Australian Paediatric Surveillance Unit

Annual Report 2004

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Minister for Health and Ageing
The Honourable Mr Tony Abbott MP

The Australian Paediatric Surveillance Unit is the only national surveillance program for rare childhood conditions in Australia. Over the past 12 years, the studies conducted by the APSU have generated findings which have been used to inform education strategies for clinicians and raise awareness of many rare health conditions here and abroad. I am also pleased that my Department continues to provide support to this important program.

The information generated by the APSU has enabled monitoring of public health interventions such as our national immunisation programs. The APSU conducts research and surveillance on a limited number of vaccine preventable conditions, such as congenital rubella, polio (under surveillance as acute flaccid paralysis), and whooping cough. This work has informed more effective targeting of vaccine delivery.

Many of the studies conducted by the APSU have important flow-on effects to stimulate further research. For instance, APSU work has led to the identification of an association between Rett Syndrome and specific genetic mutations, which in turn has led to the use of genetic testing to aid diagnosis.

The success of the APSU relies largely on the collaboration across sectors and governments. In partnership with the Royal Australasian College of Physicians, the Faculty of Medicine at The University of Sydney, The Children's Hospital at Westmead, and paediatricians throughout Australia, the APSU will continue to work towards the goal of improving the health and wellbeing of Australia's children.

Foreword

President, Paediatrics and Child Health Division Royal Australasian College of Physicians Associate Professor Neil Wigg

The Paediatrics & Child Health Division of The Royal Australasian College of Physicians is proud to have continuing association with the Australian Paediatric Surveillance Unit (APSU).

More than ninety percent of Australian paediatricians have regular monthly contact with APSU, notifying potential study children to researchers undertaking projects coordinated through the Unit. The suite of research studies, using APSU case-identification methods, provides a valuable research and research-training vehicle for paediatricians and College trainees.

APSU has a well deserved international and national reputation. Unit staff and associated researchers make a significant contribution to the College's Congress (formerly Annual Scientific Meeting), enabling direct feedback about nation wide studies to the participating paediatricians who have reported cases.

The work of APSU in providing valuable clinical epidemiologic information about uncommon conditions in childhood is highly commendable. The Paediatrics & Child Health Division of the College congratulates Associate Professor Elizabeth Elliott and her staff for their outstanding achievements again this year.

Chief Executive Officer, Royal Australasian College of Physicians Mr Craig Glenroy Patterson

As a Unit of the Paediatrics & Child Health Division, The Royal Australasian College of Physicians is appreciative of the valuable work undertaken by the Australian Paediatric Surveillance Unit (APSU).

The College also acknowledges the high level of voluntary co-operation of Fellows providing information through the Unit and the College's Board of Continuing Professional Development has agreed that participation in the APSU will be a practice-related Continuing Medical Education activity under current Maintenance of Professional Standards guidelines.

In addition, a session is to be included in the Paediatric Program of future RACP Congresses (formerly Annual Scientific Meetings) to highlight the importance of the APSU activities.

The College, through its Paediatrics & Child Health Division, looks forward to a continued and rewarding association with the APSU.

Dean of The Faculty of Medicine University of Sydney Professor Andrew Coats

As Dean of the Faculty of Medicine at the University of Sydney it gives me great pleasure to acknowledge the hard work of all the clinicians who contribute important data to the APSU. We take pleasure in supporting the work of the APSU and congratulate the APSU Director, Associate Professor Elizabeth Elliott and Assistant Director, Dr Yvonne Zurynski. I also acknowledge the strong support from our partners the Australian Government Department of Health and Ageing, the Paediatrics and Child Health Division of the Royal Australasian College of Physicians, The Children's Hospital at Westmead and our funding partners.

Every month clinicians around Australia report children with rare or uncommon disorders to the APSU. These conditions occur too infrequently for individual clinicians to gain enough consolidated experience to determine disease clusters, document aetiological factors and monitor trends over time. Over the last 12 years important information has been obtained for a number of these conditions and a list of key findings is included in the early pages of this report. Between 1993 and 2004 , APSU has monitored 34 uncommon childhood conditions. In many cases the data was unique and has lead to greater insight of these conditions. The monthly response rate of contributing clinicians has been very impressive, exceeding 90% in all years. The APSU has maintained some long term studies. Some studies exceeded a decade, a duration that is most difficult to maintain with normal funding regimes and study parameters. I invite you to browse through the report and see the fascinating information and important insights that can be obtained along with the lists of related reports and the contributions of all involved. My congratulations to all the staff who have contributed to this important work. We are proud at the Faculty of Medicine at the University of Sydney to help in some way with ongoing maintenance of this important initiative.

Chair, APSU Board Professor Carol Bower

In the past, short-term and uncertain funding has limited the activities of the APSU. It is therefore extremely encouraging to see that increased support from a number of different sources has been obtained for the Unit. The APSU Board is keen to secure additional funding both to strengthen the infrastructure of the APSU and to expand the activities of the APSU.

Plans for expansion include the promotion of national collaborative partnerships, raising the profile of the APSU and attracting new researchers. Research projects conducted through the APSU have largely been investigator driven. While we wish to maintain this option, enhanced collaboration would enable relevant and rapid responses to national health priorities and threats. For example, if the need arose, we could initiate immediate surveillance to monitor the emergence of bird flu or SARS in Australian children. Collaboration with health professionals in areas that are poorly served by paediatricians, such as rural and remote, often Indigenous communities would also be a valuable addition to the APSU, so that gaps in surveillance coverage are closed.

My congratulations to Associate Professor Elliott, her team and all the contributing paediatricians for the work underpinning the valuable information on the wide variety of childhood conditions included in this Report.

APSU Director
Associate Professor Elizabeth Elliott

In their book "Children of the Lucky Country" Fiona Stanley and co-authors conclude that "the future economic prosperity of our nation depends upon us focusing more on the developmental health and well-being of our children." They contend that prevention of illness and improvement in the quality of life of disadvantaged and ill children will minimise the burden of ill-health and mental illness and translate into economic prosperity. However, in order to advocate for children, to influence health outcomes, and to address inequalities we first need to collect accurate, current, national data.

Paediatricians should be congratulated for their continued support of the research program run by the Australian Paediatric Surveillance Unit. Over eleven years specialists have contributed to the collection of national data on over 30 uncommon conditions. Monthly reporting rates remain over 90% and there is ongoing interest from paediatricians wishing to initiate new studies. Studies conducted by the APSU concentrate on uncommon childhood conditions, complications of common conditions or adverse effects of treatment that have significant impacts on families and health resources.

The study on adverse effects of complementary and alternative medicines (CAM) led by Mike South is a timely example, in light of the evidence of increasing use of CAM in the Australian population. Through this study, a number of adverse effects resulting either from the use of CAM or the omission of conventional therapy have been identified. While it is likely that many children with adverse effects from CAM either do not present to paediatricians, or are not identified as users of CAM, this study alerts us of a need to take a careful history for CAM use in our patients.

APSU collects baseline information on emerging conditions of national public health importance. It is evident from Prof Jones' study of Hepatitis C infection that almost all such infections in Australian children are acquired perinatally and that mothers are infected primarily through the use of IV drugs, during procedures (including piercing and tattooing), or through receipt of blood overseas. This study will provide important data to inform policy for screening and management.

Monitoring conditions over time allows for evaluation of trends. Prof Kaldor's study of HIV/AIDS and perinatal exposure to HIV is a case in point. Over the eleven year study, there has been an increase in the number of women who acquired infection through heterosexual contact and a fall in the number using IV drugs. An increase in antenatal diagnosis of HIV in women allowing for use of interventions and resulting in a significant fall in the rate of transmission of infection has also been documented. In 2004 all cases reported nationally were of perinatal exposure to HIV and there were no new HIV infections, in Australian women.

APSU studies are important to raise awareness of uncommon conditions and to estimate the burden of disease. Prof Bower's data on Fetal alcohol syndrome (FAS) confirm the high social and medical costs of this condition and documents that opportunities for prevention have been missed. Less than one half of the children reported live with a biological parent, most use multiple health education and community services, and one third have an affected sibling. Research stimulated by this study suggests that the diagnosis is often missed or withheld and that education of health professionals and the community is required before prevention can be achieved.

Molecular epidemiological studies can also be facilitated through APSU as indicated by the Rett Syndrome study. This study, which has allowed Dr Leonard's group to describe the clinical features of Rett syndrome, determine disease burden, and make phenotype/genotype correlations, will greatly enhance clinical management of this complex and chronic disease. The international phenotype and genotype databases (Rettnett) and (InteRett) arising as an extension of this project confirm Australian researchers as leaders in this field.

APSU has led the way internationally with the recent addition of studies of mental health disorders in children (e.g. early onset eating disorders, conversion disorder). This broadens the Unit's remit to address one of Australia's health priorities and provides internationally unique data.

Our relationship with Units internationally was consolidated at the 3rd Business and Scientific meeting of the International Network of Paediatric Surveillance Units (INoPSU). We are grateful to the Portuguese Paediatric Society who hosted and sponsored the meeting in Lisbon in April 2004. Prof Rudi von Kries from the

German unit and Dr Rob Pereira from the Netherlands were elected as co-convenors as I stood down. Standardised data on a number of conditions, (including FAS, early onset eating disorder, acute flaccid paralysis and herpes simplex virus infection) are being collected simultaneously by several units, which will allow for international comparisons. An invited presentation by INoPSU at the 2004 International Paediatric Association meeting in Mexico resulted in considerable interest from countries seeking to establish new surveillance units.

The APSU program is a truly collaborative national effort, involving voluntary participation by paediatricians and involvement of organizations throughout Australia and investigators from a range of scientific disciplines. On behalf of all APSU investigators I acknowledge these contributions. I thank the APSU staff, Yvonne Zurynski, Rosemary Robertson, Paula Cronin and Ingrid Charters for their hard work in running the Unit and producing this report. I am grateful for the ongoing support of our Patron Prof Fiona Stanley; the Department of Health and Ageing; the Royal Australasian College of Physicians and its Division of Paediatrics and Child Health; the Faculty of Medicine in the University of Sydney and The Children's Hospital at Westmead which houses the APSU. Thanks also to the APSU Board, Board Chair Prof Carol Bower and members of the Scientific Review Panel for all their work.

We have challenges ahead. We must initiate and support research addressing national health priorities and to consolidate our infrastructure funding and enable adequate staffing. We need to embrace information technology to enable web-based reporting, increase efficiencies and decrease the workload for busy paediatricians. We need to establish new collaborations to strengthen data quality and address gaps in surveillance coverage e.g. in remote, often indigenous communities in order to maximise case ascertainment. We have an increasing role facilitating education through conduct of seminars and development of materials for paediatricians and parents; and also must ensure timely dissemination of data from APSU studies to inform policy and clinical practice.

[1] Stanley F, Richardson S, Prior M. Children of the Lucky Country? How Australian society has turned its back on children and why children matter, Pan Macmillan Australia, 2005.

APSU BOARD AND SCIENTIFIC REVIEW PANEL

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The University of Western Australia

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John Ziegler
Clinical Immunologist and Head, Department of Immunology and Infectious Diseases, Sydney Children's Hospital. Associate Professor, School of Women's and Children's Health, University of New South Wales.

* Board and Scientific Review panel members

INSTITUTIONS COLLABORATING WITH THE APSU 1993-2004

National Organisations

- 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 Society of Clinical Immunology and Allergy
- Commonwealth Department of Health and Ageing
- National Centre in HIV Epidemiology and Clinical Research
- National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases
- National Notifiable Diseases Surveillance System
- National Perinatal Statistics Unit
- National Polio Reference Laboratory
- OzFoodNet: Australian Enhanced Foodborne Disease Surveillance
- Rett Syndrome Association of Australia & AussieRett

New South Wales

- Bankstown Hospital
- CAMSHNET
- Centre for Kidney Research
- Centre for Mental Health, NSW Health
- Children's Hospital at Westmead
- Gastroenterology & 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 Heath
- Paediatric HIV Services Unit, Sydney Children's Hospital
- Royal Prince Alfred Hospital
- Royal North Shore Hospital
- Sydney Children's Hospital
- University of NSW
- University of Sydney
- South Eastern Sydney Area Health Service
- South Eastern Area Laboratory Services
- Westmead Hospital
- South Western Sydney Area Health Service

Victoria

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

Queensland

- Mater Children's Hospital
- Princess Alexandra Hospital
- Queensland University of Technology
- Royal Children's Hospital, Brisbane
- Tropical Public Health Unit
- University of Queensland

South Australia

- Flinders 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
- King Edward Memorial Hospital, Perth
- Pathcentre Queen Elizabeth II Medical Centre
- Princess Margaret Hospital for Children, Perth
- Royal Perth Hospital
- Telethon Institute for Child Health Research

Tasmania

- Royal Hobart Hospital

Northern Territory

- Alice Springs Hospital
- Royal Darwin Hospital

International Organisations

- Great Ormond St Hospital, London UK
- Hospital for Sick Children, Toronto Canada
- Oakland Childrens Hospital, USA
- Westkids, Auckland NZ

International Network of Paediatric Surveillance Units (INoPSU)

- British Paediatric Surveillance Unit
- Canadian Paediatric Surveillance Programme
- Cyprus, Greece Paediatric Surveillance Unit
- German Paediatric Surveillance Unit
- Latvian Paediatric Surveillance Unit
- Malaysian Paediatric Surveillance Unit
- Netherlands Paediatric Surveillance Unit
- New Zealand Paediatric Surveillance Unit
- Papua New Guinea Paediatric Surveillance Unit
- Portuguese Paediatric Surveillance Unit
- Swiss Paediatric Surveillance Unit
- Trinidad and Tobago Paediatric Surveillance Unit
- Republic of Ireland Paediatric Surveillance Unit
- Welsh Paediatric Surveillance Unit

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The Royal Australasian College of Physicians
Paediatrics & Child Health Division

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

The University of Sydney
AUSTRALIA

The Faculty of Medicine 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 children's hospital at Westmead

The Children's Hospital at Westmead, provides office space and infrastructure support for the APSU.

Healthway, WA

A Healthway health promotion research grant contributes towards the surveillance of Fetal Alcohol Syndrome, which was initiated in 2001.

Roche

Roche Products Pty Ltd sponsors the ongoing surveillance of Vitamin K deficiency bleeding with an annual grant.

MOUNT MAJURA
VINEYARD

Majura Wines has generously sponsored the APSU wine draw prize since 2002.

Financial supporters for individual studies include:

- Acute flaccid paralysis: Department of Health & Ageing
- Adverse effects from complementary and alternative medicine: Women's and Children's Health, The Royal Women's Hospital and The Royal Children's Hospital, Melbourne
- Congenital cytomegalovirus infection: Virology Division, Department of Microbiology, South Eastern Area Laboratory Service, Sydney Children's Hospital
- Early onset eating disorders: Centre for Prevention of Psychological Problems in Children, The Children's Hospital at Westmead
- Fetal alcohol syndrome: HealthWay WA and Telethon Institute for Child Health Research
- HIV/AIDS and perinatal exposure to HIV: National Centre in HIV Epidemiology and Clinical Research
- Neonatal herpes simplex virus infection: Department of Immunology and Infectious Diseases, The Children's Hospital at Westmead, Herpes Simplex Virus Research Laboratory
- Rett syndrome: Telethon Institute for Child Health Research, USA National Institutes of Health, Rett Syndrome Association of Australia
- Vitamin K deficiency bleeding: NSW Health, Roche.

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- Allen & Hanburys
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- Child Protection Unit, The Childrens Hospital at Westmead
- The Financial Markets Foundation for Children
- Glaxo Smith Kline
- Clive and Vera Ramaciotti Foundation (Perpetual Trustees)
- CSL Pharmaceuticals
- Davies Collison Cave Attorneys
- NSW Department of Health
- Nutricia Australasia
- Orlando Wines
- Paediatric Research Society of Australia and New Zealand.

The APSU

The Australian Paediatric Surveillance Unit (APSU) is a national resource, established in 1993 to facilitate active surveillance of uncommon childhood diseases, complications of common diseases or adverse effects of treatment. Diseases are chosen for their public health significance 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). For many childhood conditions, the APSU is the only national mechanism for data collection.

APSU has been used by over 160 individual researchers, to run 34 surveillance studies and has been influential in the development of international surveillance units. Currently there are 15 surveillance units worldwide (Table 8). 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 (Table 1).

