in sub-Saharan Africa. The majority of infants exposed to HIV (58; 92%) were born in Australia in 2005–2008 and five were born overseas. The mother’s HIV infection was acquired through: heterosexual exposure in a high HIV prevalence country (24; 38%); injecting drug use (8; 13%); heterosexual contact with a partner from a high prevalence country (9; 14%); an injecting drug using partner (5; 8%) or other partner at risk in (5; 8%); heterosexual contact not further specified (10; 16%); and the source of exposure to HIV was other/undetermined in (2; 3%).

The mother’s HIV infection was diagnosed antenatally in 55 (87%) cases, postnatally in 7 (11%) cases and was not reported for one mother. Of the mothers diagnosed antenatally: 24 (44%) used all available interventions including antiretroviral therapy during pregnancy, elective caesarean delivery and avoided breastfeeding; 25 (45%) used two of the available interventions: antiretroviral therapy in pregnancy and avoided breastfeeding; and two (4%) did not use any intervention. No cases of mother-to-child transmission occurred among women whose HIV infection was diagnosed antenatally. Six of seven (86%) children born to mothers whose HIV infection was diagnosed postnatally acquired HIV infection. Two of these six children with HIV infection were Australian born.

For case classification details and reported rates please see Tables 3 and 4, pages 16–18.

Study Highlights and Impacts:
- The APSU makes a substantial contribution to monitoring perinatal exposure to HIV in Australia and has been the only source of information in 90% of cases reported in the last six years.
- National surveillance indicates that perinatal exposure to HIV and mother-to-child HIV transmission remains rare among children in Australia.
- The risk of mother-to-child transmission is minimised among women whose HIV infection is diagnosed antenatally and who make use of interventions. Mother-to-child transmission continued to occur among women whose HIV infection was diagnosed postnatally or who did not make use of interventions.
- Many women acquire HIV through heterosexual contact with partners from high prevalence countries.

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Original Articles:

Abstracts:

Presentations:

Surveillance Reports:
Hyperinsulinaemic Hypoglycaemia of Infancy (HI) – Final Report
R Greer, A Cotterill, R Walker, D Cowley, J Bell, M Thomsett, M Jack

Case definition: Any child under 10 years of age seen in the previous month with newly diagnosed hyperinsulinaemic hypoglycaemia, defined as:
- Low blood sugar (<2.6 mmol/L) with low blood fats and low ketones AND
- Inappropriately high insulin level AND
- Persistent or recurrent hypoglycaemic episodes and/or glucose infusion for more than 10 days

Background: For complete study protocol including rationale and objectives please see www.apsu.org.au

Results: During the period 2005–2007, there were 116 notifications of HI and the questionnaire return rate was 87% (n=101). There were 43 infants confirmed with HI. Three infants had definitive diagnoses: one had Beckwith-Wiedemann Syndrome, one had Congenital Disorder of Glycosylation, and one had ABCC8 mutation (these diagnoses were not made at the time of the APSU report, but were ascertained later when these infants and their families participated in follow-up research). Thirty eight (88%) infants presented at a maternity unit, neonatal unit or birth centre, with the remaining five at another hospital facility. Thirteen (30%) infants presented with seizures and 11 (26%) were 'jittery'. Other signs included 'floppy', 'funny turn', 'sweaty', 'staring episode', or poor feeding. Ten infants were reported as asymptomatic at diagnosis. Sixteen infants (37%) were detected through routine neonatal blood sugar surveillance and 19 (44%) had suggestive signs or symptoms. Mean birthweight was 3016 g (range 1350-4740g). In most infants, HI was attributed to 'small for gestational age', or no specific diagnosis was assigned. Genetic diagnosis for rare conditions is hampered by the high cost of mutational analysis. Approximately 50% of infants received ongoing treatment with diazoxide. Case follow-up after discharge from the neonatal unit was not possible for most infants, due mainly to the necessary de-identification of data collected via the APSU. It was not possible to identify risk factors for persistent hypoglycaemia from this study.

For case classification details and reported rates please see Tables 3 and 4, pages 16–18.

Study Highlights and Impacts:
- A definitive diagnosis was specified in only three of 43 infants with HI, highlighting the difficulties in identifying the cause of HI, the need for increased awareness among clinicians of this condition and for improved diagnostic services.
- In most infants, HI was attributed to 'small for gestational age', or no specific diagnosis was assigned. This together with the low rate of definitive diagnosis makes it difficult to provide a reliable incidence estimate of HI from this study.
- A high proportion of infants were discharged home on diazoxide.
- Our related study (Longitudinal follow-up in infants with HI) suggests that the median age for cessation of diazoxide in medically treated patients is around 13-14 years of age, therefore our APSU study raises the question of how many of the infants identified through the APSU study will require long-term therapy, as diazoxide therapy is not totally benign and has significant side effects.
- One quarter of infants had a seizure at presentation, highlighting the need for clinician awareness of the possibility of hypoglycaemia, which has the potential to cause brain damage.

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Original Articles: None

Abstracts:

Presentations:
Severe Complications of Influenza

Y Zurynski, David Lester-Smith, R Booy, M Festa, A Kesson, E Elliott

**Case definition:** Any child aged <15 years with laboratory confirmed influenza AND admitted to hospital AND with any of the following complications:

- Pneumonia (X-ray confirmed)
- Requirement for Ventilation
- Encephalitis / encephalopathy with or without seizures
- Myocarditis; Pericarditis; Cardiomyopathy
- Rhabdomyolysis
- Purpura fulminans
- Disseminated coagulopathy
- Transverse myelitis
- Polyneuritis
- Guillain-Barré
- Shock (requiring >40 ml/kg fluid resuscitation)
- Acute renal failure
- Reye’s Syndrome
- Laboratory proven secondary bacterial infection; Bacteraemia; Septicaemia; Bacterial pneumonia
- Death
- **Exclusion:** Simple febrile seizures

**Background:** For complete protocol including rationale and objectives please see www.apsu.org.au

**Results:** Surveillance was conducted for one month only during September 2007 in response to a request from the Australian Department of Health and Ageing following several reported deaths among children aged <5 years due to influenza. This study confirmed the feasibility of using the APSU for rapid response surveillance. We used a modified APSU surveillance method to obtain timely data on a weekly basis. The weekly report card return rate was 93% for the month of September. There were 15 children aged <5 years; most with influenza A. In addition, the APSU received reports of four children older than 5 years who had very severe complications requiring admission to a paediatric intensive care unit (PICU). No child had been vaccinated and none of the eligible children had received oseltamivir as treatment.

The surveillance was repeated in 2008, this time covering the influenza season July to September and including children aged <15 years. There were 59 confirmed cases: 14 (NSW); 16 (NT); 16 (QLD); 9 (SA); 2 (VIC) and 2 (WA). There were no cases reported in ACT or Tasmania. Again, a range of severe complications were reported, pneumonia the most common. ATSI children are over-represented (29%). One third required ventilatory support in PICU with a median length of stay of five days and the most severely unwell admitted to PICU were older (median age 5.1 years). Three of six children with encephalopathy had ongoing neurological dysfunction at discharge. Influenza vaccination remains uncommon even among children with risk factors and eligible for vaccination according to the current National Immunisation Program (17% of eligible children were vaccinated). The use of specific anti-influenza agents (oseltamivir) in eligible patients (aged >6months; n = 48) was rare (11%).

For case classification details and reported rates please see Tables 3 and 4, pages 16–18.

**Study Highlights and Impacts:**

- Influenza can cause severe complications in children including pneumonia, shock, encephalitis and cardiomyopathy and can lead to lengthy hospitalisations and ongoing medical problems.
- Few children were vaccinated, including those with underlying chronic conditions and eligible for vaccination under the National Immunisation Program.
- Although most of the children were more than six months old and eligible for treatment with the influenza specific antiviral, oseltamivir, few children had been treated.
- A universal annual influenza vaccination for children from six months of age and prompt treatment with oseltamivir, may prevent the severe complications of influenza in children and reduce the burden on health services and families.
- APSU continues to contribute data on the severe complications of influenza during the 2009 H1N1 influenza pandemic.

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**Original Articles:**

**Abstracts:**

**Presentations:**
Acute Intussusception (IS)

M Danchin, J Buttery, C Lloyd-Johnsen, D Strong, Y Zurynski, E Elliott, R Booy, P Richmond, V Krause, S Beggs, M Nissen, M Gold, J Bines

Case Definition: Any case of newly diagnosed acute intussusception in any child aged ≤24 months where intussusception is confirmed on air/liquid contrast enema, ultrasound or surgery. If diagnosis is by ultrasound, this should include the demonstration of an intra-abdominal mass by abdominal ultrasound with specific characteristic features (target sign or doughnut sign on transverse section and a pseudo-kidney or sandwich sign on longitudinal section) that are proven to be reduced by hydrostatic enema on post-reduction ultrasound.

Background: For complete protocol including rationale and objectives please see www.apsu.org.au

Results: During 2007 and 2008 there were 213 notifications of IS and the questionnaire return rate was 79% (n=169). One hundred and fourteen children were confirmed with IS, the majority of which were based in VIC (33%) and NSW (33%) in keeping with the larger child populations in these states. There were 70 (61%) males and 41 (36%) females; gender was unknown in three children. The median age was 7 months (1–24 months). Most children were diagnosed on abdominal ultrasound (67%) or by a combination of an abdominal ultrasound and an abdominal X-ray (10%). There were a total of 31 stool samples collected from the 114 children with confirmed IS, with only one sample was positive for rotavirus.

Immunisations were reported to be up to date according to the current Immunization Schedule in 83/114 (73%) children and of these, 28 (34%) had received a rotavirus vaccine. In ten children, IS developed within two weeks of vaccination for rotavirus. The median age was lower and rate of surgical intervention was higher for these ten children compared with the whole group of children with IS (Table 6).

Table 6. IS: Total APSU cases versus cases within 2 weeks of a rotavirus vaccine

<table>
<thead>
<tr>
<th></th>
<th>All cases</th>
<th>Cases within two weeks of rotavirus vaccine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of patients</td>
<td>114</td>
<td>10</td>
</tr>
<tr>
<td>Median age (months)</td>
<td>7</td>
<td>3.5</td>
</tr>
<tr>
<td>Surgical intervention</td>
<td>38 (33%)</td>
<td>5 (50%)</td>
</tr>
<tr>
<td>Resection</td>
<td>23 (20%)</td>
<td>3 (30%)</td>
</tr>
</tbody>
</table>

For case classification details and reported rates please see Tables 3 and 4, pages 16–18.