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 activities of the APSU are funded in part by the Australian Government Department of Health and Ageing through their communicable diseases program, by the Faculty of Medicine, University of Sydney and through competitive research funding. The APSU Board oversees the management of the Unit and the APSU Scientific Review Panel evaluates applications to conduct studies through the Unit for suitability and scientific merit.

Aims:

To provide a national active surveillance mechanism that can be used to:

- study the epidemiology, clinical features, current management and short term outcomes of rare childhood conditions in Australia;
- respond to epidemiological emergencies such as outbreaks and emerging disease conditions.

To initiate and facilitate national collaborative research consistant with national child health priorities, including a 'healthy start to life' and to fill knowledge gaps.

To produce and disseminate evidence that will support:

- the development of effective educational strategies and clinical guidelines for clinicians;
- the development of appropriate prevention strategies and community awareness campaigns;
- and the development of evidence based policy.

Contributors to the APSU

Contributors to the APSU are clinicians working in paediatrics and child health throughout Australia. These are predominantly Fellows of the Division of Paediatrics and Child Health of the RACP, however other child health specialists including paediatric surgeons and child psychiatrists also participate in surveillance. Clinicians are identified through the Division of Paediatrics and Child Health RACP, the Australasian Association of Paediatric Surgeons and subspecialty interest groups. In 2004 an average of 1112 clinicians participated in monthly surveillance. Fifty four percent of clinicians were in general paediatric practice, 39% were subspecialists, 4.6% were paediatric surgeons and the remaining 2.4% practiced in child and adolescent psychiatry.

Operation of the APSU

Each month all contributing clinicians are asked to report children newly diagnosed with any of the conditions listed on the report card. Investigators conducting a study are informed weekly by the APSU of any new cases reported by APSU contributors. The investigator then sends a brief questionnaire to the clinician requesting further de-identified information. Investigators are responsible for collation, analysis and publication of this data (Figure 1), and report study findings annually to the APSU.

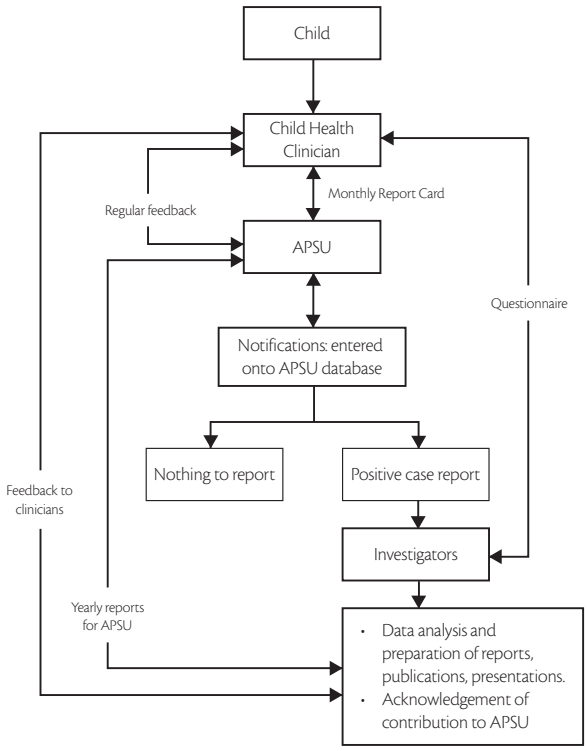

Figure 1. Operation of the APSU

Selection of conditions for study

Individuals or organisations may apply to study a condition through the APSU and applications undergo a process of peer review and revision before being listed on the monthly report card.

To satisfy the criteria for study a condition must:

1. be sufficiently uncommon so that the system is not over-burdened;
2. usually result in referral to a paediatrician or related specialist;
3. provide information that satisfies the study aims and that is not available from other sources.

Conditions are usually studied for one to three years, although provision for on-going study may be granted for diseases of public health significance or with very low incidence.

Conditions Studied

Between 1993 and 2004, the APSU monitored 34 uncommon childhood conditions. Some of the major findings of studies conducted through the APSU are documented in Table 1.

Case Classification

The APSU aims to estimate the incidence of selected conditions and to provide information which is representative of the Australian population. Maximal and unbiased case ascertainment is a high priority. Over-reporting rather than under-reporting of cases will help achieve this and duplicate reports are encouraged. Accurate classification of reported cases is facilitated by use of a unique identification code that is provided by clinicians on the study questionnaires. This enables investigators to identify duplicate reports.

To confirm a case, the clinical information reported in the questionnaire is used to ensure that the case definition criteria are met. The method of classifying cases is shown in Figure 2.

The APSU encourages Investigators to use multiple sources of case ascertainment where possible. This is particularly important in remote areas, where children have limited access to paediatricians and are often seen by general clinicians. Reported rates for conditions ascertained through the APSU therefore represent a minimum estimate of these conditions in the relevant Australian populations.

Table 1. Key findings of national surveillance studies conducted through the APSU 1993-2004

Conditions Under Surveillance	Commencement date	Key findings, implications and publications
Infectious / vaccine preventable including congenital infections		
Acute flaccid paralysis	Mar 1995	APSU reports via DoHA Polio Expert Committee to WHO and data contributed to 'polio-free' certification by WHO. Most (~70%) AFP cases are due to Guillain-Barre syndrome or transverse myelitis. All classified cases were non-polio AFP. Continued surveillance of polio is required in Australia, in view of recent reports of imported cases of wild poliovirus into Indonesia. (1,2)
Congenital cytomegalovirus infection	Jan 1999	APSU provides the only national data collection for cCMV. Observations of maternal and neonatal symptoms have increased understanding of phenomenology of cCMV. cCMV remains under-diagnosed. Although most cases are diagnosed by urine culture, use of PCR for urinary screening for cCMV may increase diagnostic yield. Universal neonatal hearing screening programs may also help identify new cases. (3)
Congenital varicella	Mar 1995-Dec 1997	Identified that birth defects also occur with 3rd trimester infection; pregnancies should be monitored and infants' eyes examined for visual impairment. (4)
Neonatal varicella infection	Mar 1995-Dec 1997	Early identification, treatment (acyclovir, Ig) recommended. (4-6)
Congenital rubella	May 1993	Women born in countries with poorly developed vaccination programs should have serological testing for rubella after arrival in Australia, and be vaccinated if appropriate. Travel to rubella endemic counties in the first trimester by women with no prior rubella immunity poses a risk to the fetus of congenital rubella (7)
Haemolytic uraemic syndrome	Jul 1994-Dec 2001	APSU study identified Shiga-toxin producing *E.coli* prevalent in Australia; provided national data during HUS outbreak and informed code of production for fermented meats. (8,9)
Hepatitis C virus infection	Jan 2003	APSU is monitoring an emerging disease of national significance. Most (>80) HCV infection in Australian children is acquired perinatally. Infants at risk were born to mothers who used IV drugs (~60%) or had invasive procedures or received blood overseas. Most HCV-infected children are clinically asymptomatic with mildly elevated liver function test at diagnosis. (10)
HIV/AIDS, Perinatal exposure to HIV	May 1993	APSU enhances mandatory reporting, identifies perintal exposure and maternal risks. Most cases of HIV are due to perinatal exposure. Fifty five percent of mothers were exposed to HIV through heterosexual contact in a high HIV prevalence country or in Australia with a partner from a high prevalence country. Thirty two percent used IV drugs or had a partner who used IV drugs. The transmission rate of infection has declined with increased use of interventions (including anti-retrovirals) in women diagnosed antenatally. (11)

Table 1 continued. Key findings of national surveillance studies conducted through the APSU 1993-2004

Conditions Under Surveillance	Commencement date	Key findings, implications and publications
Infectious / vaccine preventable including congenital infections		
Hospitalised pertussis in infancy	Jan 2001-Dec 2001	Identified adults as main source of infection and informed revision of immunisation schedule in 2003 to recommend vaccination of teenagers. Identified children less than 2 months at most risk and need for trials of early vaccination. (12)
Invasive *Haemophilus influenzae* infection	Jan 1998-Dec 2000	Confirmed success of Haemophilus influenzae Type B vaccination; influenced infection prevention policy. (13)
Kawasaki disease	May 1993-Jun 1995	Identified that young children may not fulfil international diagnostic criteria. (14)
Neonatal herpes simplex infection	Jan 1997	HSV type 1 identified as the cause of neonatal infection in 50% Australian cases. Many infants present without typical skin or mucosal lesions. Disseminated HSV infection may present with pneumonitis which requires early antiviral therapy. (15)
Non tuberculous mycobacterial infection	July 2004	Usually presents with lymphadenopathy in healthy children aged < 5 yrs. *Mycobacterium avium intracellulare* and *mycobacterium fortuitum* most commonly isolated. Relapse in 10% regardless of the medical therapy used.
Subacute sclerosing panencephalitis	Jan 1995-Dec 1998	Very rare, reflecting high uptake of measles vaccination. (16)
Congenital / genetic disorders		
Arthrogryposis multiplex congenita	Jan 1996-Dec 1998	Documented risk factors, informed development of new congenital classification and informed causal pathways. (16)
CHARGE association	Jan 2000-Dec 2002	Increased awareness of diagnostic criteria for CHARGE; diagnosis of 87% of cases in first year of life. (17)
Congenital adrenal hyperplasia	Aug 1995-Dec 1997	Enabled cross validation of potential neonatal screening program. (18)
Congenital & idiopathic nephrotic syndrome	Jul 1998-Jun 2001	Identified non-adherence to evidence-based management guidelines. (19)
Extrahepatic biliary atresia	May 1993-Dec 1996	Identified late diagnosis and need for education. Quantified transplantation needs. (20)
Fetal alcohol syndrome	Jan 2001- Dec 2004	Indigenous children over-represented; children often in foster care, have affected siblings. Informed causal pathways and educational strategies. (21,22)
Haemoglobinopathies	Jan 2004	The study aims to estimate incidence, types of Haemoglobinopathies and the distribution amongst ethnic groups.
Hirschsprung disease	Jan 1997-Dec 2000	Primary surgical procedure most used is Soave operation.(23)
Prader-Willi syndrome	Jan 1998-Dec 2000	First DNA confirmed estimate of birth prevalence. PWS often missed clinically in infants – education needed. (24)
Primary immunodeficiency	Jan 1997-Dec 1999	Documented numbers affected, need for immunotherapy and bone marrow transplant. (22)
Rett syndrome	May 1993-Apr 1995; Jan 2000	Enabled molecular epidemiological study of national cohort, phenotype/ genotype correlation; establishment of international database. (25-27)
Severe combined immunodeficiency	May 1995-Dec 2001	Confirmed good outcome bone marrow transplant. (17)
Mental health issue		
Childhood dementia	May 1993-Jun 1995	First national study worldwide. Clarified diagnostic criteria, identified large group with no identified cause. (28)
Childhood conversion disorder	Jan 2002-Dec 2003	First study to document the burden of illness in Australian children and to clarify psychosocial risk factors. (29)
Munchausen by proxy syndrome	Jan 2000-Dec 2003	First study to document impact of the diagnosis on clinicians; data informed development of management policy. (30)
Early onset eating disorder	Jul 2002	First national study of children <13 yrs. Contributing to debate on relevance of adult diagnostic (DSM) criteria to children. Simultaneous Canadian and British study. (29)

Table 1. Key findings of national surveillance conducted through the APSU 1993-2004

Conditions Under Surveillance	Commencement date	Key findings, implications and publications
Other injury/illness		
Anaphylaxis following food ingestion	Jul 2002-Dec 2003	Peanut most common cause; also other nuts, soy, shellfish implicated. (29)
Adverse effects with the use of complementary and alternative medicine	Jan 2001-Dec 2003	Sentinel adverse effects documented in infants and children range from mild to fatal. Dietary restrictions; use of CAM in pregnancy; and use in place of conventional medications pose significant risks. (31)
Near drowning	May 1993-Dec 1996	Neurological outcomes poor; age determines near drowning site; most commonly home pool. (32)
Vitamin K deficiency bleeding	May 1993	Monitoring disease during policy changes to vitamin K prophylaxis and universal use of new vitamin K preparation. (29)

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Valid

- A **confirmed case** is one confirmed by the investigator to satisfy the case definition criteria.
- A **probable case** is one that does not completely meet the case definition criteria but is highly probable on the basis of information available.

Invalid

- A **duplicate case** is one that has already been reported
- An **error** is a case that has been reported:
  1) which does not fulfil the case definition criteria, or
  2) has had the diagnosis revised by the referring clinician, or
  3) for which the APSU report card was ticked by mistake.

Unknown

insufficient follow-up information is available to the investigator or information is not received by APSU from the investigator.

Figure 2. Classification of reported cases

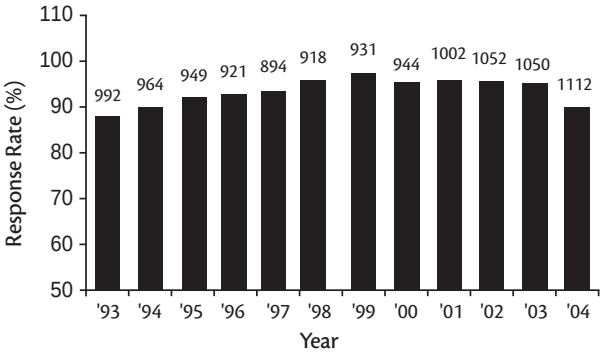

Figure 3. APSU mean monthly response rate (%) and average number of contributing clinicians 1993-2004

State	Response Rate	No. (%) of Reporting Clinicians	Prop (%) of Children < 15 yrs
ACT	95%	21 (2%)	1.5%
NSW	91%	448 (39%)	34%
NT	80%	11 (1%)	1%
QLD	90%	175 (15%)	20%
SA	89%	94 (8%)	7%
TAS	98%	15 (2%)	2.5%
VIC	88%	269 (24%)	24%
WA	88%	105 (9%)	10%

Table 2. Response rate; by state and territory; number of clinicians reporting to the APSU; and proportion of children < 15yrs of age.

Response Rates

In 2004, 1112 clinicians participated in monthly surveillance of 13 conditions, with an overall response rate of 94% (Figure 3). This maintains the excellent participation level of contributing clinicians since APSU's inception in 1993. Reporting by e-mail was introduced in February 2001. In 2004, 55% of clinicians reported by e-mail.

NSW has the greatest proportion of children (34%), Victoria has 24% and Queensland 20%. Correspondingly, NSW has the greatest proportion of participating clinicians (39%), Victoria (24%) and Queensland (15%) (Table 2).

Respondent workload

During 2004 the majority of clinicians (82%) had no cases to report. Twelve percent of clinicians reported one case, 4% reported two cases and 2% reported 3 or more cases.

Summary of surveillance study results 2004

Incidence rates represent the number of newly diagnosed cases of disease in a defined population over a defined period of time. Only children seen by child health specialists are represented in APSU data. Despite the fact that the conditions surveyed are highly likely to be referred to specialists; that the monthly reporting rate is high; and that clinical data are obtained for most cases reported; 100% case ascertainment is unlikely to be achieved by any one surveillance scheme. Thus, where available, cases notified to investigators through other means are included in the total number of cases. In this report the '*reported rate of disease*' represents an estimate of minimum incidence. Reported rate of disease 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 aged 5 years and under or aged 15 years and under per annum. Population figures for the denominator are obtained from the Australian Bureau of Statistics[1].

Table 3 shows reported rates of disease to December 2004, for conditions studied through the APSU. For conditions where case ascertainment has also occurred through complementary sources, (including Perinatal exposure to HIV, Acute flaccid paralysis, Haemolytic uraemic syndrome and Rett syndrome) cases ascertained from all sources for the study period are presented.

[1] Austats: *Population by age and sex, Australian states and territories. Australian Bureau of Statistics 2004: 3201.0*

Table 3. Summary of results, number of cases and annual reported rate for studies conducted to December 2004

Conditions Under Surveillance	Commencement date	Duration of study (years)	Total confirmed cases	Reported Rate [a b c d]
Infectious / vaccine preventable				
Acute flaccid paralysis	Mar 1995	9.75	338	0.87[b]
Congenital cytomegalovirus infection	Jan 1999	6	48	3.85[a]
Congenital rubella (with defects)	May 1993	11.5	50	0.11[b]
Hepatitis C virus infection	Jan 2003	2	24	0.3[b]
Perinatal exposure to HIV (Birth Prev)	May 1993	11.5	253	8.37[d]
Perinatal infection after exposure to HIV (Birth Prev)			39	1.29[d]
Neonatal herpes simplex virus infection	Jan 1997	8	71	4.1[a]
Non tuberculous mycobacterial infection	July 2004	0.5	20	*
Congenital / genetic disorders				
Fetal alcohol syndrome	Jan 2001	4	76	0.48[b]
Rett syndrome	Jan 1993	12	276	0.88[c]
Haemoglobinopathies	Jan 2004	1	24	0.60[b]
Mental health issues				
Early onset eating disorder	Jul 2002	2.5		*
Other injury / illness				
Adverse events associated with the use of				
Complementary and alternative medicine	Jan 2001	4	38	0.24[b]
Vitamin K deficiency bleeding	May 1993	11.5	29	0.99[a]

* Rate for Non-Tuberculous mycobacterial infection are not calculated as only 6 months of surveillance is completed.
Rate for Early onset eating disorder not calculated. Refer to page 22.

a Reported rate per 100,000 live births

b Reported rate per 100,000 children <15 years

c Reported prevalence per 100,000, in December 2004, in Australian born females, aged 5-18yrs. (includes incident cases ascertained through APSU and prevalent cases through the Rett Syndrome Association of Australia and other sources, since 1993)

d Reported birth prevalence (95% CI) per 100,000 live births

ACUTE FLACCID PARALYSIS (AFP)

Study Highlights

- In 2004 Australia exceeded the WHO AFP surveillance target of 1 case 100,000 children aged <15 years per annum.
- The majority (~70%) of AFP cases are due to Guillain-Barre syndrome or transverse myelitis. All cases classified by the Polio Expert Committee were non-polio AFP [1].
- Continued surveillance is required to keep Australia polio free, especially in view of recent reports of imported cases of wild poliovirus into Indonesia.

Background

In 2004, AFP surveillance continued to be co-ordinated by staff at the National Polio Reference Laboratory (NPRL) in collaboration with the APSU. With the importation of wild polioviruses into six "polio free" countries during 2004 the WHO polio eradication program is facing immense challenges. Australia and other countries certified free of circulating wild poliovirus need to continue a sensitive surveillance system for AFP cases and timely laboratory testing of faecal specimens from cases of AFP.

Objectives

- To determine the notification rate of AFP in children aged <15 years of age in Australia;
- To determine whether AFP is caused by poliovirus infection and, if so, whether it is a wild, vaccine or vaccine-derived strain of poliovirus;
- To determine other causes, and the clinical picture, of AFP in Australia.

Case Definition

Any child resident in Australia and aged <15 years with acute onset of flaccid paralysis in one or more limbs or acute onset of bulbar paralysis.

Results

Since 1995 there have been 535 notifications of AFP (338 confirmed cases and 58 unclassified cases) (Figure 4). In 2004, 45 cases from 62 notifications were classified by the Polio Expert Committee (PEC) as non-polio AFP (19 APSU, 26 Victorian Infectious Disease Reference Lab VIDRL). Of the 62 notifications 58 (94%) had clinical information. There were 9 duplicate notifications, 4 errors and 4 unclassified cases. Twenty four cases were reported in NSW (23 non-polio AFP, 1 unclassified), 9 each in QLD and Victoria, 3 in SA (2 non-polio AFP, 1 unclassified) 1 each in Tasmania and NT and 2 unclassified cases in WA. All Australian states except for Western Australia, Australian Capital Territory and Victoria reached or exceeded the WHO target rate. Paediatricians in Victoria notified 0.9 cases per 100,000 children the highest rate the state has achieved since the introduction of AFP surveillance in 1995.

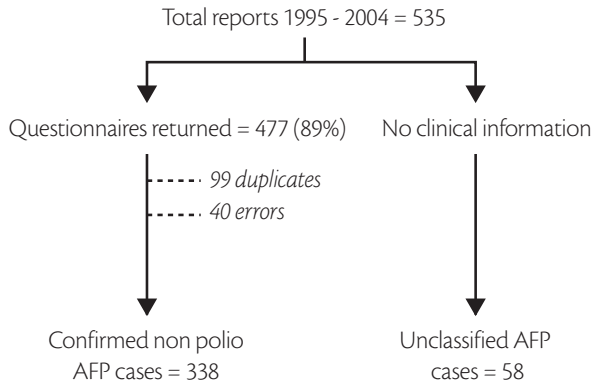

Figure 4. AFP surveillance data summary 1995-2004

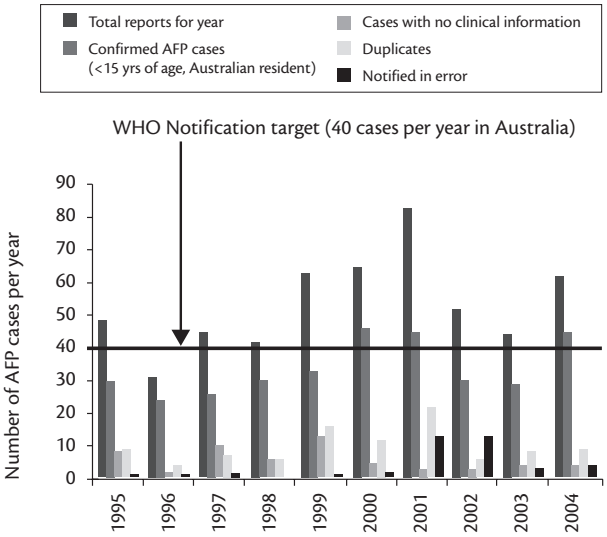

Figure 5. WHO notification target 1995-2004.

In 2004, Australia reached the WHO expected target for non-polio AFP in a non-endemic country for the third time since 1995 (Figure 5), confirming that the disease incidence is at least one case per 100,000 children aged less than 15 years. However only 40% of cases had faecal specimens collected within 14 days of onset of paralysis, below the 80% target level identified by WHO.

Direct notifications to the NPRL are encouraged and specific instructions and contact details for the NPRL are included on the monthly APSU report card. This strategy aims to increase the

number of cases with adequate faecal specimens according to the WHO protocol. Of the 18 cases with adequate faecal specimens in 2004, 16 were notified directly to the NPRL. Forty four percent of cases classified as non-polio AFP by the PEC were diagnosed as Gullian Barre Syndrome. A poliovirus type 1 isolated from an AFP case gave discordant intratypic differentiation test results, and was subsequently sequenced with 99.7% homology to the parental strain of Sabin vaccine. The case was classified as non-polio AFP and diagnosed as infant botulism by the polio expert committee (PEC).

References referred to in text:

1. Morris AMS, Elliott EJ, D'Souza RM, Antony J, Kennett M, Longbottom H. Acute Flaccid paralysis in Australian children. *Journal of Paediatrics and Child Health*, 2003; 39:22-26.

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ADVERSE EFFECTS FROM COMPLEMENTARY ALTERNATIVE MEDICINES (CAM) FINAL REPORT

Study Highlights

- CAM have the potential for adverse effects ranging from mild to fatal.
- Clinicians need to be particularly aware of the dangers associated with dietary restriction, use of CAM in pregnancy and the use of CAM, in place of conventional medication.
- CAM have potential risks in overdose and there is a need for safe storage/child resistant packaging.

Background

CAM are commonly used in Australia to treat children with both common and serious or chronic conditions. CAM are often used because they are perceived to be natural and therefore harmless. However, like all treatments there exists the potential for adverse effects. There are currently limited data about CAM-associated adverse events. Given the diversity of CAM, the systematic collection of adverse event data is problematic. APSU provided a means of collecting CAM-related adverse events data nationally.

Objectives

- To obtain data on major adverse events associated with the use of CAM in children in Australia;
- To develop information for paediatricians and other health practitioners about specific adverse events associated with the use of particular forms of CAM.

Case Definition

The occurrence of any adverse event, suspected or confirmed, associated with the use of CAM, occurring in children up to 15 years of age, where:

- An **"adverse event"** is any unfavourable and unintended sign (including an abnormal laboratory finding), symptom or disease associated with the use of CAM, whether or not it is confirmed to be related to the therapy.
- **"Complementary or Alternative Medicine (CAM)"** includes any health care practice other than one intrinsic to the current conventional system.

Severity of adverse events are classified as follows:

- **Mild** – an adverse experience which is easily tolerated by the patient, causing minimal discomfort and not interfering with everyday activities (eg. a minor rash).
- **Moderate** – an adverse experience, which is sufficiently discomforting to interfere with normal everyday activities (eg. nausea and vomiting requiring time away from school).
- **Severe** – an adverse experience which is incapacitating and prevents normal everyday activities and/or requires therapeutic intervention such as the use of a prescription drug or hospitalisation.
- **Life threatening** – the patient is perceived to be at risk of death from the event as it occurred (eg. an anaphylactic reaction).
- **Fatal** – the patient died.

Study results and conclusions

The 39 reports to the CAM study can be broadly categorised into:

- Adverse events associated with a failure to use conventional therapy (Table 4).
- Adverse events associated with the use of medicinal CAM (Table 5).

Table 4. Adverse events associated with the failure to use conventional therapies.

CAM	ADVERSE EVENTS
CAM used instead of anticonvulsants	Life threatening seizures
Multiple CAM treatments	Delayed management of cerebral palsy
Chiropractic treatment	Delayed diagnosis of UTI in an irritable infant
Naturopathy for diabetes	Symptomatic hyperglycaemia and change to insulin dose
Failure to immunise in favour of CAM	Hib pneumonia
Failure to use anticoagulant in favour of CAM	Lung infarction

(includes multiple cases of CAM)

Table 5. Adverse events associated with the use of medicinal CAM

CAM	ADVERSE EVENTS
Homeopathy and dietary restriction	Malnutrition, sepsis and death
Homeopathic medicines*	Seizure and apnoea
Herbal remedy for vomiting*	Liver failure requiring liver transplant
Herbal medicines*	Intra-operative bleeding
Diet and fluid restriction	Dehydration, encephalopathy and refeeding syndrome
Alternative medical practitioner prescribed triiodothyronine for obesity	Admission with thyrotoxicosis
IM injections of vitamins daily	Left sciatic neuropraxia
Oral and IV calcium supplements from alternative practitioner	Severe hypercalcaemia
Ginkgo and brahmi overdose	Admitted for observation
Food supplement with taurine and inositol	Vomiting requiring IV rehydration
Homeopathy (plus atilla) for cough	Vomiting, drowsiness and fever
Homeopathy for dietary restriction	Malnutrition with oedema
High dose Vitamin B	Carotenaemia
Rice milk for constipation	Failure to thrive
Vitamin mixture	Allergic reaction
Chamomile tea in excessive amounts	Worsening constipation
Herbal medicine adulterated with steroid	Steroid excess

* Mechanism unknown.

(includes multiple cases of CAM)

Between 2001 and 2004 there were 46 notifications of CAM and 39 confirmed cases (Figure 6). The geographical distribution of the confirmed cases is presented in Figure 7.

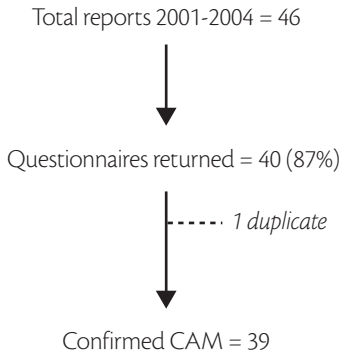

Figure 6. CAM surveillance data summary 2001-2004

Figure 7. Distribution of confirmed CAM cases 2001-2004

The severity of adverse events ranged from mild to fatal. Of particular concern were those adverse events associated with significant dietary restriction. Any therapy that advocates dietary restriction is potentially extremely dangerous. Other identified areas for concern have included CAM use in pregnancy and the potential fetal effects; risks associated with accidental ingestions; and potential risks in some circumstances of changes to conventional therapy in favour of a CAM therapy.

The number of reports is likely to be an underestimation of the actual number of events occurring. This may be due to paediatricians failing to report events or not recognising them; events may be detected by other health care workers such as general practitioners; or families may not disclose CAM use or present for review.

Study Investigators

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CONGENITAL CYTOMEGALOVIRUS INFECTION (cCMV)

Study Highlights

- This is the first national study of cCMV in Australia, a major infectious cause of malformations.
- cCMV infection was associated with maternal illness in only 8/24 cases in 2004 and should be considered regardless of maternal history.
- cCMV probably remains under-diagnosed. Although most cases are diagnosed by urine culture, use of PCR for urinary screening for CMV may increase diagnostic yield [1].
- Universal neonatal hearing screening programs may also increase identification of new cCMV cases.

Background

Congenital Cytomegalovirus Infection (cCMV) is a major infectious cause of malformation in Australian children. Until collection of the APSU data there was no national data for Australia. Previous limited studies had suggested rates of symptomatic cCMV of 0.5-1.5%, and were limited to single states. The current study updates these estimates, and has provided the basis for ongoing surveillance and further research into mechanisms of vertical viral transmission. Observations regarding maternal and neonatal symptoms have added to our understanding of the phenomenology of this congenital infection.

Objectives

The study aims:

- To determine the incidence of cCMV and suspected cCMV in Australian children;
- To determine the presenting features and clinical spectrum of disease due to cCMV;
- To determine the current therapy in use for cCMV infection;
- To determine the epidemiology of cCMV including prevalent cCMV subtypes prior to trials of vaccines and antivirals.

Case Definition

Definite congenital cCMV infection

- Any child from whom cCMV is isolated in the first three weeks of life, from urine, blood, saliva, or any tissue taken at biopsy.

Suspected congenital cCMV infection

- Any child up to 12 months of age, in whom cCMV 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 cCMV infection.

Study results and conclusions

Since 1999 there have been 48 children with definite and 58 children with suspected cCMV infection identified (Figure 8). In 2004 there were 36 notifications of cCMV. Of these, 17 children had definite cCMV and 7 had suspected cCMV. There were 11 definite or suspected cases in NSW, 5 each in QLD and WA, 3 in SA and none in NT, ACT, VIC or TAS.

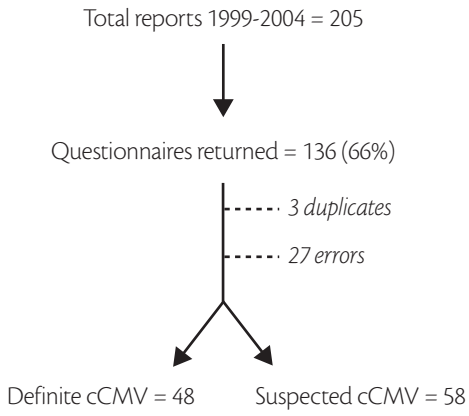

Figure 8. cCMV surveillance data summary 1999-2004

All symptomatic infants with cCMV presented at less than one week of life. The most common presenting features were splenomegaly, hepatomegaly, thrombocytopenia, hepatitis, jaundice and thrombocytopenia. Anaemia and low weight for gestational age were less frequent signs. Encephalitis, microcephaly and intracranial calcifications were presenting signs in three children. No seizures, cataracts or microphthalmia were reported in any case. Importantly, five of the seven probable cases of cCMV from NSW through this program where universal neonatal hearing screening has been introduced, were diagnosed at one to six months of age.

Evidence of seroconversion was documented in three mothers whose neonates were infected and asymptomatic. We would expect much higher numbers of asymptomatic but infected infants and hence it is likely a significant number of children are

not recognized. This may reflect maternal screening practices. Two notifications of cCMV were documented on the basis of paternal cCMV infection, with no maternal illness noted -one child was asymptomatic and the other was noted to have chorioretinitis.

We have previously noted high rates of maternal illness in both suspected and definite cCMV cases. In 2004 maternal illness was noted in only eight of the 24 cases – one of these from the suspected cases and seven from the definite infection group.

Urine culture continues to be the most common diagnostic test for cCMV with increasing use of urine CMV PCR. At the time of reporting no child had received anti viral treatment for cCMV.

In conclusion it is unlikely that cases are underreported. Laboratory diagnoses could be increased by the use of urinary screening for cCMV, using PCR. It will be interesting to determine the contribution of neonatal hearing screening programs to diagnosis of cCMV.

References referred to in text:

1. Trincado DE, Rawlinson WD. Congenital and perinatal infections with cytomegalovirus. *Journal of Paediatrics and Child Health*, 2001; 37:187-192.

Study Investigators

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CONGENITAL RUBELLA

Study Highlights

- The only reported case in 2004 was born to an unvaccinated woman born overseas. We have previously documented that this group is 'at risk'[1].
- Women born in countries with poorly developed vaccination programs should have serological testing for rubella after arrival in Australia, and vaccination when appropriate.
- Travel to rubella endemic counties in the first trimester by women with no prior rubella immunity poses a risk to the fetus of congenital rubella.