Study Highlights and impacts:
- Of all IS cases 20% occurred in infants who had received at least one dose of a rotavirus vaccine and 9% were within the first two weeks after vaccination. With such small numbers, an association between either Rotarix or RotaTeq and IS has not been demonstrated.
- IS within two weeks of rotavirus vaccination occurred in younger infants and the rate of surgical intervention and intestinal resection was higher compared with all children with IS. In comparison, the US VAERS (The Vaccine Adverse Event Reporting System) data showed there were 47 IS cases that occurred 1-21 days after RotaTeq vaccination, of which 47% required surgery and 23% required intestinal resection (Haber P, et al. Pediatrics. 2008; 121(6):1206-1212).
- While there is under-reporting of IS cases and limited vaccination data, these data justify the need for ongoing IS surveillance to further explore any possible relationship between the number of observed IS cases and the age at vaccination, dose and vaccine given. APSU data will be supplemented by the Paediatric Active Enhanced Disease Surveillance (PAEDS) system which relies on active case finding by specialist surveillance nurses to explore potential relationships between rotavirus vaccination and IS.

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Original Articles: None.

Abstracts:

Presentations:
Neuromuscular disorders of childhood (NMD)

M Ryan, A Kornberg, P Lamont, K North, P Rowe, K Sinclair

Case definition: Any child aged 15 years or less, seen in the previous month with a newly diagnosed, inherited or chronic auto-immune neuromuscular disorder (see table on APSU website: www.apsu.org.au).

Inherited neuromuscular disorder refers to any genetic disorder of the lower motor neuron i.e. disorders of anterior horn cell, motor and/or sensory peripheral nerve, neuromuscular junction or muscle.

Chronic auto-immune neuromuscular disorders are acquired immune-mediated disorders of peripheral nerve, neuromuscular junction or muscle, causing permanent or persistent (>3 months duration) symptoms. These disorders include chronic inflammatory demyelinating polyneuropathy (CIDP), myasthenia gravis and dermatomyositis.

Background: For complete protocol including rationale and objectives please see www.apsu.org.au

Results: During 2007 and 2008, 274 NMD notifications were received and the questionnaire return rate was 83% (n=227). Neuromuscular disorders were confirmed in 146 children, of which 38 (26%) were diagnosed at <1 year, 58 (40%) were diagnosed at 1–5 years, and 46 (32%) were diagnosed at 6–15 years (age was not specified in four).

Forty-eight (33%) children had a family history of NMD. Sixty-two (42%) children had a classic ‘floppy infant’ presentation, while 77 (53%) children were referred for delayed motor development. Antenatal diagnosis was made in only one case. Targeted genetic testing was diagnostic in 97 (66%) children. Forty-five (31%) children underwent neurophysiological studies and 43 (29%) children required muscle biopsies to establish a specific diagnosis.

The most common NMD were muscular dystrophies (49; 34%), congenital myopathies (31; 21%), spinal muscular atrophy (24; 16%) and inherited neuropathies (26; 18%). Myotubular myopathy appeared to be the most common congenital myopathy and Charcot-Marie-Tooth 1A disease, the most common form of inherited neuropathy.

Epidemiological studies such as this are the first step towards natural history and other research studies, and facilitate inclusion of Australasian subjects in multicentre international clinical trials.

For case classification details and reported rates please see Tables 3 and 4, pages 16–18.

Study Highlights and Impacts:

• Diagnosis of children with neuromuscular disorder was most common for the 1–5 year age group.
• The most common form of presentation was referral for delayed motor development and targeted genetic testing was the most common form of diagnosis.
• The most common forms of neuromuscular disease in childhood are muscular dystrophies, congenital myopathies, inherited neuropathies and spinal muscular atrophy.
• Increasing focus on epidemiological studies of common and severe diseases such as Duchenne Muscular Dystrophy (DMD) has led to establishment of a working group which is now preparing a report and recommendations for establishment of a National DMD Register.
• The facility to undertake studies such as this has led Australasia to be represented on the management board of the Cooperative Neuromuscular research Group (CINRG) and in the Therapeutics Advisory Group of TREAT-NMD; CINRG and TREAT-NMD are the two major international clinical trial networks for paediatric neuromuscular disorders.

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Original Articles:

Abstracts:
Non Tuberculous Mycobacterial Infection (NTMI) - Final Report

P Palasanthiran, C Blyth, E Best, C Jones, A Daley, G Henry, D Burgner, C Nourse, P Goldwater

Case definition: Any child under 15 years of age seen in the previous month newly diagnosed as:

1. **Definite NTMI**: Any child in whom a non-tuberculous mycobacterium species has been identified either by isolation on culture or by polymerase chain reaction (PCR) from a sample from a sterile site OR
2. **Probable NTMI**: A child who presents with any clinical features compatible with NTMI (see below) AND has undergone one or more of the supportive investigations (see below) AND in whom Mycobacterium tuberculosis (TB) infection is unlikely.

**Compatible clinical features**
- Lymphadenopathy (any site)\(^a\)
- Pulmonary disease with or without Constitutional symptoms\(^b\)
- Skeletal infection
- Cutaneous infection
- Ear disease

**Supportive investigations (one or more)**
- Microbiology: Acid fast bacilli (AFB) seen on sample or biopsy specimens or AFB grown from non-sterile site sample or positive AFB PCR on non-sterile site sample.
- Histopathology: Granulomatous inflammation or caseous necrosis or AFB seen.
- Skin testing: Tuberculin PPD skin testing ≥ 5mm and less than 15mm and/or Avian PPD ≥10mm.

\(^a\)Clinical features of NTMI lymphadenitis includes typical firm LN consistency +/- overlying skin changes (e.g. violaceous hue), with no associated constitutional symptoms.

\(^b\)Constitutional symptoms referable to NTMI infection include unexplained lethargy, fevers and/or anorexia and weight loss, generally only occurring with disseminated infections or pulmonary infections in chronic lung disease.

\(^c\)Avian PPD, manufactured by Commonwealth Serum Laboratories (CSL) Limited. Intradermal dose 10 IU.

Background: For complete protocol including rationale and objectives please see www.apsu.org.au

Results: There were 192 notifications of NMTI for the study period, July 2004–September 2007. The questionnaire return rate was 80% (n=153). One hundred and two children (44 confirmed and 58 probable cases) were diagnosed with NTMI during the study period. The median age was 2.9 years. Predisposing conditions were infrequent and included chronic respiratory disease (12) and immunosuppression (6). Lymphadenitis was the most frequent presentation (68) with pulmonary and disseminated disease infrequent (14 and 3, respectively). Isolates were collected from 68 children with *Mycobacterium avium-intracellulare* complex being most frequently isolated (33/68; 48.5%). Surgery was performed in 78 children and 42 children were treated with antimycobacterial therapy. Twenty-five children had surgery and antimycobacterial therapy. Follow-up data were available for 77 children with recurrence observed in 18 (23.4%) children despite having therapy. Complete excision was associated with a higher rate of treatment success when compared with all other therapies (OR: 9.48 [95%CI: 2.00–44.97], p = 0.001). *M. lentiflavum* infection accounted for 4.4% of culture confirmed cases and had a lower rate of treatment success than other species (0% vs. 78.2%; p = 0.016).

For case classification details and reported rates please see Tables 3 and 4, pages 16-18.

Study Highlights and Impact:
- Median age of children with NMTI was 2.9 years.
- Most children with NMTI have no predisposing conditions.
- Lymphadenitis is the most frequent presentation.
- *Mycobacterium avium-intracellulare* is the most frequently isolated organism.
- There is significant variation in surgical and medical therapies administered by Australian doctors.
- Complete surgical excision is associated with a higher rate of treatment success.
- Treatment success is influenced by Mycobacterial species, with a lower rate of treatment success following *M. lentiflavum* infection.
- Despite therapy, there were recurrences in 23.4% of affected children.

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Original Articles:

Abstracts: None

Presentations:
Rett Syndrome
H Leonard, C Bower, N de Klerk, S Silburn, L Nagarajan, S Fyfe, J Christodoulou, C Ellaway, H Woodhead, S Reilly, D Ravine

Case definition: A child <16 years age with newly diagnosed or possible Rett syndrome according to the clinical criteria tabled below or by genetic testing.

- Apparently normal prenatal and perinatal history
- Psychomotor development largely normal through the first 6 months or may be delayed from birth
- Normal head circumference at birth
- Postnatal deceleration of head growth in the majority
- Loss of achieved purposeful hand skill between ages 1/2 – 2 1/2 years
- Stereotypic hand movements such as hand wringing/squeezing, clapping/tapping, mouthing and washing/rubbing automatisms
- Emerging social withdrawal, communication dysfunction, loss of learned words, and cognitive impairment
- Impaired (dyspraxic) or failing locomotion

Background: For complete protocol including rationale and objectives please see www.apsu.org.au

Results: Mean weight, height and BMI z-scores were lower for girls with Rett syndrome than for the general population and decreased steadily with age. Twenty percent of girls needed enteral nutrition support and this was more common in those who were aged between 12 and 17 years. Those with late truncating mutations had significantly less enteral nutrition support than other mutation groups.

Data from a recently developed video-based evaluation tool showed that general but not complex gross motor skills declined with age. Those with a p.R133C, p.R294X, or a p.R255X mutation appear to have better motor skills overall than those with a p.R270X or large deletion mutation.

Fracture incidence in our cohort was nearly four times the population rate and there was even higher risk in girls with epilepsy or p.R270X or p.R168X mutations. Targeting interventions may decrease fracture incidence.

We found that those children with an initial diagnosis of autism had significantly milder symptomatology such as remaining ambulant, having some functional hand use and not developing scoliosis. Those with a p.R306C or p.T158M mutations were more likely than other mutations to have an initial diagnosis of autism. It is important to monitor girls presenting early with autism for subsequent signs and symptoms of Rett syndrome.

The investigative team based at the Children’s Hospital at Westmead identified large multi-exonic deletions in 12/149 apparently mutation-negative RTT patients using multiplex ligation dependent probe amplification. The patient group included an affected brother and sister with a large MECP2 deletion also present in their carrier mother. Our study reaffirmed that large MECP2 deletions are an important cause of Rett syndrome.

For case classification details and reported rates please see Tables 3 and 4, pages 16-18.

Study Highlights and Impacts:
- APSU contributes to the continuing enrolment of new cases of Rett syndrome with the Rett Syndrome cohort now consisting of 355 cases.
- Guidelines for best practice in the management of scoliosis in Rett syndrome to be disseminated in 2009.
- A consumer reference group ensures family representation and input to the study.
- At the World Rett Syndrome Congress held in Paris in 2008 our group contributed fifteen oral presentations, including two plenaries, and several poster presentations. These related to a wide variety of topics including genotype phenotype relationships, scoliosis, epilepsy, fractures and osteopenia, hand and gross motor function and data management.

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Original Articles:


Abstracts: None.

Presentations:


**Congenital Rubella**

C Jones, P McIntyre

**Case Definition:** Any child or adolescent <16 years of age who in the opinion of the notifying paediatrician has definite or suspected congenital rubella, with or without defects, based on history, clinical and laboratory findings.