Background

Maternal rubella infection in the first trimester can result in Congenital rubella syndrome in the fetus, characterised by deafness, cataracts, growth retardation, mental handicap and cardiac abnormalities. While reinfection with rubella can occur, prior maternal immunity (by natural infection or effective rubella vaccination) usually protects against fetal damage caused by the virus. The rash of rubella is non specific. Pregnant women with a rash of unknown cause or history of exposure to rubella should have their rubella titre checked irrespective of a history of prior vaccination or past documentation of rubella antibody.

Objectives

- To document the incidence of congenital rubella infection;
- To determine the vaccination status of mothers of affected infants;
- To monitor the effectiveness of the current vaccination program.

Case Definition

Any newly diagnosed 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.

Study results and conclusions

There have been 106 notifications of Congenital rubella since 1993 (Figure 9), including 3 notifications of Congenital rubella infection in 2004. Clinical data were available for two of these and 1 was a duplicate, leaving 1 case. The 1 confirmed case was from NSW. The infant notified in 2004 was born to a woman who was born outside Australia and had not been vaccinated against rubella. The mother developed a rubella-like illness with rash while in Indonesia in the first months of pregnancy. The infant was born with bilateral deafness; intrauterine growth retardation, bilateral cataracts, and a patent ductus arteriosus.

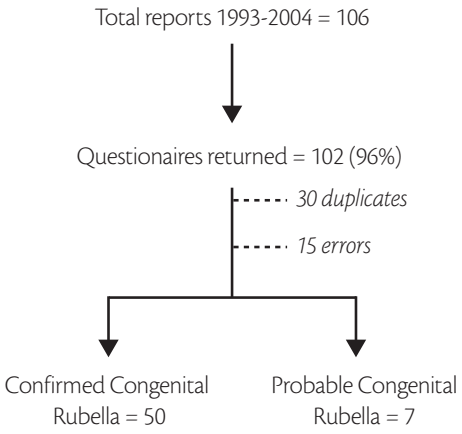

Figure 9. Congenital rubella surveillance data summary 1993-2004

As previously reported, offspring of women born outside Australia in countries with poorly developed vaccination programs have the highest risk of being born with congenital rubella syndrome in this country.

References referred to in text:

1. Forrest J, Donovan T, Burgess M. A resurgence of congenital rubella in Australia? *Communicable Diseases Intelligence*, 2003; 27:533-535.

Study Investigators

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EARLY ONSET EATING DISORDER (EOED)

Study Highlights

- Children as young as five may present with determined food avoidance and display the cognitive symptoms of an eating disorder listed in DSM-IV diagnostic criteria
- In addition to weight loss or failure to gain weight, many children with early onset eating disorder have significant medical and/or co-morbid physical and/or mental health problems.

Background

Epidemiological studies suggest that the incidence of eating disorders, including anorexia nervosa, has been increasing in adolescents over the last 50 years. However, there are few available estimates of incidence of eating disorders in children under 13 years of age. In addition to food avoidance and weight loss (or failure to gain weight), current DSM-IV criteria for anorexia nervosa require that the patient has concerns about his/her body weight and there is disturbed body image and fear of weight gain. However, these criteria may not accurately reflect the clinical features in young children. The objective of this study was to identify the incidence of early onset eating disorders (EOED) seen by child health specialists in Australia and to describe the range of clinical features at presentation.

Objectives

- To estimate the incidence of EOED seen by child health specialists in Australia;
- To describe the epidemiology of EOED;
- To describe the range of clinical features at presentation, including other psychiatric illness;
- To compare the features at presentation in this population with current DSM IV criteria;
- To describe the acute medical complications experienced by children with EOED;
- To describe the therapeutic interventions used in management;
- To contribute data to an international comparison of the diagnosis and management of EOED.

Case Definition

Children aged 5-13 years inclusive, newly diagnosed with an EOED, defined as:

- Determined food avoidance AND
- Weight loss or failure to gain weight during a period of growth, not due to any identifiable organic cause AND
- Child is admitted to hospital OR managed within the community.

Study results and conclusions

The classification of the 152 reports of EOED made to the APSU between July 2002 and December 2004 is presented in Figure 10. Of the 84 confirmed cases, 84% were female and 17% were under 10 years of age. In 2004 there were 39 children with confirmed EOED, 16 in Victoria, 13 in NSW, 6 in QLD, 2 in SA, 1 each in WA and TAS and none in NT.

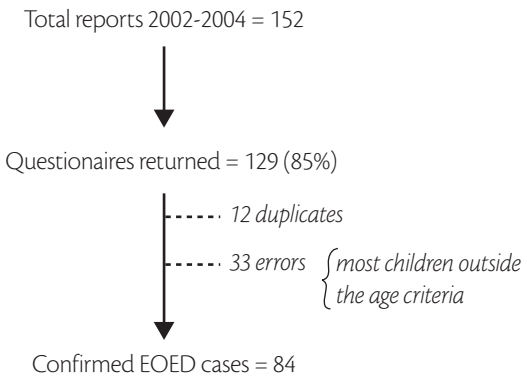

Figure 10. EOED Surveillance data summary 2002-2004

The median duration of eating disorder symptoms prior to presentation was 13 months (Range: 1-156 months). In the six months prior to diagnosis eight children (9%) failed to gain any weight and decreased weight was observed in 79% of cases. Median weight loss was 5 kg (Range: 0.2 to 38 kg). Twelve of the 14 girls who had reached menarche had secondary amenorrhoea. Clinical features are described in Table 6.

Sixty percent of Australian children with EOED also had a concurrent mental health concern, most commonly anxiety in 32/84 (38%).

Table 6. Clinical features at presentation in Australian children with early onset eating disorders.

SYMPTOMS at PRESENTATION	%
Food Avoidance	100
Preoccupation with food	91
Denial of severity	85
Weight loss	79
Fear of weight gain	71
Preoccupation with weight	69
Misperception body shape	62
Excessive exercise	54
Self-induced vomiting	12
Diuretic or laxative abuse	0
CLINICAL SIGNS AT PRESENTATION	
Bradycardia (<36 beats/min)	42
Temperature (<35.5ºC)	31
Hypotension (systolic BP<80)	18

Seventy six percent of the children notified were hospitalised and the mean duration of hospitalisation was 23 days (range 2-75 days).

These preliminary findings indicate that while not all children reported with EOED meet full DSM-IV criteria for anorexia or bulimia nervosa, some children as young as 5 years of age will display cognitive symptoms consistent with these criteria and many are at significant biological and/or psychological risk.

Study investigators

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FETAL ALCOHOL SYNDROME (FAS) FINAL REPORT

Study Highlights

- Fetal alcohol syndrome (FAS) continues to be diagnosed in Australia. Children with FAS have a wide range of medical, psychological and behavioural problems and are demanding of health, education and community resources.
- Children with FAS are often in foster care, are born to mothers with multiple substance use and have similarly affected siblings. Over 60% of children reported to APSU were identified as Indigenous.
- Data from this study have been requested by decision making bodies including the Intergovernmental Committee on Drugs and the Ministerial Council on Drug Strategy; and have been disseminated in the media and through educational sources.

Background

FAS is caused by maternal alcohol consumption during early pregnancy and represents the most severe effects of exposure to alcohol *in utero*. Children with FAS display a wide range of physical defects and disabilities, however the cardinal features are: minor cranio-facial abnormalities; prenatal and/or postnatal growth deficiency; and evidence of damage to or dysfunction of the central nervous system. Data from the Western Australian Birth Defects Registry suggest a birth prevalence for FAS of 0.18 per 1,000 live births (0.02 per 1,000 non-Indigenous live births; 2.76 per 1,000 Indigenous live births)[1].

Objectives

- To estimate the incidence of newly diagnosed FAS seen by child health specialists in Australia;
- To describe the epidemiology of FAS (gender, age, geography, ethnicity, SES);
- To describe the clinical features of FAS including developmental and physical co-morbidity;
- To describe use of health services by children with FAS;
- To document the use of other harmful substances by mothers of children with FAS;
- To increase clinicians' awareness of FAS by providing information on clinical features and diagnostic criteria.

Case Definition – consistent with the Institute of Medicine[2]

Clinicians were asked to report any child aged <15 years with newly diagnosed:

1. Fetal Alcohol Syndrome – alcohol exposure confirmed, defined as:
   - Evidence of prenatal alcohol exposure AND
   - All characteristic cranio-facial abnormalities AND
   - Pre-natal or post-natal growth deficiency AND
   - Structural abnormalities or dysfunction of the CNS
2. Partial Fetal Alcohol Syndrome – alcohol exposure confirmed, defined as:
   - Evidence of prenatal alcohol exposure AND
   - All characteristic cranio-facial abnormalities AND
   - Pre/post natal growth deficiency OR
   - Some characteristic cranio-facial abnormalities AND
   - Structural abnormalities or dysfunction of the CNS
3. Suspected Fetal Alcohol Syndrome – alcohol exposure not confirmed, defined as:
   - All characteristic cranio-facial abnormalities AND
   - Pre/post natal growth deficiency AND
   - Structural abnormalities or dysfunction of the CNS

Study results and conclusions

Since 2001, 76 reported cases met the definition for FAS, suspected FAS or partial FAS used in this study[2] (Figure 11). The median age at the time of diagnosis was 2.8 years (range newborn to 12 years), 51% were male, and 61% were identified as Indigenous. Only 42% of these children were living with their biological

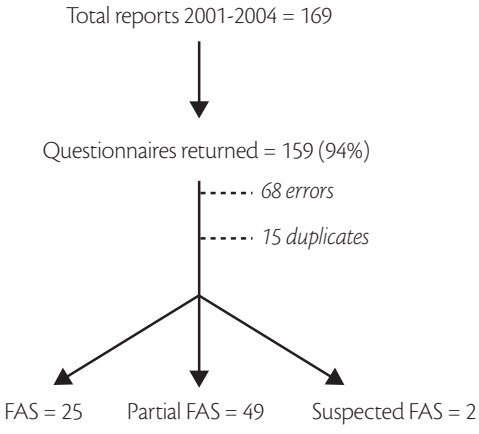

Figure 11. FAS surveillance data summary 2001-2004

parent(s), 17% lived with grandparents or other relatives, and 40% were adopted or fostered. Seventy six percent of children with FAS were exposed to other substances *in utero* including nicotine (65%) and marijuana (25%). All children had been referred to one or more health related agencies including specialty paediatric services (78%), child development services (49%), department of community services (67%) and remedial education services (30%).

From 55 notifications in 2004, 22 children with confirmed FAS, partial FAS or suspected FAS, were identified. There were 9 cases in QLD, 6 in NSW, 3 in NT, 2 in WA, 1 each in SA, and ACT and none in Victoria and TAS. The remaining cases did not fulfil the case definition or were duplicate notifications.

The APSU study of FAS has provided valuable descriptive data and an estimate of the number of children under 15 years of age with newly diagnosed FAS seen by paediatricians between 2001-2004. APSU data show that FAS contributes to significant social, medical and educational burdens for affected children, families and the community.

Data collection for this study is now finalised. We wish to thank clinicians who notified cases of FAS and provided us with clinical and other information.

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1. Bower C, Silva D, Henderson TR, Ryan A, Rudy E. Completeness of ascertainment: the effect of adding a new source of data. *Journal of Paediatrics and Child Health 2000;* 36:574-576.
2. Stratton K, Howe C, Battaglia F. *Fetal alcohol syndrome: Diagnosis, epidemiology, prevention and treatment.* National Academic Press, Washington, 1996.

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HAEMOGLOBINOPATHIES

Study Highlights

- The number of children with Haemoglobinopathies in Australia is unknown.
- Current screening practices in Australia may be inadequate to detect carrier individuals.
- This study aims to identify the type of haemoglobinopathies seen in Australia and their distribution among ethnic groups, to produce evidence to support expanded screening for haemoglobinopathies, and to facilitate prevention.

Background

Haemoglobinopathies are recessively inherited blood disorders for which there is usually no cure, except under certain circumstances, such as bone marrow transplantation. While haemoglobinopathies are rare, the frequency of the carrier states in certain populations is high (eg. for ß-thalassaemia, the carrier rate is 1 in 5 in Greece and Italy; for sickle cell disease, the rate is 1 in 5 in equatorial Africa). Alpha thalassaemia and Haemoglobin E carrier status are common amongst Asian populations.

Given the changing composition of the Australian population the number of cases of haemoglobinopathies may be increasing, however there are no national data to support this[1]. Conversely, the incidence of these disorders may be decreasing overseas because of widespread screening programs[2].

In all states of Australia, selective screening is the current policy to identify carriers of haemoglobinopathies.[3] Thalassaemia carrier testing is recommended on an ad hoc basis to individuals from high-risk ethnic groups during the teenage and early adult years. There are many individuals in Australia who may not be recognised as being from high-risk ethnic groups (such as second or third generation Southern Europeans). Consequently they may be missed by targeted screening programs and unaware of their carrier status or the potential risks of these conditions for their children.

Carriers can also be detected on routine blood films carried out in early pregnancy. Screening with a full blood examination alone will not detect carriers of sickle cell disease and haemoglobin electrophoresis is necessary.

Study Objectives

In this study we seek to estimate the incidence and types of haemoglobinopathies seen by Australian paediatricians and their distribution amongst ethnic groups. We also collect information on the timing and method of diagnosis of haemoglobinopathies in Australia. This information will inform efforts to improve disease detection and outcomes for affected children.

Case definition

All children under 15 years of age seen in the previous month with a newly diagnosed haemoglobinopathy including:

- structural haemoglobin abnormalities resulting from changes in the amino acid sequence of the globin chains
- thalassaemias, in which the synthesis of one or more of the globin chains is decreased or totally suppressed.

Conditions to be reported include:

- Hb SS disease (sickle cell anaemia)
- Hb CC disease
- Hb EE disease
- ß-thalassaemia major
- Hb E/ ß-thalassaemia
- Hb S/ ß-thalassaemia
- Hb SC disease
- Hb H disease
- Hb Barts disease
- Other rarer, severe haemoglobin variants

Study results and conclusions

The classification of the 45 notifications in 2004 is indicated in Figure 12. There were 24 confirmed cases; 11 in NSW, 2 each in Victoria and QLD, 1 each in ACT and SA, 7 in WA and none in NT or TAS.

Of the confirmed cases 15 were Australian born, 4 were diagnosed with ß-thalassaemia major, 2 with Hb E/ ß-thalassaemia, 6 with Hb H disease, 1 Hb S/ ß-thalassaemia, 1 HbE/alpha thalassaemia, 1 Hb Zurich disease and 9 with Hb SS disease. One child with diagnosed Hb SS died. This study will continue in 2005.

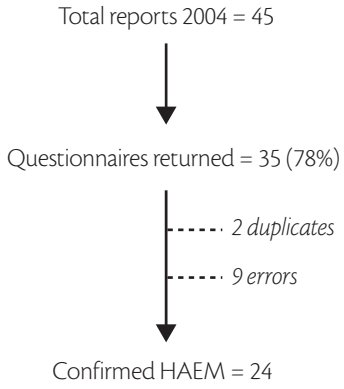

Figure 12. Haemoglobinopathies surveillance data summary 2004

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1. Guidelines for diagnosis and management of haemoglobinopathies. NSW Health Department Circular August 1999; 98/4807-5.
2. Cao A, Galanello R, Rosatelli MC. Prenatal diagnosis and screening of the haemoglobinopathies. Baillieres Clinical Haematology 1998;11(1): 215-38.
3. National Public Health Partnership. An overview of public health surveillance of genetic disorders and mapping of current genetic screening services in Australia. National Public Health Partnership Report October 2002

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HEPATITIS C VIRUS INFECTION (HCV)

Study Highlights

- Most HCV infection in Australian children is perinatally acquired. Children most at risk are those born to women with one or more maternal risk factors, including intravenous drug use, tattooing, body piercing, needle sharing, or receipt of blood products or invasive procedures, either overseas or before 1990 in Australia.
- The reported number of infected children is lower than predicted by Federal de-identified laboratory notifications. This may be a result of under-diagnosis and/or under-reporting and variable maternal screening practices.
- Most HCV infected children are clinically asymptomatic with mildly elevated liver function tests at diagnosis.

Background

Over 170 million people worldwide and an estimated 1.1% of adults in Australia are infected with Hepatitis C virus (HCV). The incidence of HCV infection: in children is unknown. The most common route of childhood HCV infection is by vertical transmission from mother to child. This occurs almost exclusively in pregnant women with HCV viraemia. The transmission rate from a mother who is HCV antibody and RNA positive during pregnancy, to her child is estimated at 5%. HIV co-infection during pregnancy further increases the risk of vertical HCV transmission up to 20%.

Objectives

- To determine the reported incidence of newly diagnosed HCV infection in Australian children;
- To describe the clinical presentation, investigation and management of newly diagnosed HCV infection;
- To document the presence of known risk factors for HCV infection;
- To determine the prevalence of co-infection with Hepatitis B Virus (HBV) and/or Human Immunodeficiency Virus (HIV) in Australian children with newly diagnosed HCV infection.

Case Definition

Any child <15 years of age with newly diagnosed hepatitis C virus infection, defined as:

- at least one confirmed positive anti-HCV antibody test performed at age ≥ 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.

Study results and conclusions

Since 2003, 57 notifications have been received by the APSU and 24 cases of HCV infection confirmed (Figure 13). In 2004, there were 12 confirmed cases of HCV. Four cases were from Victoria, 3 each in QLD and NSW, 2 in WA and none in NT, SA, TAS and ACT.

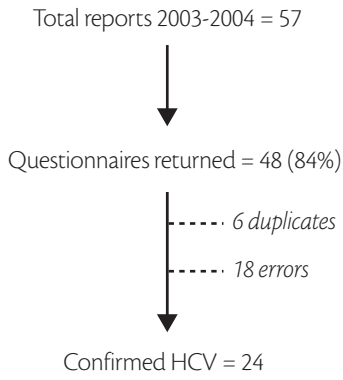

Figure 13. HCV surveillance data summary 2003-2004

Of the 24 confirmed cases of HCV reported since the study commenced, most children were born in Australia (67%) to an HCV-infected mother (83%). Maternal risk factors for HCV infection included maternal IV drug use in 15 (63%); invasive procedures in 5 (21%), tattoos in 7 (29%). Some mothers had more than one risk factor recorded. One woman received a vaccination and another underwent home electrolysis in an HCV endemic country.

Other childhood risk factors for HCV included IV drug use (3/24) and parenteral exposure to HCV in a high prevalence country (1/24). Of the 3 children with documented IV drug use, 2 had HCV negative mothers, and the HCV status of the other child's mother was unknown.

The median age of children at diagnosis was 5.3 years (range 1m-15y); 25% of children were diagnosed at less than 2 years of age, and 67% at less than 6 years of age. Most HCV infected children (19/24) were asymptomatic at diagnosis. Reported clinical features at diagnosis were: lethargy (2), bruising (1), hepatomegaly (1) and failure to thrive (in a child with lethargy). Mildly elevated alanine transaminase levels at diagnosis were recorded in 17/20 (85%): median aspartate aminotransferase (AST) value was 105 IU/ml (range 38-232). Only 1/24 had had a liver biopsy by the time of notification.

The majority of HCV infected children in Australia are born to HSV infected mothers, and are asymptomatic at diagnosis with mildly abnormal liver function tests.

Study investigators

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HIV INFECTION, AIDS AND PERINATAL EXPOSURE TO HIV

Study Highlights

- No new cases of HIV infection were identified in Australian children in 2004.
- All cases reported in 2004 were of perinatal exposure to HIV. Consistent with our previous data 55% of these mothers were exposed to HIV through heterosexual contact in a high HIV prevalence country or in Australia with a partner from a high prevalence country and 32% used IV drugs or had a partner who used IV drugs[1].
- Supporting previously reported trends[1], the proportion of children with perinatal HIV exposure who become infected declined from 41.2% (children born 1995-1996) to 2.4% (children born 2003-2004) due to increasing use of interventions (antiretroviral agents, lower segment caesarian section (LSCS) and avoidance of breastfeeding) in women diagnosed antenatally.

Background

National surveillance for perinatal exposure to HIV, paediatric HIV infection and AIDS is carried out through the APSU in collaboration with the National Centre in HIV Epidemiology and Clinical Research (NCHECR). The study aims to provide more complete ascertainment of perinatal exposure to HIV among Australian children than is available through existing surveillance mechanisms for notifying diagnosed cases of HIV infection and AIDS.

Perinatal exposure to HIV is now the most frequently reported source of HIV infection in Australian children, following the virtual elimination of the risk of exposure to HIV through the receipt of contaminated blood or blood products[1]. Approximately 75% of children born to women with HIV infection who are untreated do not acquire HIV infection perinatally. Some women are unaware of their HIV infection during pregnancy. Thus there is potentially an underestimate of the rate of perinatal exposure to HIV.

The risk of mother-to-child HIV transmission may be significantly reduced among women whose HIV infection is diagnosed before delivery, by interventions such as use of antiretroviral therapy in pregnancy, elective caesarean delivery and avoidance of breastfeeding.

Objectives

- To identify new cases of perinatal exposure to HIV, paediatric HIV infection and AIDS;
- To describe the pattern of perinatal exposure to HIV in Australia;
- To monitor the perinatal HIV transmission rate, and use of interventions for reducing the risk of mother-to-child transmission;
- To describe the natural history of paediatric HIV infection.

Case Definition

Any child born to a woman with HIV infection, whether or not the child has HIV infection, and any child under 16 years of age with diagnosed HIV infection or AIDS

Study results and conclusions

Since 1993 there have been 401 notifications of perinatal exposure to HIV. After exclusion of duplicate cases and reporting errors, 253 children have been identified with confirmed HIV exposure, of whom 39 have developed HIV infection (Figure 14). Nine of those children with HIV infection have since died.

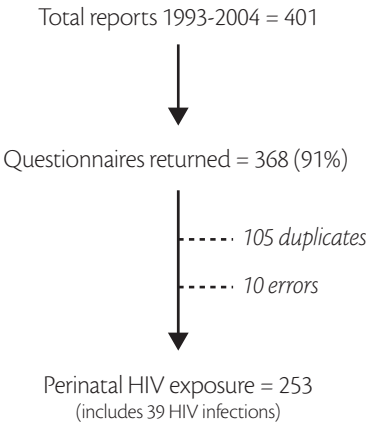

Figure 14. HIV surveillance data summary 1993-2004

In 2004, 27 children were reported with perinatal HIV exposure (1 child born in 2002 is excluded from the commentary in this report). Twelve of the cases reported in 2004 were in NSW, 10 in QLD, 2 in Victoria and 2 in WA. Fifty five percent of the 26 mothers of HIV exposed children, had heterosexual contact in a high HIV prevalence country or had a partner from a high prevelance country. Eight (29.6%) mothers were exposed due to injecting drug use or heterosexual contact with a partner with a history of injecting drug use and 4(14.8%) were due to heterosexual contact with a partner with an unspecified risk of HIV infection.

Of the 26 exposed children, 25 were born to women whose HIV infection was diagnosed prior to delivery. Fourteen of these women reported use of antiretroviral treatment in pregnancy, elective caesarian delivery and avoidance of breastfeeding.

References referred to in text:

1. McDonald AM, Li Y, Cruickshank MA, Elliott EJ, Kaldor JM, Ziegler JB. Use of interventions for reducing mother-to-child transmission of HIV in Australia. *Medical Journal of Australia*, 2001; 174:449-52.

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NEONATAL HERPES SIMPLEX VIRUS INFECTION (HSV)

- Over a half of all neonatal HSV infections in Australia are caused by HSV type 1, in contrast to the USA where HSV type 2 predominates.
- Typical herpetic lesions of the skin, eye or mouth are not evident in approximately half the infants identified with neonatal HSV infection, which makes early diagnosis difficult.
- Disseminated HSV infection in the newborn may be associated with the early onset of pneumonitis in infants (in whom the chest X-ray may be normal). This is highly lethal unless antiviral therapy is initiated.

Background

Neonatal HSV infection is thought to most commonly present as disease localised to the skin, eye or mouth, but can also present as encephalitis, or multi-organ failure. Mortality without antiviral therapy is high. The commonest source of infection is from the genital tract of a mother who experiences her first episode of genital herpes during pregnancy. However infection can be transmitted across the placenta, or post partum from direct contact with lesions of a caregiver. Because an estimated 30% of cases are not associated with skin lesions, and signs are often subtle, there are often significant delays between the onset of infection and the initiation of antiviral agents.

Objectives

- To estimate the incidence of neonatal HSV infection in Australia;
- To determine the proportion of babies with disseminated HSV infection, localised disease or encephalitis;
- To identify the type of HSV causing infection;
- To determine the mode of acquisition of HSV infection.

Case Definition

Any baby ≤ 28 days of age with clinical evidence suggestive of HSV infection:

- HSV isolated from any site **OR**
- HSV detected in CSF by PCR (in the presence of CSF pleocytosis or other evidence of HSV encephalitis) **OR**
- Specific HSV-IgM detected in baby's serum **OR**
- Mother sero-converted or IgM positive and baby has typical clinical manifestations **OR**
- HSV isolated from mother around the time of delivery, and the baby has typical clinical manifestations.

Study results and conclusions

The classification of cases notified to the APSU since 1997 is shown in Figure 15.

Of the 10 confirmed cases reported in 2004, 5 were in NSW, 3 in Victoria, and 2 in WA. Five of these cases were caused by HSV-1, 3 by HSV-2 and 2 were untyped. One third of cases in 2004 presented as skin, eye or mouth disease alone. One third presented as disseminated infection (with or without encephalitis, skin or mucosal involvement). One third of infections were confined to the central nervous system (not disseminated) and with or without skin or mucosal involvement. Overall, half of the HSV-infected infants did not manifest typical herpetic lesions in the skin or mucosal surfaces at presentation. There were two deaths in 2004. One of these children was diagnosed post mortem. Two infants had early, laboratory confirmed HSV recurrences at the time of notification.

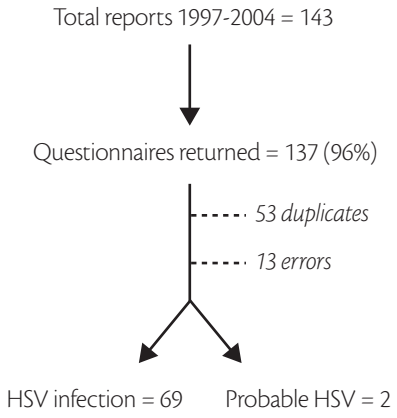

Figure 15. HSV surveillance data summary 1997-2004

Consistent with our findings from 1997-2003, two thirds of infants were born at term (≥ 37 weeks), and the majority (7/10) were born by vaginal delivery

Nine of the ten reported infants in 2004 received antiviral therapy with intravenous acyclovir. Most (8/9) infants were prescribed the recommended dose of acyclovir for neonatal HSV disease (60 mg/kg/day I.V divided into 3 doses), for the recommended duration for the type of disease presentation.

Neonatal HSV disease remains a highly lethal condition despite the availability of affective antiviral therapy. Non specific signs and the absence of classical herpetic lesions at presentation make early diagnosis difficult.

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NON TUBERCULOUS MYCOBACTERIAL INFECTION (NTM)

Study Highlights

- In accordance with the literature, reported cases of non tuberculous mycobacterial infection usually presented with lymphadenopathy in otherwise healthy children aged <5 years. Surgery was performed in the majority (70%) of cases.
- *Mycobacterium avium intracellulare* and *Mycobacterium fortuitum* were the commonest organisms isolated
- Relapse was uncommon (10%) despite the wide range of medical therapies used.

Background

Non tuberculous mycobacteria (NTM) are free living soil and water organisms, causing a spectrum of diseases including lymphadenitis, pulmonary disease, skin and soft tissue infections, ear infections, skeletal infections and disseminated infection. The annual incidence of NTM infections in the developed world is believed to be increasing, however the magnitude of this problem in Australian children is unquantified. The natural history of NTM infection has not been well described and optimal management remains unclear. Information from the study will contribute knowledge to improve the detection and guide management of affected children.

Objectives

- To estimate the incidence of newly diagnosed NTM infection in children seen by child health specialists in Australia;
- To describe the epidemiology and spectrum of disease and document known risk factors;
- To describe diagnostic investigations used in Australia; frequency of use of skin testing and the clinical utility of the test, including differential skin testing;
- To describe the management of NTM in Australia and the response to treatment.

Case definition

Any child under 15 years of age seen in the previous month newly diagnosed with:

1. DEFINITE NTM:
   - 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 NTM:
   - A child who presents with any clinical features compatible with NTM AND
   - has undergone one or more of the supportive investigations AND
   - in whom Mycobacterium tuberculosis (TB) infection is unlikely.

Study results and conclusions

In six months of active surveillance, there were 46 notifications of NTM infection, 8 definite and 12 probable cases. The classification of the 20 cases is presented in Figure 16. Of Of the 20 cases, 8 were in NSW, 7 in Victoria, 3 in QLD, 1 each in WA and SA and none in NT, ACT and TAS.

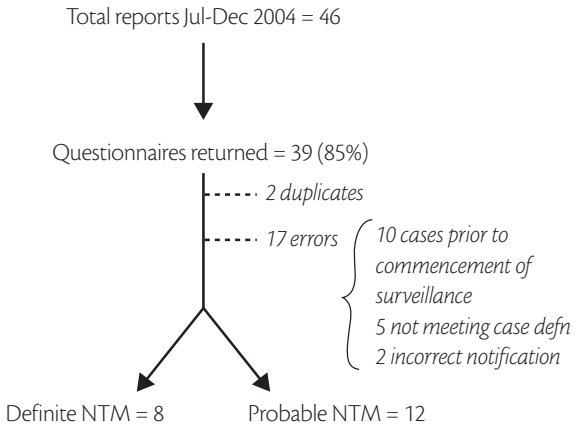

Figure 16: NTM surveillance data summary 2004

Preliminary results indicated that the most common presentation in Australia is isolated lymphadenopathy without any associated systemic features. Most patients were under 5 years of age and did not have any predisposing risk factors. A specific organism was isolated by mycobacterial culture in 65% of patients, with *Mycobacterium avium intracellulare and Mycobacterium fortuitum* the most frequently isolated organisms. Surgical therapy was performed in 70% of cases. The rest were offered medical therapy but there was a significant variation in the treatment prescribed. Despite the diversity of treatment, relapse occurred in only 10% of patients, all of whom had predisposing medical conditions.

Study investigators

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RETT SYNDROME

Study Highlights

- The Australian Rett Syndrome Study has important implications for clinical practice both in Australia and overseas.
- Regular monitoring of trends in incidence and prevalence are important for predicting present and future use of medical & disability services.
- Phenotype-genotype studies show that different mutations result in variable disease severity. This enables clinicians to give better prognostic information to families.
- As an extension of this study biennial collection of information on a cohort of children with Rett syndrome will increase our understanding of the progression and prognosis of this disorder and will be useful for clinicians diagnosing and managing the disorder.

Background

Rett syndrome is a severe neurodevelopmental disorder caused, in most cases, by mutations in the X-linked methyl-CpG-binding protein 2 gene (MECP2). The Australian Rett Syndrome Study is focussing on characterising the genotype and phenotype of children with Rett syndrome. Further understanding of the condition will be provided by the natural history of an Australian cohort of affected children and young people.

Cases identified through the APSU and through additional sources including the Rett Syndrome Association and Disability Services are included in this report.

Objectives

- To identify every person with Rett syndrome in Australia, born during or after 1976 (Incident cases are reported through APSU. Prevalent cases are reported through Rett Syndrome Association of Australia and other sources).
- To describe the epidemiology (including survival analysis of a range of endpoints) of Rett syndrome;
- To investigate the association between genotype and phenotype;
- To evaluate Rett syndrome management strategies;
- To identify factors in the family and community that promote optimal functioning and health for the child/young person with Rett syndrome and her family as a whole.
- As an extension of the APSU study, to obtain information about the progression of the disorder.

Case Definition

Any child born during or after January 1976, with newly diagnosed or possible Rett syndrome, measured against clinical criteria or genetic testing. The clinical criteria include:

- Normal head circumference at birth;
- Deceleration of head growth between five months and four years;
- Loss of acquired purposeful hand skills between ages six and 30 months temporarily associated with communication dysfunction and social withdrawal;
- Development of severely impaired expressive and receptive language and presence of apparent severe psychomotor retardation;

- Stereotypic hand movements such as hand wringing/ squeezing, clapping/tapping, mouthing and "washing/rubbing" automatisms appearing after purposeful hand skills are lost;
- Appearance of gait apraxia and truncal apraxia/ataxia between one and four years.