**Background:** For complete protocol including rationale and objectives please see www.apsu.org.au

**Results:** Since surveillance began in 1993, there have been 110 notifications of congenital rubella and of 106 questionnaires returned (95%), 51 children were confirmed with congenital rubella. Reports have decreased significantly in recent years with only one child identified in 2004, born to an immigrant woman who had not been vaccinated against rubella. One child was notified in 2007 but was identified as a prevalent case previously reported in 2004. There were three notifications in 2008. One child was seen in a rehabilitation setting and it is likely that the child is not a new case. One child was notified in error, and there was one confirmed case. This was a child born to an immigrant woman from India whose vaccination history could not be confirmed. Serological testing was not performed.

*For case classification details and reported rates please see Tables 3 and 4, pages 16-18.*

**Study Highlights and Impacts:**
- Immigrant women born in countries with poorly developed vaccination programs should have serological testing for rubella after arrival in Australia and vaccination where appropriate.
- Travel to rubella endemic countries in the first trimester of pregnancy by women with no prior rubella immunity poses a risk of congenital rubella to the fetus.
- Continued vigilance for this rare congenital infection is essential given the seriousness of congenital rubella syndrome which is characterised by deafness, cataracts, growth retardation, mental handicap and cardiac abnormalities.

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**Original articles:** None

**Abstracts:** None

**Presentations:**
Serious Seatbelt Injuries – Final Report
Y Zurynski, E Elliott, L Bliston, M McCaskill, A Dilley, F Leditschke

Case definition: Any child aged 12 or under, restrained in a motor vehicle at the time of a crash in one of the following:
- Approved child restraint
- Booster seat combined with adult lap-sash belt or lap only belt
AND presenting with either (1) abdominal injuries OR (2) thoraco-lumbar spine injuries OR (3) cervical spine injuries.

Results: During 2006 and 2007, 85 reports were received and of these, 50 children (29 male) met case definition criteria for definite (n=47) or probable (n=3) injury related to the use of age-inappropriate restraints and seatbelts, or seatbelt misuse. No children aged <2 years were reported and there were only seven children aged <4 years. Most injured children reported to the study were aged 4 to 12 years (n=43). A high proportion (80%) of children aged 4 to 8 years were not appropriately restrained for their age and were using adult seatbelts rather than booster seats or approved child restraints. Information about seatbelt or restraint misuse was available for only 33 cases and seatbelts and restraints were misused in 64% of these. Misuse was highest (94%) among the 6–8 years age group.

Abdominal, spinal, head and neck and brain injury were the most common injuries sustained. This is in keeping with injuries related to the use of age-inappropriate restraints and seatbelts and to seatbelt misuse. There were four deaths and two children had severe spinal cord injury resulting in complete paraplegia.

For case classification details and reported rates please see Tables 3 and 4, pages 16-18.

Study Highlights and Impacts:
- Eighty percent of 4 to 8 year old injured children reported to this study were travelling in age-inappropriate restraints - usually in adult seatbelts rather than in booster seats or other approved child restraints.
- The rate of seatbelt misuse is high particularly among children aged 6 to 8 years.
- The burden of seatbelt related injuries is significant: four children died and four sustained severe spinal cord injuries leading to paraplegia in two; the average stay in hospital for the whole group was 11 days.
- Results from this study informed the new National Transport Commission recommendations for child restraints under the 7th Amendment to Australian Road Rules.

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Original Articles:

Abstracts:

Presentations:
Simple Vitamin D Deficiency Rickets (SVDD) – Final Report

C Munns, M Zacharin, C Rodda, E Davis, M Harris, J Batch, M Pascoe, J Fairchild, A Lafferty, A Whybourne, L Ward, R Morley, S Garnett, D Burgner, M Williams, Y Zurynski

Case definition: Any child aged <15 years of age with rickets secondary to simple Vitamin D deficiency (also known as nutritional rickets) confirmed biochemically and/or radiographically.

Biochemical criteria for inclusion as a case of Vitamin D deficiency (nutritional) rickets:

- Low serum 25-hydroxy vitamin D (25OHD)
- Elevated serum alkaline phosphatase

Background: For complete protocol including rationale and objectives please see www.apsu.org.au

Results: During the 18month study period (January 2006–August 2007), there were 851 notifications of vitamin D deficiency rickets and 805 questionnaires were returned (95%). Four hundred children were confirmed with vitamin D deficiency rickets. There was evidence of seasonal variation with a greater number of cases reported in winter and spring than in summer and autumn. The mean age at diagnosis was 6.3 yrs ± 4.8 SD. In the infant population, prolonged breast feeding was not evident. The significant biochemistry were reduced 25-hydroxy vitamin D (28.1 nmol/L), elevated Alkaline phosphatase (614.8 IU/L) and elevated Parathyroid hormone (16.2 pmol/L).

Case ascertainment was predominantly through refugee clinics, with 75% of reported children coming from these sources. Of the 400 children, 260 were reported from VIC, 74 from NSW and 71 from WA. Eighty percent of the mothers of children with vitamin D deficiency rickets were from Africa. Darker skin colour was common, with 85% of children having dark skin, 13% intermediate and 2% fair skin colour. The mothers of infants had high rates of veiling with 28% reporting always being veiled when outside, 63% usually and 9% never veiled. Veiling was also seen in girls with vitamin D deficiency rickets: 28% always; 87% usually and 9% never.

Presenting symptoms of affected children varied although 72% were detected through screening programs. Presentations included limb deformity (9%), bone pain (5%), poor growth (4%), motor delay (3%), seizure (3%), fracture (2%), hypotonia (1%) and respiratory problems (1%).

For case classification details and reported rates please see Tables 3 and 4, pages 16-18.

Study Highlights and Impacts:

- Presentation of data has highlighted the resurgence of vitamin D deficiency rickets in Australia with 400 children confirmed with vitamin D deficiency rickets.
- Risk factors for vitamin D deficiency rickets are dark skin colour, veiling of mother or child and being a recent migrant.
- Although screening programs detected 72% of affected children, public health campaigns are required to address this resurgence of vitamin D deficiency rickets.

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Original Articles: None

Abstracts:

Presentations:
**Congenital Varicella**

R Booy, D Burgner, M Nissen, J Buttery, Y Zurynski, E Elliott, M Gold, E Peadon

**Case definition:** Any stillbirth, newborn infant, or child <2 years who, in the opinion of the notifying paediatrician has definite or suspected congenital varicella syndrome, with or without defects and meets at least one of the following criteria:

- Cicatricial skin lesions in a dermatomal distribution and/or pox-like skin scars and/or limb hypoplasia
- Development of herpes zoster in the first year of life
- Spontaneous abortion, termination, stillbirth or early death following varicella infection during pregnancy

**Background:** For complete protocol including rationale and objectives please see [www.apsu.org.au](http://www.apsu.org.au)

**Results:** This study commenced in 2006. From four notifications with 100% questionnaire return rate, there was one infant confirmed with congenital varicella in 2006 and one in 2007. In the 2007 case, the foetus was infected at 14 weeks of gestation. The mother had received zoster immunoglobulin and acyclovir. The child presented with pox skin lesions. Congenital varicella remains an uncommon complication of varicella infection in the community.

*For case classification details and reported rates please see Tables 3 and 4, pages 16-18.*

**Study Highlights and Impacts:**

- The rate of congenital varicella remains low with one case reported in 2007 and none reported in 2008.
- Ongoing surveillance is needed to determine if the national varicella immunisation program has an impact on the incidence of congenital varicella.

**Correspondence to:** Professor Robert Booy, Director, NCIRS, The Children's Hospital at Westmead. Locked Bag 400, Westmead NSW 2145. Email: robertb2@chw.edu.au. Ph: 02 9845 1415.

**Original Articles:** None

**Abstracts:**


**Presentations:** None
Neonatal Varicella
R Booy, D Burgner, M Nissen, J Buttery, Y Zurynski, E Elliott, M Gold, E Peadon

Case definition: Any infant who, in the opinion of the notifying paediatrician, has neonatal varicella based on history, clinical and/or laboratory findings in the first month of life without features of congenital varicella syndrome. Features of neonatal varicella infection include pox-like rash which may be papulovesicular, vesiculopustular or haemorrhagic, and fever. Other systemic symptoms may be present. Complications of neonatal varicella include bacterial superinfection, neurological and haematological problems and general visceral involvement. The diagnosis of neonatal varicella can be made when an infant in the first month of life presents with clinical features of varicella infection. There may be a history of maternal varicella infection in the last 1–4 weeks of pregnancy or contact with a varicella infected person after birth.

The diagnosis can be confirmed by laboratory tests to detect:
- viral antigen/viral isolate from scrapings of the skin lesions or viral DNA from lesion fluid
- varicella specific IgM in a serum sample from the infant (or from the contact)

Background: For complete protocol including rationale and objectives please see www.apsu.org.au

Results: This study commenced in 2006. From 21 notifications with 90% questionnaire return rate, there have been fourteen infants confirmed with neonatal varicella: nine in 2006; five in 2007; and none in 2008. Laboratory confirmation of varicella was made in 7 (50%) cases. The median day of onset was 13.5 days (range 5–30 days). All neonates presented with skin lesions. One infant developed pneumonia and required ventilator support. Four out of 14 (29%) infants received either zoster immunoglobulin and acyclovir, immunoglobulin alone, or acyclovir alone. Six infants were exposed postnatally to varicella. All infecting contacts were family members including siblings, parents and other relatives, and the majority were unvaccinated.

For case classification details and reported rates please see Tables 3 and 4, pages 16-18.

Study Highlights and Impacts:
- There has been a marked decrease in cases of neonatal varicella since 2006 with no confirmed cases in 2008.
- Family members are the most common infecting contact for neonatal varicella and the majority are unvaccinated.
- Approximately one-third received either zoster immunoglobulin and anti-viral therapy, zoster immunoglobulin alone, or anti-viral therapy alone.
- There is a need for well disseminated guidelines on the management of perinatal exposure to varicella.

Correspondence to: Professor Robert Booy, Director, NCIRS, The Children’s Hospital at Westmead, Locked Bag 4001, Westmead NSW 2145. Email: robertb2@chw.edu.au. Ph: 02 9845 1415.

Original Articles: None

Abstracts:

Presentations: None
Severe Complications of Varicella Infection
R Booy, D Burgner, M Nissen, J Buttery, Y Zurynski, E Elliott, M Gold, E Peadon

Case definition: Any child aged 1 month or more, up to 15 years, hospitalised with varicella AND complicated by one or more of the following:
- Bacteraemia / septic shock
- Toxic shock syndrome/ toxin mediated disease
- Septic arthritis or other focal purulent collection
- Necrotising fasciitis
- Encephalitis
- Purpura fulminans/disseminated coagulopathy
- X-Ray evidence of pneumonia
- Fulminant varicella (multi-organ involvement)
- Reye’s syndrome
- Ataxia

Virological testing: In order to confirm varicella we recommend collection of a sample of vesicle fluid. Please Collect the sample and send to your local laboratory for culture or PCR or IF as per usual practice. The investigators will liaise with your virology laboratory regarding transporting the samples for genotyping.

Background: For complete protocol including rationale and objectives please see www.apsu.org.au

Results: This study commenced in 2006. From 44 notifications with 77% questionnaire return rate, there have been 26 children confirmed with severe varicella reported to the APSU: 14 in 2006; five in 2007; and seven in 2008. Laboratory confirmation was only used in 11% of cases. The median age at diagnosis was 4 years (range 0.5–12 years). Fifteen children (58%) presented with secondary bacterial infections including bacteraemia (8), meningitis (1), septic arthritis (4), osteomyelitis (2) and abscesses (4). Four children had X-ray confirmed pneumonia. Eight children presented with neurological complications including ataxia (3), encephalitis (1), meningitis (1), Guillain-Barre syndrome (1), opsoclonus-myoclonus syndrome (1) and a CSF-communicating intracranial dermoid cyst (1). One child had a history of immunosuppression. The median length of hospital admission was 10 days (range 3–40 days). Two children were admitted to an Intensive Care Unit. Seven children had ongoing complications or were still hospitalised at time of reporting. None of the 26 children had been eligible for varicella vaccination under the current National Immunisation Program but one child had previously been vaccinated against varicella. The source of varicella infection was identified in 15 cases; all infecting contacts were children.

For case classification details and reported rates please see Tables 3 and 4, pages 16-18.

Study Highlights and Impacts:
- There was a marked decrease in the number of cases in 2007 compared to 2006 but there was an increase in cases in spring and summer of 2008.
- The median age at diagnosis was 4 years (range 0.5–12 years).
- Fourteen children (56%) presented with secondary bacterial infections including bacteraemia, septic arthritis, osteomyelitis and abscesses, and eight children (32%) presented with neurological complications including ataxia, encephalitis, Guillain-Barre syndrome and opsoclonus-myoclonus syndrome.
- Most children admitted to hospital with severe complications of varicella were previously well children with no risk factors for severe complications. Ninety-six percent of cases were unimmunised against varicella. Other children including siblings were the identified infecting contacts.
- Further surveillance is needed to determine if there is a true decrease in cases of severe varicella since the inclusion of varicella in the National Immunisation Program in late 2005.

Correspondence to: Professor Robert Booy, Director, NCIRS, The Children’s Hospital at Westmead, Locked Bag 4001, Westmead NSW 2145. Email: robertb2@chw.edu.au Ph: 02 9845 1415.

Original Articles: None

Abstracts:

Presentations: None
Vitamin K Deficiency Bleeding (VKDB)
B Jalaludin, K Chant, P Loughnan, L Taylor, E Elliott

Case definition: Any infant <6 months of age with spontaneous bruising/bleeding or intracranial haemorrhage associated with prolonged clotting time, not due to an inherited coagulopathy or disseminated intravascular coagulation.

Background: For complete protocol including rationale and objectives please see www.apsu.org.au

Results: Since 1993, 140 notifications have been received and the questionnaire return rate was 98% (n=137). There were 32 infants with confirmed VKDB and seven infants with probable VKDB. In 2007, there were three confirmed infants, one each in NSW, QLD, and SA, and in 2008, there were three confirmed infants, one each in NSW, WA and Tasmania.

Skin bruising occurred in 15 infants, gastrointestinal bleeding in 11 infants and intracranial bleeding in seven infants. For all other infants, bleeding was noted either at the circumcision site, the umbilicus or at the Guthrie heel prick site. Three infants died.

Of the 32 infants confirmed with VKDB, 25 (76%) were late onset cases and seven were early/classical cases of VKDB. Of the 25 infants with late onset VKDB, liver disease was identified in 12 (48%) infants and three had died. Thirteen of the 25 (52%) infants had received Vitamin K at birth and of these, 10 (40%) infants had liver disease. Of the three infants who died, one had liver disease and died despite receiving vitamin K at birth, however, the other two infants that died had no liver disease but had not received vitamin K at birth. Of those infants with early/classical onset VKDB, no infants had liver disease and there were no deaths. Four of these infants received vitamin K at birth.

In five of six infants confirmed with VKDB in 2007 and 2008, consent for administration of vitamin K at birth was refused by parents.

There was a higher incidence of VKDB (2.09/100,000 live births) during the period when oral administration of Vitamin K was recommended (Jan 1993–Mar 1994) compared to VKDB incidence of 0.9/100,000 live births when intramuscular administration was recommended (April 1994 onwards), although this was not statistically significant.

For case classification details and reported rates please see Tables 3 and 4, pages 16-18.

Study Highlights and Impacts:
- The majority of infants with VKDB had late onset disease and approximately half of them had liver disease. Ongoing morbidity was more common in late onset disease and deaths occurred only in this group.
- We continue to see cases of VKDB in newborns due to withheld parental consent to provide vitamin K at birth (eight of ten cases since 2005), suggesting a need for education of parents during the antenatal period, about the serious potential consequences of refusing vitamin K administration at birth.
- Given that errors in administered dose and time of administration also have occurred, there is a need for increased awareness among health professionals about protocols for the administration of vitamin K at birth.

Correspondence to: A/Prof Bin Jalaludin, Epidemiology Unit, South Western Area Health Service, Private Mail Bag 7071, Liverpool NSW 1871. Email: b.jalaludin@unsw.edu.au Ph: 02 9828 8000 Fax: 02 9828 8012.

Original Articles: None
Abstracts: None
Presentations: None
Severe Complications of Influenza

Y Zurynski, David Lester-Smith, R Booy, M Festa, A Kesson, E Elliott

BACKGROUND

Influenza is a common childhood disease with a wide spectrum of severity from minor respiratory symptoms to severe respiratory illness and life-threatening multi-system complications.\textsuperscript{1,2} Significant morbidity and mortality has been reported in Australia, where 82/100,000 hospitalisations and 0.2/100,000 deaths are attributed to influenza in children aged < 5 years.\textsuperscript{3} Of 22 children admitted with complications of influenza to one paediatric intensive care unit (PICU) over a short period in 2003, 3 died and none had been immunized.\textsuperscript{10} During the 2007 influenza season a number of child deaths, attributed to influenza and its complications, were reported in the media. None of these cases was identified by national surveillance systems.

Currently, influenza surveillance systems in Australia are based on laboratory reporting. They provide the number of confirmed cases, trends over time and geographic distribution.\textsuperscript{14} However, they do not and cannot provide timely and detailed information about clinical presentation and risk factors, investigation and hospital management, complications, treatment, and outcomes of laboratory confirmed cases. A system of enhanced surveillance able to provide timely details about children with serious complications of influenza would facilitate a rapid response by health systems and may prevent further serious events. These surveillance data may also inform future immunisation policy and guidelines for diagnosis and treatment of influenza. In 2007 the APSU demonstrated the usefulness of collecting detailed clinical data on the severe complications of influenza.\textsuperscript{15}

The surveillance is to be conducted for 3 months (July to September) during the influenza season each year.

STUDY OBJECTIVES

This study aims to document in children hospitalised with severe complications of influenza:

- presentation, diagnosis and management,
- short-term outcome,
- known risk factors to inform future immunisation policy.

REPORTING INSTRUCTIONS

Please report any new episode of a severe complication of influenza in any child <15 years of age and diagnosed according to the criteria provided in the case definition.

CASE DEFINITION

Any child aged < 15 years with laboratory confirmed influenza AND admitted to hospital AND with any of the following complications:

- Pneumonia (X-ray confirmed)
- Requirement for Ventilation
- Encephalitis / encephalopathy with or without seizures
- Myocarditis; Pericarditis; Cardiomyopathy
- Rhabdomyolysis
- Purpura fulminans
- Disseminated coagulopathy
- Transverse myelitis
- Polyneuritis
- Guillain-Barré
- Shock (requiring >40 ml/kg fluid resuscitation)
- Acute renal failure
- Reye’s Syndrome
- Laboratory proven secondary bacterial infection; Bacteraemia; Septicaemia; Bacterial pneumonia
- Death
- Exclusion: Simple febrile seizures

FOLLOW UP NOTIFICATIONS

A brief questionnaire requesting details about the diagnosis will be sent to clinicians who notify a case of influenza to the APSU.

Please report cases of any children with severe complications of laboratory proven influenza and admitted to hospital as soon as possible by:

Fax: 02 9845 3082
Phone: 02 9845 3005
Mail: Australian Paediatric Surveillance Unit, Research Building Level 2,
The Children’s Hospital at Westmead, Locked Bag 4001, Westmead NSW 2145
INVESTIGATOR CONTACT DETAILS (Principal Investigator and contact person)

Dr Yvonne Zurynski* Deputy Director APSU and Senior Lecturer, Discipline of Paediatrics and Child Health, Sydney Medical School, The University of Sydney at Westmead, NSW

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Professor Robert Booy Deputy Director, National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases, The Children's Hospital at Westmead, NSW

Dr Marino Festa Staff Specialist, Department of Paediatric Intensive Care Unit, The Children's Hospital at Westmead, NSW

Associate Professor Alison Kesson Department Head, Infectious Diseases and Microbiology, Discipline of Paediatrics and Child Health, The Children's Hospital at Westmead, NSW

Professor Elizabeth Elliott Director APSU and Professor Discipline of Paediatrics and Child Health, Sydney Medical School, The University of Sydney, The Children's Hospital at Westmead, Sydney

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12. Smitherman HF, Caviness AC, Macias CG, Retrospective review of serious bacterial infections in infants who are 0 to 36 months of age and have Influenza A infection. Pediatrics 2005; 115(3):710-718.


Systematic Lupus Erythematosus

F Mackie, G Kainer, J Munro, K Murray, AR Rosenberg, B Wainstein, J Ziegler, D Singh-Grewal, C Boros, N Adib, E Elliott, R Fahey

BACKGROUND

SLE is a severe autoimmune disease that can present in childhood or adult life. Studies in children vary depending on the source of the study with incident rates of 0.22, 0.4, 0.47, 0.8 and 0.9 per 100 000 per year in The Czech Republic [1], Southern Sweden [2], Japan [3], United Kingdom (Midlands) [4] and Finland [5] respectively. Many studies have found a higher incidence or prevalence in those of African or Asian background [6] including the UK study [4] in children < 17 years which found that the incident rate in Asians was 5.6, Blacks 3.1 and Whites 0.4 per 100 000 per year. There are no data on incidence of SLE in Australia but it has been reported that there is a high prevalence of the disease in Australians of indigenous background (1: 1360) [7]. The magnitude of the problem of a severe disease such as SLE is not known in Australia and may well be considerable given the increase in the number of people of Asian background in Australia as well as the known increased prevalence in the indigenous population. Obtaining accurate data on our incidence and closely examining the ethnicity of the patients may indicate whether environmental factors in Australia play a role in the development of the disease. UV radiation has long been known to precipitate systemic and cutaneous lupus [8].

We are entering a new era in the management of lupus with many biological therapies being used/developed to treat SLE in a more targeted approach to control the inflammatory response [9]. Agents specifically targeting B cells (rituximab) and T/B cell interactions (abatacept) in particular show considerable promise. It still needs to be determined which type of patients will benefit from such therapies. This study, by determining the nature and severity of presentation of SLE children in Australia, may allow some estimation of how many patients may benefit and require such biological therapies. The follow-up questionnaire may provide a clinical snapshot of current practice and how many have received these various types of therapies in the first year of diagnosis.