Study results and conclusions

Update of study 1993-2004

Since the first APSU study commenced in 1993, 278 children (276 females and 2 males) born during or after 1976 have been categorised as having classical or atypical Rett syndrome. For the purpose of this analysis the 2 males have been excluded. Ninety five percent (264/276) of the children were born in Australia. The mean age at diagnosis was 5.3yrs (SD 3.9). Cases were proportionately distributed by State and Territory as for the Australian population.[1]

Eighty eight percent of cases have undergone molecular genetic testing since its introduction in 1999. Of these, 74%(179) tested positive with MECP2 mutations. Mutations p.T158M and p.R168X were the most common in the cohort at 11.5% each, followed by p.R294X (9.8%), p.R270X (8.7%), p.R255X (7.1%), p.R306C and p.R133C (5.5%) each.

Survival in the full cohort was 98% at 10 yrs of age and 77.8% at 25 yrs of age. Twenty five (9.1%) cases have died from a variety of causes. The most common cause of death was pneumonia (n=10) followed by respiratory failure (n=4) and aspiration/asphyxiation (n=3).

There was no difference in survival between children with classical and atypical presentation.

In 2004 there were 31 notifications (24 APSU, 7 other) of Rett syndrome. Clinical information was obtained for 28, indicating a response rate of 90%. Of these, 15 were confirmed Rett syndrome cases with 1 probable case and the remainder were reporting errors or duplicate cases. Six children diagnosed with Rett were in NSW, 4 in VIC, 2 in QLD, 1 each in WA, SA and TAS and none in ACT and NT.

Children with Rett syndrome have a significant impact on health services. Sociodemographic, phenotypic and genetic characteristics are all determinants of health service use. For example, younger cases, those with more severe phenotype and those with random X inactivation are the highest users of health services[2].

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VITAMIN K DEFICIENCY BLEEDING (VKDB) INCLUDING HAEMORRHAGIC DISEASE OF THE NEWBORN

Study Highlights

- Vitamin K deficiency bleeding (VKDB) has a clinical spectrum from mild to severe.
- Particularly in classical VKDB, International normalised ratio (INR) values of 2 which correct with vitamin K administration may be encountered. This has led us to redefine the case definition for 'probable' cases to include children who present with bleeding and INR levels between 2 and 4.
- Newborns most at risk of VKDB are those who have not received vitamin K in any form, hence it is important that parents are carefully counselled about the risks and benefits of vitamin K prophylaxis
- Ongoing surveillance of VKDB is required following the introduction of Konakion MM

Background

In December 1999 the Australian Drug Evaluation Committee registered Konakion MM Paediatric, a new formulation of vitamin K (phytomenadione) containing 2mg in 0.2ml for intramuscular (IM) and oral use. This is currently the only preparation available in Australia.

The current NHMRC recommendations[1] for prophylaxis with Konakion MM are as follows:

- For all healthy neonates:

  1 mg by intramuscular injection at birth (preferred because of reliability of administration). Alternatively, 2 mg orally at birth, at the time of newborn screening (three to five days of age) and at four weeks of age.
- For neonates with special risk factors*:

  1 mg by intramuscular injection at birth. If the neonate has special risk factors and weighs less than 1.5 kg, then 0.5 mg is recommended.

*(infants who are pre-term, unwell or unable to tolerate or absorb vitamin K)

It is imperative that surveillance for VKDB continues following the introduction of Konakion MM. This report summarises the data collected through the APSU during the period 1993 to 2004.

Objectives

- To describe the epidemiology of VKDB in Australia;
- To estimate the morbidity and mortality associated with VKDB;
- To evaluate the efficacy of various regimes of vitamin K prophylaxis.

Case definition

Any infant less than six 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) and in whom the bleeding disorder corrects with vitamin K.

Case Classification

A broad reporting definition was used to ensure complete case ascertainment. Notified cases were reviewed by study investigators and classified according to the following criteria:

Confirmed cases – Infants with coagulation studies as follows: INR greater than 4, prothrombin time greater than 4 times control value, platelet count normal or elevated; and in whom coagulation abnormalities corrected within 24 hours of vitamin K administration.

Probable cases – Infants with a clinical history and findings that strongly suggest a diagnosis of VKDB, and in whom coagulation studies were either cited as abnormal but results were not available to the investigators; were not performed; or showed an INR level between 2-4.

Unknown cases – Infants with a clinical history and findings that made a diagnosis of VKDB possible but unlikely, and in whom there were no laboratory data to confirm or refute the diagnosis.

Errors – cases for which laboratory investigations and response to vitamin K therapy excluded the diagnosis of VKDB.

Study results and conclusions

In the period January 1993 to December 2004, the APSU received 113 notifications of VKDB (Figure 17). Clinical characteristics of the confirmed cases for 1993 to 2004 are shown in Table 7.

In 2004 there were 4 notifications of VKDB: two errors, one duplicate and one probable case (notified in Victoria).

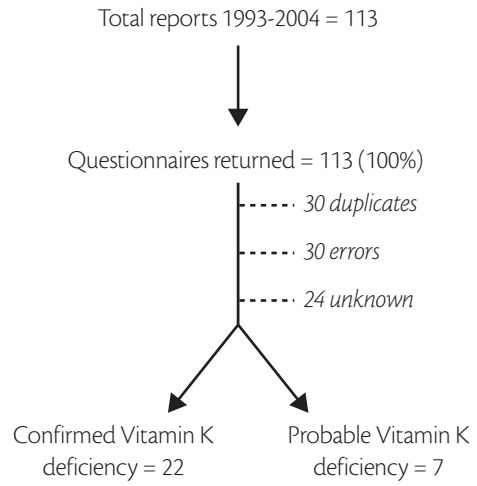

Figure 17: Vitamin K deficiency surveillance data summary 1993-2004

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1. NHMRC Joint Statement and recommendations on Vitamin K administration to newborn infants to prevent Vitamin K deficiency bleeding in infancy. NHMRC 2000. ISBN 0642450676.

Study investigators

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Table 7. Characteristics of definite & probable cases of Vitamin K Deficiency bleeding, 1993-2004

Australia (n=29)		
	n	%
Case Classification		
Confirmed	22	76
Probable	7	24
Disease profile of confirmed cases		
Classical	2	9
Early onset	1	4.5
Late onset	19	86.5
Vitamin K given at birth		
Yes	15	68
Not given	7	32
Type of Vitamin K at birth		
IMI	7	32
Oral	6	27
IVI	2	9
Breast fed	21	95
Term gestation (37-42 weeks)	20	90
Liver disease	11	50
Site of bleeding*		
Skin	12	55
Gastrointestinal tract	6	27
Intracranial	5	23
Umbilical	3	18
Nose	1	4
Circumcision	1	4
Median age of onset in days	47	

*Bleeding may be from multiple sites

STUDY PROTOCOL
HYPERINSULINAEMIC HYPOGLYCAEMIA

Background

Hyperinsulinaemic hypoglycaemia (HI) is a biochemical profile reflecting hyperinsulinaemic, hypoketotic, hypofattyacidaemic hypoglycaemia with increased glucose requirements[1,2]. HI is the most common cause of persistent hypoglycaemia in the neonatal period after the first few hours of life. In severe cases hypoglycaemia is devastating. It may be difficult to control even in a hospital setting and may be associated with early brain damage[1,2]. Published overseas data suggests an incidence of about 1/40,000 births[4].

HI includes a continuous spectrum of conditions with differing genetic aetiology. Clinical features range in severity from subtle signs such as 'floppiness', 'jitters,' 'twitchiness' and poor feeding, through to overt signs such as seizures. While mild disease may be controlled by frequent feeding, severe disease may require medication (diazoxide) and severe unremitting hypoglycaemia may only be relieved by surgical removal of the pancreas (up to near-total resection)[5]. Pancreatic histology from surgical cases is usually abnormal. Disease may be focal on a background of normal pancreatic tissue or diffusely abnormal tissue may be located throughout the pancreas. It has been suggested that different histological types will require varying degrees of surgical resection, although currently this is unclear[5].

HI is a well known cause of neurological damage,[2,6,7,8] thus, is it essential that HI is rapidly diagnosed and managed. Patients who respond well to medical treatment do not need pancreatic resection, however patients who are either non-responsive or unreliably responsive to medical intervention are at risk of brain damage. In the latter group, the decision to undertake pancreatic resection is very difficult because the risk of diabetes must be balanced against the risk of brain damage. Early definitive molecular diagnosis is an important goal because this may help us predict which patients do not respond to medication and require surgery. This is important because early surgery is associated with a reduced risk of diabetes[8].

The Incidence of HI in Australia is unknown. European and Middle Eastern data suggest that the incidence of HI varies from 1/2,500 in consanguineous populations to 1/50,000 births[3,4]. Our collaboration has identified over 70 children from Australia and New Zealand since 1977 giving an estimated rate of around 1/70,000 births. However these children were identified retrospectively and this is likely to be an under-estimate of the true incidence.

Objectives

In this study we seek to document the epidemiology of HI in Australian children and to record known risk factors. With the assistance of reporting clinicians, we will also attempt to recruit notified cases to a longitudinal follow-up study. This will allow collection of prospective data about response to treatment and will help us identify factors associated with good or poor outcomes. This information will contribute to efforts to improve the detection and outcome of HI in affected children.

Case definition

All children under 10 years of age seen in the previous month with newly diagnosed hyperinsulinaemic hypoglycaemia.
That is:

- 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* requiring glucose infusion for more than 10 days

Follow-up of reported cases

A brief questionnaire requesting further details will be forwarded to clinicians who report a case of HI to the APSU. Reporting clinicians will also be invited to send a study information sheet to families of affected children to inform them how to contact study investigators should they wish to participate in the longitudinal study of HI in Australian children.

Study investigators

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This study has the support of the Australasian Paediatric Endocrine Group

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STUDY PROTOCOL
NEONATAL GROUP B STREPTOCOCCUS- SEPSIS

Background

Neonatal and infant *Strepococcus agalactiae* or group B streptococcus (GBS) infection emerged in the 1970s as the commonest cause of neonatal and obstetric sepsis, mainly due to serotype III. In the 1990s it was recognised as an an increasingly common cause of septicaemia in adults, mainly due to serotype V. GBS is carried in the vagina of 25-50% of healthy pregnant women and often transmitted to their infants before or during birth. Usually, colonisation is benign, but ~1% of the infants of carriers (~2/1000 overall) develops life-threatening sepsis.

In a high proportion of cases, early onset infection is initiated (*in utero*) as a result of ascending infection, even in the presence of intact membranes. In some cases there are no obvious clinical risk factors, but the risk is increased by any condition that reduces the mother's ability to contain the organism, especially if conditions favourable to invasive infection occur. A past history of a GBS-infected infant and a previous GBS UTI are markers of increased risk and reflect a poor immune response to carriage. The risk is increased in women with conditions associated with immunosuppression (e.g HIV infection, diabetes or immunosuppressive therapy) or conditions that increase the risk of ascending infection (e.g. prolonged rupture of membranes or instrumental delivery). Preterm labour, with or without premature rupture of membranes, may be either a risk factor for, or a clinical sign of, intrauterine GBS infection. The risk of neonatal infection can be reduced by intrapartum antibiotic prophylaxis in women whose infants are at risk but there is controversy about how best to identify those at risk. Routine antenatal screening for GBS carriage is recommended but has poor specificity. Development of safer, more efficient ways to prevent GBS will require:

- better understanding of bacterial virulence and host susceptibility;
- surveillance to monitor genotype distribution and antibiotic resistance;
- methods to identify the small subset of GBS carriers whose infants are at risk.

Objectives

This study aims to determine:

1. the current incidence of early and late onset neonatal GBS infection
2. the incidence of currently accepted maternal and infant risk factors in children with GBS
3. the proportion, if any, of early onset GBS infections occurring in infants of women who have been given intrapartum antibiotic prophylaxis
4. the short-term mortality and morbidity of early and late onset GBS infection
5. the distribution of GBS genotypes among invasive isolates from different types of neonatal sepsis
6. differences in distribution of genotypes between isolates from infected neonates, pregnant women who are vaginal carriers and adults with bacteraemia.

Case Definition

Any infant with group B streptococcal disease confirmed by isolation of GBS from a normally sterile site e.g. blood, cerebrospinal fluid, joint fluid. Report all incident cases, irrespective of symptoms, in infants aged 0-7 days (early onset) or 8 days to 12 months (late onset) of age.

GBS may present clinically as:

- Early onset neonatal sepsis (birth to 7 days) with symptoms and signs varying in severity from overwhelming multi-organ system disease with shock, respiratory failure, meningitis, Disseminated intravasculor coagulation 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 fullterm infants)
- Late onset sepsis (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.

Study investigators

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International Network of Paediatric Surveillance Units (INoPSU)

The International Network of Paediatric Surveillance Units (INoPSU) was established in 1998 to enhance communication and collaboration among units.

Mission

The mission of INoPSU is "the advancement of knowledge of uncommon childhood infections and disorders and the participation of paediatricians in surveillance on a national and international basis so as to achieve a series of benefits".

Aims

- to facilitate communication and cooperation between existing national paediatric surveillance units;
- to assist in the development of new units;
- to facilitate sharing of information and collaboration between researchers from different nations and scientific disciplines;
- to share information on current, past and anticipated studies and their protocols;
- to encourage the use of identical protocols to potentially enable simultaneous or sequential collection of data on rare paediatric disorders in two or more countries;
- to share and distribute information of educational benefit to constituent units, notably on study and surveillance methodologies;
- to share surveillance techniques and models of evaluation for units;
- to peer review and evaluate existing and proposed units;
- to identify rare disorders of mutual interest and public health importance for simultaneous surveillance through each national unit;
- to collaborate with and provide information to other groups interested in rare childhood diseases such as parent support groups;
- to respond promptly to international emergencies relating to rare childhood conditions where national and international studies can make a contribution to science or public health.

There are currently 15 members of INoPSU, including 13 full members and 2 associate members (Cyprus/Greece and Trinidad and Tobago). A total of 84 uncommon childhood disorders had been studied by the end of 2004. Conditions under surveillance in 2004 are listed in Table 8.

3rd INoPSU Conference, Lisbon Portugal 2004

In April 2004, the 3rd INoPSU Business and Scientific Meeting was held in Lisbon, Portugal. HIghlights of this meeting included the appointment of a new Co-convenors Professor Rudi von Kries (Germany) and Dr Rob Pereira (Netherlands), in place of outgoing Convenor Associate Professor Elizabeth Elliott (Australia). Richard Lynn (UK) will take on the role of communications liaison officer for INoPSU.

The development of a new unit (Trinidad and Tobago), was reported. This unit is expected to commence surveillance in 2005. The first study under consideration is the vertical transmission of HIV/AIDS.

The international collaboration between units was highlighted by development and subsequent publication of proposed guidelines on authorship and acknowledgement for investigators conducting epidemiological research through paediatric surveillance units: (Periera-da-silva L, von Kries R, Rose D, Elliott E. Acknowledging contribution to surveillance studies. *Archives of Disease in Childhood* 2005; 90:768). Papers entitled the Public Health Impacts of Studies Conducted Through National Paediatric Surveillance Units, and Haemolytic Uraemic Syndrome: An International Perspective, are in preparation.

International Paediatric Association Meeting, Cancun Aug 2004.

Associate Professor Elizabeth Elliott (Australia), Dr Danielle Grenier and Andrea Madaglia (Canada) presented on behalf of INoPSU at the International Paediatric Association Conference in Cancun Mexico, August 15-20, 2004. This session focused on collaborative INoPSU research opportunities and stimulated interest in forming surveillance units from Argentinian, Mexican and Venezuelan delegates.