There is a paucity of information about the demographics and severity of presentation of SLE in children in Australia and this study will provide the first national dataset on SLE presentation, diagnosis and treatment.

The case definition for paediatric lupus is based upon the agreed international criteria for classification for SLE [10] Appendix. However we have also expanded our definition to a practical one where fewer manifestations are acceptable (with a positive ANA) as it is acknowledged that it may take time for these to develop. These patients will be analysed subsequently as ‘likely’ cases. By also adding patients with a tissue diagnosis of SLE we allow inclusion of patients recently diagnosed but yet to develop the standard criteria. Given a pathological diagnosis we plan to analyse these as ‘definite’ cases.

STUDY OBJECTIVES

To estimate the incidence, geographical distribution and ethnic background of SLE in Australian children aged ≤15 years. This study will also describe the mode and severity of clinical presentation as well as 1 year outcome of SLE in children in Australia. The clinical specialty of the initial treating physician as well as types of medications and any medication related adverse outcomes used in the management of paediatric SLE in the first year after diagnosis will be described.

CASE DEFINITION

Any child ≤ 15 years of age who meets any of the 3 definitions below:

1. Any child ≤ 15 years of age fulfilling the clinical diagnostic criteria for SLE ie. presenting with 4 or more of the 11 listed and defined in Table 7, namely:
   1. Malar rash
   2. Discoid rash
   3. Photosensitivity
   4. Oral ulcers
   5. Arthritis
   6. Serositis
   7. Renal disorder
   8. Neurological disorder
   9. Haematological disorder
   10. Immunological disorder
   11. Antinuclear antibody
   OR

2. Any child ≤ 15 years of age who presents with 1 or more of the above clinical features AND a positive antinuclear antibody >1:320.
   OR

3. Any child ≤ 15 years of age who presents with a tissue diagnosis of SLE renal biopsy diagnostic of SLE or skin biopsy consistent with SLE.

REPORTING INSTRUCTIONS:

Please report ALL cases of children ≤ 15 years of age diagnosed with SLE in the last month. Please report all children in whom you suspect SLE even if results of tests are pending.

FOLLOW-UP OF REPORTED CASES

A questionnaire requesting further details will be forwarded to practitioners who report a case. A follow-up questionnaire will be forwarded 12 months later.
Table 7. Criteria for diagnosis of Systemic Lupus Erythema (SLE)

<table>
<thead>
<tr>
<th>Criterion</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Malar rash</td>
<td>Fixed erythema, flat or raised, over the malar eminences, tending to spare the nasolabial folds</td>
</tr>
<tr>
<td>2. Discoid rash</td>
<td>Erythematous raised patches with adherent keratotic scaling and follicular plugging; atrophic scarring may occur in older lesions</td>
</tr>
<tr>
<td>3. Photosensitivity</td>
<td>Skin rash as a result of unusual reaction to sunlight, by patient history or physician observation</td>
</tr>
<tr>
<td>4. Oral ulcers</td>
<td>Oral or nasopharyngeal ulceration, usually painless, observed by a physician</td>
</tr>
<tr>
<td>5. Arthritis</td>
<td>Non-erosive arthritis involving 2 or more peripheral joints, characterised by tenderness, swelling or effusion</td>
</tr>
<tr>
<td>6. Serositis</td>
<td>a. Pleuritis-convincing history of pleuritic pain or rub heard by a physician or evidence of pleural effusion OR b. Pericarditis-documented by ECG or rub or evidence of pericardial effusion</td>
</tr>
<tr>
<td>7. Renal disorder</td>
<td>a. Persistent proteinuria greater than 0.5 grams per day or greater than 3+ if quantitation not performed OR b. Cellular casts- may be red cell, haemoglobin, granular, tubular or mixed</td>
</tr>
<tr>
<td>8. Neurological disorder</td>
<td>a. Seizures - in the absence of offending drugs or known metabolic derangements eg, uremia, ketoacidosis or electrolyte imbalance OR b. Psychosis - in the absence of offending drugs or known metabolic derangements eg, uremia, ketoacidosis or electrolyte imbalance</td>
</tr>
<tr>
<td>9. Haematologic disorder</td>
<td>a. Haemolytic anaemia – with reticulocytosis OR b. Leukopenia- less than 4000/mm³ total on 2 or more occasions OR c. Lymphopenia- less than 1500/ mm³ on 2 or more occasions OR d. Thrombocytopenia- less than 100 000/ mm³ in the absence of offending drugs</td>
</tr>
<tr>
<td>10. Immunologic disorder</td>
<td>a. Anti-DNA: antibody to native DNA in abnormal titer OR b. Anti-Sm: presence of antibody to Sm nuclear antigen OR c. Positive finding of antiphospholipid antibody based on: 1. An abnormal serum level of IgG or IgM cardiolipin antibody 2. Positive test result for lupus anticoagulant using a standard method OR 3. False positive serologic test for syphilis known to be positive for at least 6 months and confirmed by Treponema pallidum immobilization or fluorescent treponemal antibody absorption test</td>
</tr>
<tr>
<td>11. Antinuclear antibody</td>
<td>An abnormal titer of antinuclear antibody by immunofluorescence or an equivalent assay at any point in time and in the absence of drugs known to be associated with “drug-induced lupus” syndrome</td>
</tr>
</tbody>
</table>

INVESTIGATOR CONTACT DETAILS (*Principal Investigator and contact person)
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Dr Brynn Wainstein, Sydney Children’s Hospital, Randwick, Sydney
Dr Jane Munro, Royal Children’s Hospital, Melbourne
Dr Kevin Murray, Princess Margaret Hospital for Children, Perth
A. Professor Andrew R. Rosenberg, Sydney Children’s Hospital, Randwick, Sydney
Dr Brynn Wainstein, Sydney Children’s Hospital, Randwick, Sydney
Professor John Ziegler, Sydney Children’s Hospital, Randwick, Sydney
Dr Davinder Singh-Grewal, The Children’s Hospital, Westmead, Sydney
Dr Christina Boros, University of Adelaide and CYWHS, Adelaide
Dr Navid Adib, Royal Children’s Hospital, Brisbane
Prof Elizabeth Elliott, The Children’s Hospital at Westmead, Sydney
Dr Rose Fahey, Alice Springs Hospital

REFERENCES
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### Conditions Studied

Between 1993 and 2008, the APSU has facilitated 44 studies and are listed in Table 8.

Table 8. Conditions studied from 1993-2008.

<table>
<thead>
<tr>
<th>Condition under surveillance</th>
<th>Dates of study</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Infectious/vaccine preventable conditions</strong></td>
<td></td>
</tr>
<tr>
<td>Acute flaccid paralysis</td>
<td>Mar 1995–</td>
</tr>
<tr>
<td>Acute rheumatic fever</td>
<td>Oct 2007–</td>
</tr>
<tr>
<td>Congenital cytomegalovirus infection</td>
<td>Jan 1999–</td>
</tr>
<tr>
<td>Hepatitis C virus infection</td>
<td>Jan 2003–Dec 2007</td>
</tr>
<tr>
<td>Neonatal herpes simplex virus infection</td>
<td>Jan 1997–</td>
</tr>
<tr>
<td>HIV/AIDS, Perinatal exposure to HIV</td>
<td>May 1993–</td>
</tr>
<tr>
<td>Hospitalised pertussis in infancy</td>
<td>Jan 2001–Dec 2001</td>
</tr>
<tr>
<td>Intussusception</td>
<td>May 2007–</td>
</tr>
<tr>
<td>Invasive haemophilus influenzae infection</td>
<td>Jan 1998–Dec 2000</td>
</tr>
<tr>
<td>Kawasaki disease</td>
<td>May 1993 – June 1995</td>
</tr>
<tr>
<td>Non tuberculous mycobacterial infection</td>
<td>July 2004–Sep 2007</td>
</tr>
<tr>
<td>Congenital rubella</td>
<td>May 1993–</td>
</tr>
<tr>
<td>Congenital varicella</td>
<td>May 2006–</td>
</tr>
<tr>
<td>Neonatal varicella</td>
<td>May 2006–</td>
</tr>
<tr>
<td>Severe complications of varicella</td>
<td>May 2006–</td>
</tr>
<tr>
<td><strong>Congenital/genetic disorders</strong></td>
<td></td>
</tr>
<tr>
<td>CHARGE association</td>
<td>Jan 2000–Dec 2002</td>
</tr>
<tr>
<td>Extrahepatic biliary atresia</td>
<td>May 1993–Dec 1996</td>
</tr>
<tr>
<td>Fetal alcohol syndrome</td>
<td>Jan 2001–Dec 2004</td>
</tr>
<tr>
<td>Haemoglobinopathies</td>
<td>Jul 2004–Mar 2006</td>
</tr>
<tr>
<td>Hirschsprung disease</td>
<td>Jan 1997–Dec 2000</td>
</tr>
<tr>
<td>Hyperinsulinaemic hypoglycaemia of infancy</td>
<td>Jan 2005–Mar 2007</td>
</tr>
<tr>
<td>Neuromuscular disorders of childhood</td>
<td>Jan 2007–</td>
</tr>
<tr>
<td>Primary immunodeficiency disorders</td>
<td>Jan 1997–Dec 1999</td>
</tr>
<tr>
<td>Rett syndrome</td>
<td>May 1993–Apr 1995; Jan 2000–</td>
</tr>
<tr>
<td>Severe combined immunodeficiency</td>
<td>May 1995–Dec 2001</td>
</tr>
<tr>
<td><strong>Mental health issues</strong></td>
<td></td>
</tr>
<tr>
<td>Childhood conversion disorder</td>
<td>Jan 2002–Dec 2003</td>
</tr>
<tr>
<td>Childhood dementia</td>
<td>May 1993–Jun 1995</td>
</tr>
<tr>
<td>Early onset eating disorder</td>
<td>Jul 2002–Jul 2005</td>
</tr>
<tr>
<td>Munchausen by proxy syndrome</td>
<td>Jan 2000–Dec 2003</td>
</tr>
<tr>
<td><strong>Other injury/illness</strong></td>
<td></td>
</tr>
<tr>
<td>Adverse reactions to complementary and alternative medicines</td>
<td>Jan 2001–Dec 2003</td>
</tr>
<tr>
<td>Anaphylaxis following food ingestion</td>
<td>Jul 2002–Dec 2003</td>
</tr>
<tr>
<td>Near drowning</td>
<td>May 1993–Dec 1996</td>
</tr>
<tr>
<td>Serious seatbelt injuries</td>
<td>Jan 2006–Dec 2007</td>
</tr>
<tr>
<td>Simple vitamin D deficiency rickets</td>
<td>Jan 2006–Aug 2007</td>
</tr>
<tr>
<td>Vitamin K deficiency bleeding</td>
<td>May 1993–</td>
</tr>
</tbody>
</table>
The International Network of Paediatric Surveillance Units (INoPSU) supports “the advancement of knowledge about rare and uncommon childhood infections and disorders through the participation of paediatricians in the surveillance on a national and international basis” and facilitates collaboration and information sharing among 12 active Paediatric Surveillance Units (PSUs) around the world. INoPSU member PSUs communicate regularly via e-mail and a bi-annual face-to-face meeting provides opportunity for networking, and exchange of ideas and experiences of rare disease surveillance.

The 4th Biannual Meeting of INoPSU was held in Munich, Germany on the 15th of September 2008, and provided opportunity for PSUs to present results of surveillance at a special session of the Annual Congress of the German Society for Child and Youth Medicine:

- Prevention of paraffin aspiration in children: The contribution of ESPED on the long way to child safe lamp oils in Germany. Axel Hahn
- International collaboration of surveillance units for rare diseases in children: Achievements and perspectives. Rob Pereira
- Rapid response surveillance for severe complications of influenza in children. Yvonne Zurynski
- Mad cow disease – an issue in children? Allan Colver
- The role of PPSU in the national surveillance of cerebral palsy in Portugal. Rosa Gouveia
- From surveillance to policy development: Seatbelt injuries and fetal alcohol syndrome in Australia. Yvonne Zurynski

At its business meeting INoPSU resolved to develop an international database to house information on the conditions studied by all member units, including case definitions, key findings and contact details of investigators. This is an important initiative that will facilitate information transfer and development of international surveillance studies.

Electronic reporting was high on the agenda with the Canadian Paediatric Surveillance Program (CPSP) and APSU very keen to start electronic reporting. Only three units currently conduct monthly surveillance using e-mail “cards”. To date only three units have conducted an evaluation: APSU, CPSP and BPSU, with APSU the only unit to be systematically evaluated twice. APSU will review evaluation standards for surveillance systems and produce a generic INoPSU Evaluation Method that could be adapted for evaluation of other units. Ongoing funding to support PSUs was the most common concern despite most units charging a study fee to research groups wanting to conduct surveillance.

INoPSU acknowledged the excellent leadership provided by Professor Rudi von Kries as Convenor of INoPSU and thanked Rudi and his team from The German Paediatric Surveillance Unit for organising the 2008 Bi-annual INoPSU meeting and for their generous hospitality. Dr Daniel Virella from the Portuguese Paediatric Surveillance Unit was elected INoPSU Convenor for 2008-2010. We look forward to further developments of INoPSU under his leadership.
British Paediatric Surveillance Unit (BPSU)
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The **Paediatric Active Enhanced Disease Surveillance (PAEDS)** is a pilot project initiated and coordinated by the Australian Paediatrics Surveillance Unit (APSU) and the National Centre for Immunisation Research and Surveillance (NCIRS), and funded by Population Health Division of the Commonwealth Department of Health and Ageing. PAEDS is based on the Canadian Immunization Monitoring Program Active (IMPAct) described by Scheifele and established over 10 years ago in 12 hospitals in Canada, whereby nurse monitors actively scan hospital records and collect data on inpatient cases of interest. PAEDS has proven the feasibility of conducting similar hospital-based surveillance for uncommon, serious, vaccine-related childhood conditions in Australia and is an important capacity building initiative to enhance existing public health surveillance relevant to vaccine-preventable diseases (VPDS), with the ultimate aim of improving child health outcomes.

A network of clinicians and public health researchers was established in four tertiary paediatric hospitals: The Children’s Hospital at Westmead Sydney, Royal Children’s Hospital Melbourne, Women’s and Children’s Hospital Adelaide and Princess Margaret Hospital Perth. Each employed a part time surveillance nurse. A centralised database, data management procedures, and communication strategy for the network was established and managed by the APSU.

**The unique aspects of PAEDS include its capacity for:**

- Ascertainment of cases unlikely to be detected through existing surveillance systems
- Timely case ascertainment and data review with weekly uploads into a central database
- Data not obtained by other means eg. vaccination, presentation, treatment and outcome
- Collection and analysis of biological samples linked to clinical data for the same patient
- Flexibility and responsiveness to urgent or emerging conditions; outbreaks or epidemics
- Population-based studies where populations around a reporting hospital are well defined
- Potential for verification of conditions included in data linkage initiatives, e.g. adverse events

**Key findings:**

From August 2007 to December 2008 detailed data on 389 hospitalised children meeting case definition criteria for the four conditions chosen for the pilot of PAEDS were collected.

- **Acute Flaccid Paralysis:** WHO’s **national** target for AFP surveillance achieved from PAEDS sites alone (58 cases in 15 months: 0.95/100,000 children aged <15y pa) and two stool samples were obtained <14 days after symptom onset in 48% of PAEDS compared with 45% of APSU.
- **Severe varicella:** 67 children hospitalised with varicella (9 with severe complications). Of the 15 eligible for varicella vaccine, only 6 (40%) were vaccinated, none with severe complications.
- **Intussusception (IS):** Of 138 cases identified, 83 were vaccinated against rotavirus, 9 within 7 days of developing IS. These data add significantly to passive surveillance through ADRAC.
- **Infantile seizures:** Among 126 infants aged 1 to 8 months hospitalised with seizures, only 15 (12%) had received any vaccine in the previous 7 days of whom 9 had pre-disposing conditions.

PAEDS is a powerful, sensitive active surveillance mechanism for acute serious **vaccine preventable diseases** and **adverse events following immunisation (AEFI)** in children. It also has response capacity for monitoring **disease outbreaks** e.g. pertussis or severe influenza. PAEDS provides timely data on severe disease to **support public health policy** and communication with clinicians and the community. If expanded to other states and territories of Australia, it could provide truly **national coverage**.

**PAEDS Study Group:**

- Prof Elizabeth Elliott and Dr Yvonne Zurynski - APSU
- Prof Peter McIntyre and Prof Robert Booy – NCIRS
- Prof Jim Buttery and Dr Jenny Royle, -Royal Children’s Hospital, Melbourne
- Prof Mike Gold and Dr Helen Marshall - Women’s and Children’s Hospital, Adelaide
- Dr Nicholas Wood - Children’s Hospital at Westmead
- Dr Peter Richmond - Princess Margaret Hospital, Perth

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Establishing a National Rare Diseases Working Group

Although by definition each rare disease occurs infrequently, collectively, approximately 8000 rare diseases affect approximately 6-10% of the population. This is equivalent to 30 million people in Europe, 25 million in the US and 1.2 million Australians. Comparatively, diabetes affects an estimated 1.4 million in Australia.

It is increasingly acknowledged that low prevalence does not equate with low impact. Although rare diseases vary considerably in aetiology, there are many commonalities. Rare diseases often have their onset in childhood, continue throughout the lifespan, are difficult to diagnose, are disabling, and have significant impacts and burdens on the patient, family, community and health services. Despite this, rare diseases receive little attention. For many rare conditions epidemiological and scientific data are lacking, making the development of evidence based practice and policy impossible.

After conducting a review of the literature on the impacts of rare diseases and international responses to rare diseases APSU convened a National Rare Diseases Working Group charged with developing a national plan that recognises the challenges faced by families affected by rare diseases and by clinicians who look after them, promotes research and addresses services, education needs and advocacy for people affected by rare diseases. The Rare Diseases Working Group includes researchers, child health advocates, clinicians and consumers support groups including:

1. SMILE Foundation
2. Association for the Welfare of Children in Hospitals (AWCH)
3. Association for Genetic Support Australia (AGSA)
4. European Organisation for Rare Diseases (Eurordis)
5. New Zealand Organisation for Rare Diseases (NZORD)
6. Royal Australasian College of Physicians, Paediatrics and Child Health Division
7. Royal Australian College of General Practitioners (RACGP)
8. The Children’s Hospital at Westmead
9. New South Wales Health
10. New South Wales Commission for Children
11. Australian Research Alliance for Children and Youth (ARACY)
12. The Steve Waugh Foundation
13. Anti-discrimination Commission of New South Wales

The National Rare Diseases Working Group aims to:

1. Draft a National Plan for Rare Diseases for Australia to address the burden of rare diseases by raising awareness, increasing the knowledge base about rare diseases, disseminating information relevant to parents, carers and the community, improving health care, promoting scientific and social research, providing educational resources, supporting families through peer networks and advocating for people affected by rare diseases.
2. Attract funding to support the development of a National Plan for Rare Diseases.
3. Develop a strategy for community awareness-raising on International Rare Diseases Day; 28th February 2009.

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Common front to fight rare ailments

WHEN her daughter Zoe was five months old, Nellie Evans was gripped by a powerful feeling. “Something was amiss,” she says now. “It was that innate sense that mothers have.”

It would take another two years and a heartbreaking regression in Zoe’s limited skills before the family would first hear the words Retts syndrome. These words have become central to their lives as, now four – a happy child wise adores music, Poppa Pug books and her seven-year-old sister, Ella – shuttles among appointments with specialists, speech and occupational therapists and physiotherapists.

Intensive therapy gives Zoe the best life possible under the shadow of the progressive genetic disorder that affects only girls, devastating their physical and mental development and the cause of sudden death in some cases at any time between childhood and middle age.

The Evans’ experience is common, said Elizabeth Elliott, the director of the Australian Pediatric Surveillance Unit.

“The non-specific symptoms of rare conditions unfamiliar to most doctors led to long delays in diagnosis, said Professor Elliott, whose establishment of the unit 15 years ago determined the prevalence in Australia of some rare diseases – most of which are genetic and first manifested in childhood.

Rare diseases, she said, “have been systematically neglected”. Individually, they are defined as affecting fewer than one in 10,000 people, but collectively, up to 10% of the population suffers from one.

Experts from around Australia now plan to campaign to “get rare diseases on the map, to make sure they are in equitable access to services”, for whose those disorders does not trigger a well-established package of care.

National referral clinics for doctors baffled by a patient’s unusual symptoms could shorted diagnosis time.

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*Zurynski et al. Archives of Disease in Childhood 2008; 93(12):1071-1074.*
In 1997, the APSU was the first of ten paediatric surveillance units in the world to undergo formal systematic evaluation using guidelines proposed by the Centers for Disease Control and Prevention (CDC). The Unit was re-evaluated in 2007 using the same CDC criteria. On both occasions, the APSU fulfilled most of its objectives and met CDC criteria for an effective surveillance system: usefulness, simplicity, flexibility, data quality, acceptability, sensitivity, representativeness and timeliness.