Table 8. Studies under surveillance by international paediatric surveillance units in 2004

Study	International Paediatric Surveillance Unit
Abdominal injury due to child abuse	BPSU
Acute encephalitis	PPSU
Acute flaccid paralysis	APSU, CPSP, NZPSU, SPSU
Acute rheumatic fever	SPSU
Adverse effects from complementary or alternative medicine	APSU, WPSU
Alcohol and children	IPSU
Anaphylaxis following food ingestion	APSU
Ataxia	NSCK
Atypical mycobacterial infections, Atypical tuberculous infection or Nontuberculous mycobacterial infection	ESPED, NSCK, APSU
Autism in children under 5 years	IPSU
CHARGE association/syndrome	CPSP
Conversion disorder	APSU
Complicated pneumonia including empyema	WPSU
Congenital cytomegalovirus infection	APSU, BPSU
Congenital rubella syndrome	APSU, BPSU, CPSP, NZPSU, SPSU
Congenital toxoplasmosis	BPSU
Diabetes mellitus	ESPED, IPSU, LPSU, PPSU
Down's syndrome	NSCK
Drugs (medication) related adverse events	BPSU
Early-onset eating disorder	APSU, CPSP
Fetal alcohol syndrome	APSU
Foregut & hindgut malformations	NZPSU
Fragile X	IPSU
Haemolytic uremic syndrome	NZPSU, LPSU, PPSU, SPSU
Hemoglobinopathy	NSCK, APSU
Hepatitis C virus infection	APSU, CPSP
Hereditary periodic fever syndrome	ESPED
HIV/AIDS or Perinatal Exposure to HIV	APSU, BPSU, LPSU, NSCK, NZPSU
Hodgkin's lymphoma	LPSU
Hypernatraemia	NSCK
Hypophosphatasia	ESPED
Idiopathic nephrotic syndrome	NSCK, NZPSU
Idiopathic thrombocytopenic purpura	NSCK
Imported tropical diseases: malaria, schistosomiasis, leishmaniasis	ESPED
Inborn errors of metabolism	NZPSU
Ingestion of lamp oil (intoxications)	ESPED
Inherited hypocalcemic salt-losing tubulopathies/ Bartter-like syndromes	ESPED
Insufficient breast-feeding	NSCK
Intussusception	SPSU
Invasive fungal infections in VLBW infants	BPSU
Invasive *Haemophilus influenzae* infections (all types)	ESPED
Invasive group B strepcoccus infection	ESPED, PPSU
Juvenile idiopathic arthritis	WPSU
Kawasaki disease	CGPSU, PPSU

Table 8. continued. Studies under surveillance by international paediatric surveillance units in 2004

Study	International Paediatric Surveillance Unit
Kernicterus	ESPED
Langerhans cell histiocytosis	BPSU
Lap-belt syndrome	CPSP
Leukaemia	LPSU
Malaria	NSCK
Medium-chain acyl-CoA dehydrogenase deficiency	NSCK
Meningoencephalitis	PPSU
Munchausen by proxy syndrome	APSU
Necrotizing fasciitis	CPSP
Neonatal herpes simplex virus infection	APSU, CPSP, SPSU
Neonatal hyperbilirubinemia – severe	BPSU, CPSP
Neonatal liver failure/perinatal hemochromatosis	CPSP
Neonatal sinus venous thrombosis	ESPED
Nesidioblastosis	LPSU
Neural tube defects	SPSU
Non-Hodgkin's lymphoma	LPSU
Opsoclonus myoclonus syndrome	IPSU
Pancytopenia	CGPSU
Pertussis	CGPSU
Pneumococcal sepsis/meningitis	ESPED, NZPSU
Prader-Willi syndrome	CPSP
Progressive intellectual and neurological deterioration	BPSU
Prolonged infantile cholestasis	NZPSU
Respiratory syncytial virus (RSV) disease	SPSU
Rett syndrome	APSU
Septo-optic dysplasia	WPSU
Shaken baby syndrome	SPSU
Small bowel insufficiency	NSCK
Splenectomy and hyposplenism	WPSU
Subacute sclerosing panecephalitis and complications	ESPED
Subdural haemorrhage (<2 years)	WPSU
Thrombocytopenia	IPSU
Thrombosis	BPSU
Tick-borne encephalitis	SPSU
Tuberculosis	BPSU, WPSU
Varicella/zoster infection	BPSU, ESPED, SPSU
Vitamin D deficiency rickets	CGSPU, CPSP
Vitamin K deficiency bleeding/HDNB	APSU, BPSU, NZPSU
West syndrome	CGPSU

Legend:

APSU	Australian Paediatric Surveillance Unit	MPSU	Malaysian Paediatric Surveillance Unit
BPSU	British Paediatric Surveillance Unit	NSCK	Netherlands Paediatric Surveillance Unit
CGPSU	Cyprus/Greece Paediatric Surveillance Unit	NZPSU	New Zealand Paediatric Surveillance Unit
CPSP	Canadian Paediatric Surveillance Program	PNGPSU	Papua New Guinea Paediatric Surveillance Unit
ESPED	German Paediatric Surveillance Unit	PPSU	Portuguese Paediatric Surveillance Unit
IPSU	Irish Paediatric Surveillance Unit	SPSU	Swiss Paediatric Surveillance Unit
LPSU	Latvian Paediatric Surveillance Unit	WPSU	Welsh Paediatric Surveillance Unit

INoPSU

British Paediatric Surveillance Unit (BPSU)
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Peer and Editorial Review

Original Articles

1. Brussen KA, Stambos V, Thorley BR. Annual report of the Australian National Poliovirus Laboratory 2003. *Communicable Diseases Intelligence* 2004; 28(3):339-44.
2. Colvin L, Leonard H, de Klerk N, Davis M, Weaving L, Williamson S, et al. Refining the phenotype of common mutations in Rett syndrome. *Journal of Medical Genetics* 2004; 41(1):25-30.
3. Elliott EJ, Bower C. Fetal alcohol syndrome: fact or fiction? *Journal of Paediatrics and Child Health* 2004; 40:8-10.
4. Elliott EJ, McIntyre P, Ridley G, Morris A, Massie J, McEniery J, et al. National study of infants hospitalised with Pertussis in the acellular vaccine era. *Paediatric Infectious Diseases Journal* 2004; 23(3):246-252.
5. Elliott E J Rose D. Reporting of communicable disease conditions under surveillance by the APSU, 1 January to 30 September 2003. *Communicable Diseases Intelligence* 2004; 28(1): 90-91.
6. Elliott EJ, Rose D. Reporting of communicable disease conditions under surveillance by the APSU, 1 January to 30 June 2004. *Communicable Diseases Intelligence* 2004; 28(4):529-531.
7. Elliott EJ, Rose D, Cronin P, Zurynski Y, (Eds). Australian Paediatric Surveillance Unit Surveillance Report 2002-2003. Australian Paediatric Surveillance Unit. 10, 2004 Sydney, ISSN: 1443-3524.
8. Freidman E, Mindel, A Jones CA. Epidemiological, clinical and laboratory aids for the diagnosis of Neonatal herpes – an Australian perspective. *Herpes* 2004; 11:38-40.
9. Leonard H, Slack-Smith L, Philips T, Richardson S, D'Orsogna L, Mulroy S. How can the internet help parents of children with rare neurologic disorders? *Journal of Child Neurology* 2004; 19(11):902-7.
10. Leonard H, Weaving L, Eastaugh P, Smith L, Delatycki M, Witt Engerstrom I, et al. Trisomy 21 and Rett syndrome: a double burden. *Journal of Paediatrics and Child Health* 2004; 40(7):406-9.
11. Weaving LS, Chistodoulou J, Williamson SL, Friend KL, McKenzie OL, Archer H, et al. Mutations of CDKL5 cause a severe neurodevelopmental disorder with infantile spasms and mental retardation. *American Journal of Human Genetics* 2004; 75(6):1079-93.

Research Reports containing APSU data

12. McDonald AM (Ed). National Centre in HIV Epidemiology and Clinical Research. *HIV/AIDS, Viral Hepatitis and Sexually Transmissible Infections in Australia Annual Surveillance Report 2004.* National Centre of HIV Epidemiology and Clinical Research. University of NSW, Sydney 2004. ISSN 1442-8784.
13. Australian Report for the Tenth Meeting of the Regional Certification Commission for the Certification of the Eradication of Poliomyelitis in the Western Pacific, Manila, Philippines 20-21 October 2004

Published Abstracts

14. Ager S, Leonard H, Schmitt L, Fyfe S. Associations between Rett syndrome genotype and scoliosis. Abstracts of the 9th International Congress of Human Genetics Society of Australasia, Perth, 2004;
15. Elliott EJ, on behalf of Australian Paediatric Surveillance Unit (APSU) FAS study group. Fetal alcohol syndrome (FAS) in Australia. Abstracts of the International Network of Paediatric Surveillance Units Scientific Mtg, *Portugese Surveillance Unit Bulletin* 2004; 5[1]:17 [ISSN:1645-0558].
16. Elliott EJ, on behalf of the Australian Paediatric Surveillance Unit (APSU) HUS study group: Henning P, Powell HR, Knight J, O'Loughlin E, et al. Atypical haemolytic uraemic syndrome(HUS) in Australlia. Abstracts of the 3rd International Network of Paediatric Surveillance Units Scientific Mtg, Lisbon, Portugal. *Portugese Surveillance Unit Bulletin* 2004; 5[1]:15 [ISSN:1645-0558].
17. Elliott EJ, Lynn R, on behalf of INoPSU Secretariat. Abstracts of the 3rd International Network of Paediatric Surveillance Units Scientific Mtg, Lisbon, Portugal. *Portugese Surveillance Unit Bulletin* 2004; 5[1]:17 [ISSN:1645-0558].
18. Elliott EJ, Lynn R, Schmid H, Proulx F, Wong W, Sockett P et al, on behalf of the members of the Australian, British, Canadian, New Zealand, Swiss and Portugese Paediatric Surveillance Units' HUS study groups. Surveillance of HUS: an International collaboration. Abstracts of the 3rd International Network of Paediatric Surveillance Units Scientific Mtg, Lisbon, Portugal. *Portugese Surveillance Unit Bulletin* 2004; 5[1]:14 [ISSN:1645-0558].
19. Elliott EJ, Rose D. Public health impact of studies conducted through the APSU, Abstracts of the International Network of Paediatric Surveillance Units Scientific Mtg, *Portugese Surveillance Unit Bulletin* 2004; 5[1]:11 [ISSN:1645-0558]
20. Elliott EJ, Rose D. Comprehensiveness of ascertainment of case reports in active surveillance. Abstracts of the International Network of Paediatric Surveillance Units Scientific Mtg, Lisbon, Portugal. *Portugese Surveillance Unit Bulletin* 2004; 5[1]:10 [ISSN:1645-0558]
21. Elliott EJ, Rose D. Acknowledging the contribution of clinicians to APSU surveillance. Abstracts of the International Network of Paediatric Surveillance Units Scientific Mtg, Lisbon, Portugal. *Portugese Surveillance Unit Bulletin* 2004; 5[1]:7 [ISSN:1645-0558]
22. Hardikar W, Polis S, Kesson A, Mews C, Dore G, Elliott E, Jones CA, Kaldor J. National surveillance of hepatitis C virus infection in Australian children. Abstracts of the American Association for the Study of Liver Diseases (AASLD) Annual Scientific Meeting. Boston, 2004; 40:A576.
23. Jones CA. The epidemiology and immunobiology of congenital and perinatal infections. Abstracts of the Annual Scientific Meeting of the Australasian Society for Infectious Diseases, Alice Springs, NT. 2004; A11.00:P-34.
24. Jones C, Kaldor J, Polis S, Elliott E, Rose D, Hardikar W, Kesson A, Dore G, Mews C. Hepatitis C virus infection in Australian children: Preliminary report of the APSU paediatric HCV study. Abstracts of the Annual Scientific Meeting of the Australasian Society for Infectious Diseases, Alice Springs, NT. 2004;13,30:P-29.
25. Jones CA, Isaacs, D, McIntyre P, Cunningham A, Garland S. Epidemiology of neonatal herpes simplex virus infection in Australia, results from 6 year prospective surveillance (1997-2002). Abstracts of the 29th International Herpes Virus Workshop, 2004; 4:02.
26. Jones CA. Preliminary data from APSU hepatitis C virus in childhood study. Abstracts of the 29th International Herpes Virus Workshop, 2004.
27. Leonard H. Genotype and early development in Rett syndrome: the value of international data. Abstracts of the 9th International Congress of Human Genetics Society of Australasia, Perth, 2004.
28. Munro SC, Trincado DE, Maine G, Rawlinson WD. Study of congenital infections in pregnancy (SCIP) and the outcome of congenital CMV infection. Abstracts of Perinatal Society of Australia and New Zealand 8th Annual Congress Sydney, 2004; A29P.

29. Payne J, Elliott E Haan E, Bower C. Diagnosis of fetal alcohol syndrome (FAS). Abstracts of the Paediatrics and Child Health Division, RACP Annual Scientific Mtg. Rue Wright Memorial Award Finalist. *Journal of Paediatrics and Child Health* 2004; 40(9-10):A6.
30. Polis S, Rose D, Kesson A, Mews C, Hardikar W, Dore G, Elliott E, Jones CA, Kaldor J. National Surveillance for Hepatitis C virus infection in children. Abstracts of the 16th Australiasian Society for HIV Medicines (A.S.H.M). conference, ACT. *Int Medical Journal* 2004.
31. Polis S, Kesson A, Hardikar W, Mews C, Dore G, Elliott E, Jones CA Kaldor J. National surveillance of Hepatitis C virus infection in Australian Children. *Hepatology* 2004; 40:A576.
32. Robins-Browne RM, Elliott EJ, Bennett-Wood V, Russell J, and the HUS Study Group of the Australian Paediatric Surveillance Unit. Enterohaemorrhagic *E. Coli*, diarrhoea and haemolytic uraemic syndrome in Australia. *Pediatric Nephrology* 2004; 19:C52.
33. Rose D on behalf of the Australian Paediatric Surveillance Unit Conversion study group. The complex presentation of conversion disorder in Australian children. Abstracts of the 3rd International Network of Paediatric Surveillance Units Scientific Mtg, Lisbon, Portugal. *Portugese Surveillance Unit Bulletin* 2004; 5[1]:13 [ISSN:1645-0558].
34. Rose D, Morris A. Epidemiological research in mental health and paediatrics surveillance units. Abstracts of the 3rd International Network of Paediatric Surveillance Units Scientific Mtg, Lisbon, Portugal. *Portugese Surveillance Unit Bulletin* 2004; 5[1]:16 [ISSN:1645-0558].

Invited presentations

RACP Annual Scientific Meeting, Canberra, ACT, May 2004.

FAS Symposium

35. Bower C, O'Leary C, Payne J, Elliott E (Invited). Fetal alcohol syndrome: epidemiology.
36. Elliott E, Bower C, Payne J Haan E (Invited). Fetal alcohol syndrome: clinical features.
37. Payne J, Bower C, Elliott EJ, Haan E, D'Antoine H (Invited). Health professionals' practice and opinions about alcohol consumption in pregnancy.

APSU Showcase

38. Elliott EJ (Invited). Surveillance of haemolytic uraemic syndrome: an international collaboration.
39. Emder P (Invited). Haemoglobinopathies
40. Fletcher J (Invited). Nephrotic syndrome in Australian children
41. Jones CA (Invited). Surveillance of congenital and perinatal infections.
42. Leonard H, Bower C (Invited). InterRett: international developments in Rett syndrome research.
43. Palasanthiran P, Best E (Invited). Non-tuberculosis mycobacterial infection.

International Network of Paediatric Surveillance Units Scientific Meeting. Paediatric Surveillance: Portugese Paediatric Society, Lisbon 2004

44 . Elliott E (Invited). Welcome address to delegates from the President of the International Network of Paediatric Surveillance Units.

International Paediatric Association Meeting, Cancun Mexico

45. Elliott E (Invited). Haemolytic Uraemic Syndrome and the International Network of Paediatric Surveillance Units.

Paediatric Update 2004. Childrens Hospital at Westmead, November 2004.

46. Jones CA (Invited). Hepatitis C screening in children.

Rett Syndrome Conference. Ottawa, Canada, October 2004.

47. Leonard H (Invited). Future horizons in Rett syndrome.

Australian Society of Microbiology Special Interest Meeting. Katoomba, NSW, July 2004.

48. Jones CA (Invited). Congenital viral infections – diagnosis and management.

Other presentations

Australasian Professional Society on Alcohol and Other Drugs, Fremantle, November 2004.

49. O'Leary C, Bower C, Payne J, Elliott E, Haan E. Fetal alcohol syndrome: Health professionals' knowledge and practice.

32nd International Medical Advisory Group Conference, Canberra, October 2004.

50. Bower C. Fetal Alcohol Syndrome in Australia.

Public Seminar on Fetal Alcohol Syndrome – Fetal Alcohol Spectrum Disorder International Awareness Day, Adelaide, September 2004.

51. Haan E, Payne J, Bower C, Elliott E, and contributors to the Australian Paediatric Surveillance Unit. Fetal alcohol syndrome and the APSU minimum prevalence study of FAS.

Videoconference with twenty linked sites throughout Western Australia, Perth, July 2004.

52. Bower C, Leonard H, Glasson E, Payne J, Petterson B, O'Leary C, Bourke J. Intellectual Disability and Fetal alcohol syndrome: reducing the impact of neurodevelopmental disorders in our community.