The 1260 paediatricians participating in APSU during 2007 represent 92% of Fellows in the Division of Paediatrics and Child Health within the Royal Australasian College of Physicians in active clinical practice in Australia. Anonymous evaluation questionnaires were returned by 818/1260 (65%) reporting clinicians, 32/42 (76%) principal study investigators, 15/15 (100%) Scientific Review Panel and APSU Board Members and 31/86 (36%) public health professionals. The APSU reporting mechanism was acceptable to 82% of clinicians and 97% said they returned ‘most or all’ of their report cards. An analysis of the APSU database reflected these results with an average 96% return rate for 2000-2007. Of those who reported by email (388), 99% found the email “card” user-friendly. Of the email cards 80% are returned within one week of mail out and much quicker than that of the yellow report cards. Of the 458 clinicians who had ever reported a case, 90% did not object to providing de-identified data and 74% found the questionnaires easy to complete and appropriate in length. Completed questionnaires were returned for 80–100% of all reported cases. Only 18% of clinicians felt burdened by the time involved in completing questionnaires and many (67%) felt that this could be improved by introducing on-line reporting via a secure website. We are currently developing an automated electronic reporting system.

Most (95%) of clinicians believe that the work of the APSU is valuable, specifically for generating knowledge (81%), e.g. diagnostic criteria, management of rare disease, guiding clinical practice (70%), informing future policy (70%) and evaluating current policy (68%), and identifying research priorities (75%). APSU studies have had significant impact with ~175 journal articles, ~200 presentations and ~85 media items. Results have informed policy and practice, e.g. the Fetal Alcohol Syndrome study informing the NH&MRC Australian Alcohol Guidelines. Most clinicians (83%) believed their contribution to the APSU was appropriately acknowledged in publications and 20% believed they personally benefited from their involvement.

The APSU has been shown to be flexible and responsive, responding within ten days to a request from the Commonwealth Government to conduct weekly surveillance for children hospitalised with serious influenza complications in 2007. Furthermore, the majority (90%) of clinicians indicated that they were willing to report immediately by mail/fax/phone in an epidemiological emergency and 65% had no objection to providing biological samples in this situation. Surveillance gaps for Indigenous children and children in rural and remote communities are currently being addressed by involving Aboriginal Medical Services in surveillance. The APSU’s future challenge is the acquisition of long term funding.

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Original Articles

Surveillance study related articles


**General publications**


**Letters**


Books and reports that include APSU data


Abstracts


Presentations


29. Elliott E, Hammill J. Videoconference with Tlingit and Haida Central Tribal Council of Alaska’s Fetal Alcohol Spectrum Disorder Diagnostic Team. Linked with University of Sydney, Westmead Children’s Hospital, Online Centre for Health Royal Children’s Hospital Brisbane, Alcohol Tobacco Other Drug Services Logan, Midwives Redland Bay Hospital, Central Queensland University, Mater Children’s Hospital, Queensland Association Indigenous Healthy Communities. Brisbane, September 2008.


Community and Media Impacts


Workshops and Policy Development


Awards


Clinicians Returning 100% Monthly Report Cards in 2007

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Dr Hock Leng Chua
Dr Harvey L C Coates AO
Dr Catherine H Cole
Dr Joanne Colvin
Dr Elizabeth Davis
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Dr Harry Dumbell
Dr Alan W Duncan
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Dr Philomena Fitzgerald
Dr Annkathrin Franzmann
Dr Noel P French
Dr Katharine Gardiner
Dr Gary C Geelhoed
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Dr Harvey Graham
Dr Elizabeth Green
Prof Sasson S Gubbay
Dr Anna Gubba
Clin A/Prof Ronald Hagan
Dr T Rex Henderson
Dr Ian K Hewitt
Dr Louise Houstlin
Dr Michelle Howell
Dr Lawrence T H Hu
Dr Mohammad Jeanghir

**Q L D**

Dr H Gunasekera
Dr Robert J Hardwick
Dr John G Harvey
A/Prof A Holland
Dr Maxwell Hopp
Dr Jason Hort
Dr Christine Hughes
Prof David Isaacs
Dr Heather Johnston
A/Prof Cheryl Jones
Dr Allan M Kerrigan
Dr Jan Klimek
Dr Peter Kristidis
Dr Mark Lee
Dr Ian D Lennon
Dr D Lester-Smith
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Dr Michael Lonergan
A/Prof Loughran-Fowlds
Dr Melissa Christine Luig
Dr Kristine Macartney
Dr Albert Mansour
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Dr Marilyn Rochefort
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Dr David N Schell
Dr Jacqueline A Stack
Dr J Taiz
Dr Kathryn E Thacker
Dr Rodney L Tobiansky
Prof Peter van Asperen

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Dr Tamsin Cockayne
Dr Rosemary E Fahy
Dr Deborah Fearon
Dr Alina Iser
Dr Peter S Morris
Dr Kathryn Roberts
Dr Robert Roseby
Dr Annie Whybourn

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Dr Annie Whybourn

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Dr David L Baker
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Dr Lawrence T H Hu
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Dr John Bethell
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| WA   | Dr Vanu J V Bayl | Dr Philip J Beeby | Dr Yvonne Belessis | Dr Graham J Bench | Prof David L Bennett | Dr G P Benet | Dr Jennifer Berg | Dr Viviek Bhadri | Dr Roger Blackmore | Dr Bijou Bick | Dr Paul Bloomfield | Dr Gilda B Bonacruz-Kazzi | Prof Robert Booy | Dr Jennifer R Bowen | Dr Nick Boyd | Dr J L D Burdon | Dr Donald L Butler | Dr Anne M E Bye | Dr Patrina Caldwell | Dr Ian Callander | Dr Peter J Campbell | Dr Dianne Campbell | Dr Thomas A Campbell | Dr Kathryn Carmo | Dr Jeffrey Chatlow | Dr Bronwyn J Chan | Dr Paul C Chay | Dr Kity Chee | Dr Alan Cheng | Dr H W Chilton | Dr Raymond Chin | Dr Alan Y H Chong | Dr R K C Chong | Dr David Christie | Prof J J Christodoulou | Dr Robert S L Chu | Dr Yew-Woe Chua | Dr Simon D Clarke | Dr John C Connectley | Dr Charles Hamilton | Dr Des Cohen | Dr Richard J Cohn | Dr Michael J Cole | Dr Alison F Colley | Dr Felicity A Collins | Dr Anne F Collins | Dr J S Colquhoun-Kerr | Dr J R Coomarasamy | Dr Peter John Cooper | Dr Stephen G Cooper | Dr Carolyn Cooper | Dr Elizabeth Cotterell | Dr Eric S Coudounaris | Dr Heather Caughtrey | A/Prof C Cowleton | Prof Jonathon Craig | Dr Maria Craig | Dr Peter Craven | Dr Geoffrey J Crawford | Dr Patricia Crock | Dr G E Cummings | Dr John Curotto | Dr Shane Curran | Dr Bruce Currie | Dr Julie A Curtin | Dr J Dalby-Payne | Dr Russell Dale | Dr Luce Dalla-Pozza | Dr Sarah Dalton | Dr P Davidson | Dr Robert Davies | Dr Robert Day | Dr Andrew Day | Dr John A De Courcy | Dr Mark De Souza | Dr M J Delougehery | Mr Anthony Dilley | Dr Kim Donaghey | Dr Peter John Donald | Dr Stuart F A Dorney | Dr Ana M Dosen | Dr David Dossetor | Dr Scott Dunlop | | Dr Richard J Dunstan | Dr Linda Dunojaie | Dr Shoma Dutt | Dr Peter W Ebeling | Dr Matthew J Edwards | Dr Jonathan Egan | Dr Peter D Eisman | Dr Fergus Elder | Dr Carolyn J Ellaway | Dr Philip J Emder | Dr Adrienne G Eppe | Dr Anthony D Epstein | Dr John B Erikson | Dr Elizabeth R Fagan | Dr Michael J Fairley | Dr Robert H Framsworth | Dr Bruce J Fasher | Dr Michael Fasher | Dr John Feller | Dr Penelope Field | Dr Michael J Field | A/Prof D A Fitzgerald | Dr Fiona Fleming | Dr Jeff Fletcher | Dr Bob K J Fonseca | Dr Michael R Freeland | Dr Stuart M Gadd | Dr Andrew J Gardiner | Professor Kevin J Gaskin | Dr Madlen Gazarin | Dr Maurice D Gett | Dr Deepak Gill | Dr Anna Clare Gill | A/Prof Jonathan Gillis | Dr Joanne Ging | Dr Neil D Ginsberg | Dr Anne F Glannville | Dr Rebecca Glover | Dr Chin Lum Goh | Dr Safak Goktogan | Dr Maria Linette Gomes | Dr P M Goodhew | Dr Linda Louise Goodwin | Dr Adrienne Gordon |}

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| NSW  | Dr C Abkiecikicwicz | Dr Susan Adams | Dr Julie C Adamson | Dr Lesley C Ades | Dr Ion S Alexander | Dr Stephen Alexander | Dr Hugh D W Allen | Dr Wendy Allen | Dr Frank Alvaro | A/Prof Geoff Ambler | Dr Alan F Amos | Dr Donald G Anderson | Dr Jacqueline K Andrews | Dr Michael Ansoncombe | Dr Jayne H Antony | Dr Elizabeth Argent | Dr John D Arnold | Dr Neil Atherton | Dr Jennifer E Ault | Dr Nadia S Dadawi | Dr Lynn Banna | Dr Peter A Barr | Dr Simon Battersby | Dr Karl Baumgart | Prof Louise A Baur |
| WA   | Dr Vanu J V Bayl | Dr Philip J Beeby | Dr Yvonne Belessis | Dr Graham J Bench | Prof David L Bennett | Dr G P Benet | Dr Jennifer Berg | Dr Viviek Bhadri | Dr Roger Blackmore | Dr Bijou Bick | Dr Paul Bloomfield | Dr Gilda B Bonacruz-Kazzi | Prof Robert Booy | Dr Jennifer R Bowen | Dr Nick Boyd | Dr J L D Burdon | Dr Donald L Butler | Dr Anne M E Bye | Dr Patrina Caldwell | Dr Ian Callander | Dr Peter J Campbell | Dr Dianne Campbell | Dr Thomas A Campbell | Dr Kathryn Carmo | Dr Jeffrey Chatlow | Dr Bronwyn J Chan | Dr Paul C Chay | Dr Kity Chee | Dr Alan Cheng | Dr H W Chilton | Dr Raymond Chin | Dr Alan Y H Chong | Dr R K C Chong | Dr David Christie | Prof J J Christodoulou | Dr Robert S L Chu | Dr Yew-Woe Chua | Dr Simon D Clarke | Dr John C Connectley | Dr Charles Hamilton | Dr Des Cohen | Dr Richard J Cohn | Dr Michael J Cole | Dr Alison F Colley | Dr Felicity A Collins | Dr Anne F Collins | Dr J S Colquhoun-Kerr | Dr J R Coomarasamy | Dr Peter John Cooper | Dr Stephen G Cooper | Dr Carolyn Cooper | Dr Elizabeth Cotterell | Dr Eric S Coudounaris | Dr Heather Caughtrey | A/Prof C Cowleton | Prof Jonathon Craig | Dr Maria Craig | Dr Peter Craven | Dr Geoffrey J Crawford | Dr Patricia Crock | Dr G E Cummings | Dr John Curotto | Dr Shane Curran | Dr Bruce Currie | Dr Julie A Curtin | Dr J Dalby-Payne | Dr Russell Dale | Dr Luce Dalla-Pozza | Dr Sarah Dalton | Dr P Davidson | Dr Robert Davies | Dr Robert Day | Dr Andrew Day | Dr John A De Courcy | Dr Mark De Souza | Dr M J Delougehery | Mr Anthony Dilley | Dr Kim Donaghey | Dr Peter John Donald | Dr Stuart F A Dorney | Dr Ana M Dosen | Dr David Dossetor | Dr Scott Dunlop | | Dr Richard J Dunstan | Dr Linda Dunojaie | Dr Shoma Dutt | Dr Peter W Ebeling | Dr Matthew J Edwards | Dr Jonathan Egan | Dr Peter D Eisman | Dr Fergus Elder | Dr Carolyn J Ellaway | Dr Philip J Emder | Dr Adrienne G Eppe | Dr Anthony D Epstein | Dr John B Erikson | Dr Elizabeth R Fagan | Dr Michael J Fairley | Dr Robert H Framsworth | Dr Bruce J Fasher | Dr Michael Fasher | Dr John Feller | Dr Penelope Field | Dr Michael J Field | A/Prof D A Fitzgerald | Dr Fiona Fleming | Dr Jeff Fletcher | Dr Bob K J Fonseca | Dr Michael R Freeland | Dr Stuart M Gadd | Dr Andrew J Gardiner | Professor Kevin J Gaskin | Dr Madlen Gazarin | Dr Maurice D Gett | Dr Deepak Gill | Dr Anna Clare Gill | A/Prof Jonathan Gillis | Dr Joanne Ging | Dr Neil D Ginsberg | Dr Anne F Glannville | Dr Rebecca Glover | Dr Chin Lum Goh | Dr Safak Goktogan | Dr Maria Linette Gomes | Dr P M Goodhew | Dr Linda Louise Goodwin | Dr Adrienne Gordon |
Clinicians Returning 100% Monthly Report Cards in 2008

Dr T M Grattan-Smith
Dr P Grattan-Smith
Dr Robert Guaran
Dr Hasantha Gunasekera
Dr Julie M Haas
Dr Dea Hankey
Dr Michael S Haifer
Dr Katherine Hale
Dr Nils F Hanson
Dr Ralph M Hanson
Dr Robert J Hardwick
Dr Richard K Hart
Dr John G Harvey
Dr Richard E Hawker
Prof Philip L Hazell
Mr Guy Henry
Dr Steven Hing
A/Prof Ken Ho
Dr Elisabeth M Hodson
Dr Peter Hogan
A/Prof Andrew Holland
Dr James C S Hong
Dr Peter Yee-Tai Hong
Dr Maxwell Hopp
Dr Jason Hon
Dr Clifford S Hosking
Dr Keith M Howard
Dr Neville J Howard
A/Prof R Howman-Giles
Dr Christine Hughes
Dr Paul Hutchins AM
Dr Christopher B Ingall
Prof David Isaacs
Dr Michelle M Jack
Dr Stephen Jacobe
Dr Adam Jaffe
Dr Alan James
Dr Con A James
Prof Heather E Jeffery
Dr Sandra L J Johnson
Dr Patricia M Johnson
Dr Heatherton Jones
A/Prof Alison M Kesson
Dr Debra Kennedy
Dr Allan M G Kerrigan
A/Prof Cheryl Anne Jones
Dr Kristi J Jones
Dr Colin Kible
Dr G M Kainer
Dr Alyson M Kakakios
Dr Hala Kaff
Dr Brian E Kearney
Dr Stewart J Kellie
Dr Allan Kelly
Prof A S Kemp
Dr Debra Kennedy
Dr Allan M G Kerrigan
A/Prof Alison M Kesson
Dr Bruce King
Dr Edwin P E Kirk
Dr Eli Kleiner
Dr Jan Klimek
Dr Martin R Kluckow
Dr Michael Koh
Dr Anthony Kok
Dr Philip Koles
Dr Kasia Koslowska
Dr U Krishnan
Dr Peter Kristidis
Mr Erik La Hei
Prof Alfred Lam
Dr Basiliki Lampropoulos
Dr K C Lau
Dr John A Lawson
Dr Joanne Leal
Dr Mark Lee
Dr Ian D Lennon
Dr Joyce Leong
Dr David Lester-Smith
A/Prof Florence Levy
Dr David Lillystone
Dr A S C Lim
Dr Daniel C S Lin
Dr Anthony Jun Wing Liu
Dr B H Lo
Dr Michelle Lonergan
Dr A Loughran-Fowlds
Dr O Lozynski
Dr Kei Lui
Dr Melissa C Luig
Dr Kevin T Macartney
Dr John Macdessi
Dr K T MacDonald
Dr Fiona E Mackie
Dr Albert Mansour
Dr Susan M Marks
Dr Glenn M Marshall
Dr Frank J Martin
Dr Hugh C O Martin
Dr Bradley Martin
Dr Sarah Martin
Dr Tania May
Dr Emma McCahon
Dr Robert McCarthy
Dr Mary Maccaskill
Dr Chris C Clymont
Dr Geoffrey McCowage
Dr Tim McCrossin
Dr David T McDonald
Dr Jennifer L McDonald
Dr Anne McGeechan
Dr Gayle McNerney
Dr Peter McNerney
Prof Peter B McIntyre
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Dr Fiona McKenzie
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Dr Michael Melamidowitz
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Dr Susan M Messner
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Dr Bronwyn Milne
Dr Joseph P Moloney
Dr Kieran T Moran
Dr Anne Morris
Dr David R Mowat
Dr Desmon L Mulcahy
Dr Craig Munns
Dr David N Murphy
Dr Maree W Murray
Dr Patricia E Mutton
Prof Ranjith Nanra
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Dr Karen O'Brien
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Dr Con Papadopoulos
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Dr D Singer-Remelian
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Dr Ingrid Sinnerbrink
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Dr B J N St. George
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Dr Murray T Webber
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Dr Jan Connors
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Dr Lucy Helen Cooke
Clinicians Returning 100% Monthly Report Cards in 2008

Dr Sarah McMahon
Dr Kim Alison McLennan
Dr Lynne McKinlay
Dr Neil David Dore
Dr Priya Edwards
Dr Loui Ee
Dr Ian J Findlay
Dr Paul W J Francis
Dr William Frischman
Dr Michael Gabbett
Dr Donna Gandini
Dr Michael R Gattas
Dr Leanne M Gaul
Dr John B Gavranich
Dr Andrew R Hallahan
Dr Simon Grew
Dr Andrew R Hallahan
Dr Alison Harris
Dr Margaret-Anne Harris
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Dr G J Harte
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Dr Richard Haizlewold
Dr Shivanand Hebbandi
Dr Anthony Herbert
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Dr E M Hurron
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Dr Susan Ireland
Dr Helen Irving
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Dr C M Johansson
Dr Robert N Justo
Dr Lisa Kane
Dr Sumant Kevat
Dr Paul Koch
Dr J Anne Kynaston
Dr David M L Levitt
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Dr Bruce R Lewis
Dr Helen Liley
Dr Margaret A Little
Dr Liane R Lockwood
Dr Elena J Mantz
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Dr John R McClean
Dr David B McCreary
Dr Michael McDowell
Dr Julie McGaughan
Dr James M McGill
Dr Lynne McKinlay
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Dr Sarah McMahon
Dr David C Mcmichael
Dr William R McWhirter
Dr Julian D Mellick
Dr Hilary P Mercer
Dr Ross D Messer
Dr Malcolm N Miller
Dr Ryan Mills
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Dr N Previetera
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Dr Peter C Roper
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Dr Doug C Shelton
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Dr Peter Kenneth Smith
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Dr Velencia Soutter
Dr H Stalewski
Dr S L Stathis
Dr Lisa Stephens
Dr Mark Stretton
Dr David Symmons
Dr Fiona Thompson
Dr Susan Thornton
Dr Alison Tigg
Dr Lim Ti
Dr Otilie Adrienne Tork
Dr Deanna Kathryn True
Prof David I Tudehope
Dr J Van der Westhuizen
Dr Claire E Wainwright
Dr Roslyn M Walker
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Dr Cameron J B Ward
Dr Timothy H Warmock
Dr John H N Waugh
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Dr R R Westmoreland
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Dr John S Whitehall
A/Prof Neil R Wigg
Dr Michael L Williams
Dr Judy A Williams
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Dr Suzanne Wilson
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Dr Stephen Withers
Dr David Wood
Dr Paul G Wooldgate
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SA
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Dr David A Baulderstone
Dr John Bethell
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Dr Hilary Boucaut
Dr Theresa Casey
Dr Yumin Chan
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Dr John K Freeman
Dr Liberty Gallus
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Dr Paul N Goldwater
Dr Andrew W Grieve
Dr Eric A Haan
Dr Paul Hammond
Dr T T S Han
Dr Michael G Harbord
A/Prof Ross R Haslam
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Dr Paul H Henning
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Dr Judith A Jaensch
Dr Simon L James
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Dr J D Kennedy
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Dr Paul D Machet
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Dr Ram Suppiah
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Dr Anthony J Dunstan
Dr Edmond J M Fenton
Dr Peter J Flett
Dr Evelyn Funk-Bowles
Dr Elizabeth Hallam
Dr Abu Haque
Dr Valerie M Hewitt
Dr Brendan McCann
Dr Tom McDonagh
Dr Mark M Pascoe
Dr Margaret M Phelan
Dr Ian G Stewart
Dr David Strong
Dr Charlotte M Whitelaw
Dr Michelle Williams
VIC
Dr Roger C Allen
Dr Katie J Allen
Dr David Amor
Dr Stuart G Anderson
Dr Kym P Anderson
Dr Yvla Anderson
Dr Giuliana C Antolovich
A/Prof K L Armstrong
Dr David S Armstrong
Mr Alexander W Auldist
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Dr Gordon Baikie
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Dr Peter L J Barnett
Dr Philip B Bergman
Dr John M Bishop
Dr Simon P Blair
Dr Ellen D Bowman
Dr Justin Brown
Dr Fiona D Brown
A/Prof Donald J Cameron
A/Prof Fergus J Cameron
Dr Martin Campbell
Dr William D Capell
Dr David J Caradine
Dr Elizabeth A Carse
Dr Daniel Casalaz
Dr Bronwyn A Catsell
Dr Anthony Chinn
A/Prof Caroline Clarke
Dr Tracy Coleman
Dr Kevin J Collins
Dr S Costello
Dr John M Court
Dr Noel E Cranwick
Dr Nigel Crawford
Dr Mick Creati
Dr Stuart Crisp
Dr David A Cutting
Clinical Surveillance:

Thank you to the following clinicians who participate in APSU surveillance, for their continued support of this important work. We look forward to your continued participation.

Dr A J Daley
Dr Margaret H Danchin
Dr Margot J Davey
Dr Peter Davis
Dr Roni M Davis
Dr Amol Davey
Dr Martin B Delatycki
Prof Paddy Dewan
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