Child Neurology Study Group, Margaret River, May 2004.

53. Leonard H. Rett syndrome research in Australia – past, present and future.

Perth Epidemiology Group, May 2004.

54. Laurvick C, Leonard H, de Klerk N. Optimising follow-up in the Australian Rett syndrome study.
55. Moore H, Leonard H, Carey M, de Klerk N. What makes families respond to questionnaires? A discussion of the risk factors in an international epidemiological study of Rett syndrome.

Telethon Institute for Child Health Research, Away Days, Wollaston College, Perth, March 2004.

56. Payne J, Elliott E, Bower C, Haan E. Fetal alcohol syndrome in Australia: survey of health professionals.
57. Laurvick C, Moore H, Leonard N. The future of Rett syndrome research.

Hospital Sant Joan de Déu, Paediatricians session. Barcelona Spain, October 2004

58. Leonard H. Rett syndrome research in Australia – past, present and future.

Clinicians Reporting Cases 2004

ACT
Dr Ian F Crawshaw
A Prof G David Croaker
Dr Hilary A Holmes
Dr Ian W Hufton
Dr Alison Kent

NSW
Dr C Abkiewicz
Dr Jane H Antony
Dr Philip J Beeby
Dr Srinivas Bolisetty
Dr Anne M Bye
Dr Timothy D Bohane
Dr Jennifer Bowen
Dr MP Brydon
Dr Adam Buckmaster
Dr Dianne Campbell
Dr Michael J Cole
Dr Felicity A Collins
Dr Carolyn Cooper
Dr Paul Craven
Dr Clare Cunningham
Dr P Davidson
Dr Shoma Dutt
Dr CJ Ellaway
A/Prof Elizabeth Elliott
Dr PJ Emder
Dr Adrienne Epps
Dr Elizabeth R Fagan
Dr James McL Friend
Dr Stuart M Gadd
Dr PM Goodhew
Dr Toby Greenacre
Dr G Hardache
Dr Robert Hardwick
Dr Stephen Hartman
Dr Andrew Holland
Dr PY Hong
Dr Keith M Howard
Dr Christopher Ingall
Dr Stephen Jacobe
Dr Cheryl A Jones
Dr Alison M Kesson
Dr Allan M Kerrigan
Dr M R Kluckow
Dr Michael Kohn
Dr Erik La Hei
Dr Ian D Lennon
Dr BH Lo
Dr Keith Lui
Dr Susan Marks
Dr Hugh O Martin
Dr Sloane Madden
Dr David McDonald
Dr Joseph Moloney
Dr Desmond Mulcahy
Dr Patricia E Mutton
Dr DA Osborn
Dr Ju Oei
Dr Robert A Ouvrier
Dr Keith M Power
Dr P Palasanthiran
Dr Greg Rowell
Dr S Raman
Prof W Rawlinson
Dr Susan J Russell
Dr Monique Ryan
Dr Charles M Scarf
Dr Peter Shaw
Dr Jacqueline A Stack
Dr Jacqueline Small
Dr Janine M Smith
Dr David R Starte
Dr Lee Sutton
Dr Bryan M Symons
Dr Dimitra Tzioumi
Dr Boyd Webster
Dr Mary-Clare Waugh
Dr Mark Westphalen
Dr Bruce Whitehead
Dr Gary Williams
Dr George Williams
Dr Ian Wilkinson
Dr Helen Woodhead
Prof Graham Vimpani
A/Prof John B Ziegler

NT
Dr Paul Bauert
Dr Andrew White

QLD
Dr Donald Appleton
Dr Deborah Bailey
Dr David Cartwright
Dr Richard E Cherry
Dr FL Connor
Dr John W Cox
Dr JA Cullen
Dr Tim Donovan
Dr Loui Ee
Dr Aaron Easterbrook
Dr Leonie M Gray
Dr Peter H Gray
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Dr G J Hart
Dr T E Hassell
Dr Richard Heazlewood
Dr Thomas Hurley
Dr Peter Lewindon
Dr Elena Mantz
Dr John McCreanon
Dr Robert McGregor
Dr David McMaster
Dr Anthony Morosini
Dr Susan Moloney
Dr Clare Nourse
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Dr Jose Prado
Dr David Rogers
Dr Patrick Ryan
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Dr Bruce Urquhart
Dr Claire Wainwright
Dr Timothy Warnock
Dr J Waugh
Dr Judy Williams
Dr Michael Williams
Dr Geoffrey Withers

SA
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Dr WD Ford
Dr Paul Goldwater
Dr Bevan Headley
Dr MA Higgins
Dr DM Lawrence
Dr David J Moore
Dr Scott Morris
Dr C Pearson
Dr Michael Rice
Dr Rima Staugas
Dr Heather Tapp
Dr EM Thompson

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Dr CJ Bailey
Dr Anthony Dunston
Dr Michelle Willams

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Dr Kym P Anderson
Dr PB Bergman
Dr Jim Buttery
Dr JR Carapetis
Dr Daniel Casalaz
Dr Bronwyn A Cathels
Dr Caroline Clarke
Dr Jacinta Coleman
Dr John Connelly
Dr D Cutting
Dr Martin Delatycki
Dr John H Drew
Dr P Eastaugh
Dr Geoffrey W Ford
Dr C Hamilton
Dr Winita Hardikar
Dr Richard Haslam
Dr Simon Hauser
Dr Michael Hayman
Dr John A Heath
Dr James Holberton
Dr Rod W Hunt
Prof John Hutson
Dr Susan E Jacobs
Mr C Kimber
Dr Paul Lee
Dr Robert Lim
Dr Catherine Lynch
Dr Edwin Lowther
Dr Lionel Lubitz
Dr Leslie J Markman
Dr RJ Massie
Dr Joanna McCubbin
Dr KL McKay
Dr Kathryn McMahon
Dr Tracey Merriman
A/Prof P Monagle
Prof Colin Morley
Dr C Nanayakkara
Dr Margot Nash
Dr MJ Nowotny
Dr Gillian Opie
Dr Roderic Phillips
Dr Sheryle Rogerson
Dr Kerryn R Saunders
A/Prof S Sawyer
Dr Leslie Sheffield
Dr David Sholl
Dr Lindsay J Smith
Dr Robert A Sloane
Dr Mike Starr
Dr Keith B Stokes
Dr TG Stubberfield
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Dr Brian J Timms

WA
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Dr David P Burgner
Dr Catherine H Cole
Dr GM Chaney
Dr Noel P French
Clin A/Prof R Hagan
Dr CA Jeffries-Stokes
Dr C Kikiros
Dr Cathy Kiraly-Borri
Dr Catherine F Mews
Dr Aida Partridge
A/Prof Susan Prescott
Dr JM Silberstein
Dr Karen N Simmer
Dr Janine Spencer
Dr Jeffery R Tompkins
Dr Andrew M Wawryk
Dr Helen Wright

Congratulations to the highlighted clinicians, who reported the most cases in 2004.

Clinicians returning 100% (all months) of cards in 2004

ACT

Dr Judith Bragg
Dr Ian Crawshaw
Dr Ian Hufton
Dr Paul Jenkins
Dr Penelope Johnson
Dr Z Kecskes
Dr Antony Lafferty
Dr G Malecky
Dr Michael Rosier

NSW

Dr C Abkiewicz
Dr Ion S Alexander
Dr Garth Alperstein
Dr Rosemary Ambler
Dr Alan F Amos
Dr Donald G Anderson
Dr Ian Andrews
Dr John D Arnold
Dr Jennifer E Ault
Dr Nadia Badawi
Prof Louise A Baur
Dr Philip J Beeby
Dr Graham J Bench
A/Prof David L Bennett
Dr G P Bent
Dr Mary E Bergin
Dr Martin Berry
Dr Michael Besser
Dr Alice Blick
Dr Paul Bloomfield
Dr Timothy D Bohane
Dr John Boulton
Dr J J Brereton
Dr Warwick Britton
Dr Kerry Brown
Dr M P Brydon
Dr Adam Buckmaster
Dr P Buckner
Dr Laurence E Budd
Dr Mary Burke
Dr Anne M E Bye
Dr Patrina H Y Caldwell
Dr Dianne Campbell
Dr PJ Campbell
Dr Danny Cass
Dr Paul C Chay
Dr Kity Chee
Dr Paul Chidiac
Dr Howard Chilton
Dr Alan Chong
Prof John Christodoulou
Dr Simon D Clarke
Dr John C Coakley
Dr Ralph C Cohen
Dr A F Colley
Dr Mithran Coomarasamy
Dr Peter John Cooper
Dr Michael C Copeman
Dr Eric S Coudounaris
Dr Heather Coughtrey
A/Prof Christopher T Cowell
Dr Maria Craig
Dr Patricia Crock
Dr Genevieve Cummins
Dr Clare A Cunningham
Dr Julie A Curtin
Dr J Dalby-Payne
Dr Luce Dalla-Pozza
Dr P Davidson
Dr Andrew Day
Dr John A De Courcy
Dr Michael J Deloughery
Dr Kym Donaghue
Dr Peter J Donald
Dr Clare Doocey
Dr Stuart F A Dorney
Dr Ana Maria Dosen
Dr David Dossetor
Dr John Robert Douglas
Dr Peter E Doyle
Dr Richard J Dunstan
Dr Linda Durojaiye
Dr Peter W Ebeling
Dr M J Edwards
Dr C J Ellaway
A/Prof Elizabeth J Elliott
Dr Phillip John Emder
Dr Adrienne G Epps
Dr Anthony D Epstein
Dr M J Fairley
Dr Bruce J Fasher
Dr Michael Fasher
Dr John Feller
Dr Penelope Field
Dr Dominic Fitzgerald
Dr Stuart M Gadd
Prof Kevin J Gaskin
Dr Madlen Gazarian
Dr Maurice D Gett
Dr Henry JGilbert
Dr Anna Clare Gill
Dr Deepak Gill
Dr Jonathan Gillis
Dr Joanne Ging
Dr Neil D Ginsberg
Dr Anne F Glanville
Dr Chin Lum Goh
Dr P M Goodhew
Dr Sandra P Grass
Dr P Grattan-Smith
Dr Anne Hackett
Dr Michael S Haifer
Dr Robert J Halliday
Dr Ralph M Hanson
Dr Nils F Hanson
Dr G Hardacre
Dr Robert J Hardwick
Dr Michael J Harris
Dr Richard K Hart
Dr Stephen H Hartman
Dr John G Harvey
Dr Richard E Hawker
Dr Leigh Haysom
Dr Stephen Hing
Dr Elisabeth M Hodson
Dr James C S Hong
Dr Jason Hong
Dr Neville J Howard
Dr Keith M Howard
Dr M M Jack
Dr Stephen Jacobs
Dr Con A James
Dr Robyn Jamieson
Prof Heather E Jeffery
Dr Patricia M Johnson
Dr Sandra L J Johnson
Dr Heather Johnston
Dr Owen Jones
Dr C J Jones
Dr G M Kainer
Dr Alyson M Kakakios
Dr Stewart J Kellie
Dr Allan Kelly
Dr Allan M G Kerrigan
Dr Alison M Kesson
A/Prof Henry A Kilham
Dr Eli Kleiner
Dr M R Kluckow
Dr Karen Knoll
Dr Philip Kolos
Dr Peter Kristidis
Dr M Kumaradeva
Dr Ahti T Lammi
Dr Basiliki Lampropoulos
Mr Erik La Hei
Dr K C Lau
Dr Joanne Leal
Dr J Lemoh
Dr Ian D Lennon
Dr Joyce Leong
A/Prof Florence Levy
Dr Deborah J Lewis
Dr David Lillystone
Dr Anthony J W Liu
Dr O Lozynsky
Dr Kei Lui
Dr K T MacDonald
Dr Sloane Madden
Dr Annabel K Magoffin
Dr Albert Mansour
Dr Susan M Marks
Prof Frank J Martin
Dr Hugh C O Martin
Dr Tania May
Dr Robert McCarthy
Dr Tim McCrossin
Dr Andrew D McDonald
Dr Jennifer L McDonald
Dr John R McGill
Dr Peter B McIntyre
Dr Margo McIver
Dr Tracey Merriman
Dr Desmond L Mulcahy
Dr Marea W Murray
Dr Patricia E Mutton
Dr Anandhan P Naidoo
Prof Kathryn North
Prof R Kim Oates
Dr Anthony O'Connell
Dr Jui Oei
Dr Stephen J O'Flaherty
Dr David A Osborn
Dr Dimitrios Papadopoulos
Dr Julianne Parle
Dr Deborah G Perkins
Dr Megan Phelps
Dr Elizabeth Pickford
Dr Susan Piper
Dr Jaqueline C Pollack
Dr Melvyn Polon
Dr Alison Poulton
Dr Keith M Power
A/Prof Peter G Procopis
Dr Karin L Proudman
Dr Stephen D Pryde
Dr Patrick M Rahilly
Dr Bruce B Richards
Dr Suzanne I Robertson
Dr Marilyn Rochefort
Dr Laurence G Roddick
Dr A Ronan

Congratulations to the highlighted winners of the 2004 wine prize.

Clinicians returning 100% (all months) of cards in 2004

A/Prof Andrew Rosenberg
Dr L Paul Roy
Dr John W Ruhno
Dr Monique Ryan
Dr Peter J Rye
Dr Charles M Scarf
Dr Adam M Scheinberg
Dr David N Scheil
Dr Mark Selikowitz
Dr Christopher Seton
Dr Arun S Shanker
Dr Peter Shaw
Dr E Shi
Dr Gary F Sholler
Dr Albert Shun
Dr Martin Silink
Dr David O Sillence
Dr Natalie Silove
Dr J K H Sinn
Dr D Singer-Remeljan
Dr Janine Margo Smith
Dr Helen M Somerville
Dr Velencia Soutter
Dr Barry J Springthorpe
Dr Graeme Stein
A/Prof K Steinback
Dr Glenn Stephens
Dr Michael M Stevens
Dr John E Stuart
Dr Paul R Tait
Dr Arthur Teng
Dr Kathryn E Thacker
Dr Ganesha Thambioillay
Dr Gamini D Thenuwara
Dr Sue Thompson
Dr Susan J Towns
Dr Anne M Turner
Dr Dimitra Tzioumi
Dr Peter Van Asperen
Dr Charles Verge
Dr Graham V Vimpani
Dr Anne F Vimpani
Dr Chris Wake
Dr Philip Watt
Dr Mary-Clare Waugh
Dr Boyd Webster
Prof Leslie White
Dr Bruce Whitehead
Dr Bridget Wilcken
Dr Catherine R Wiles
Dr Barry Wilkins
Dr Ian Wilkinson
Dr George L Williams
Dr Meredith Wilson
Dr Carola Wittekind
Dr Barry E Wyeth
Dr Kylie Meredith Yates
Dr Simon Young
A/Prof John B Ziegler
Dr Michael Zilibowitz

NT

Dr Paul A M Bauert
Dr Charles J J Kilburn
Dr Louise Martin
Dr Peter S Morris

QLD

Dr Jason Acworth
Dr Donald B Adsett
Dr L Ah Yui
Dr Donald Appleton
Dr Deborah Bailey
Dr Ruth Barker
Prof Jennifer Batch
Dr P Bjerragaard
Dr Andrew Blair
Dr Richard Broen
Dr J Byrne
Dr Leisha A Callaghan
Dr Gregoey Carman
Dr David Cartwright
Dr Richard Cherry
Dr Ronald Clark
A/Prof G Cleghorn
Dr John Coghlan
Prof Paul Colditz
Dr FL Connor
A/Prof David Cooper
Dr John W Cox
Dr Mark davies
Dr Peter Debuse
Dr Nigel Dore
Dr Loui Ee
Dr Ian Findlay
Dr Paul Francis
Dr Donna Gandini
Dr John Gavranich
Dr Glen Gole
Dr Bruce Goodwin
Dr Peter Gray
Dr Leonie M Gray
Dr Margaret-Anne Harris
Dr G J Harte
Dr Tim E G Hassall
Dr Richard Heazlewood
Dr Thomas M Hurley
Dr E M Hurrion
Dr Ronald W James
Dr Robert W Justo
Dr Lisa Kane
Dr Sumant Kevat
Dr J Kynaston
Mr Mervyn Lander
Dr Peter J Lewindon
Dr Bruce R Lewis
Dr John McCreanor
Dr Julie McEniery
Dr James J McGill
Dr Steven McTaggart
Dr William McWhirter
Dr Ross Messer
Dr Malcolm Miller
Dr David Moore
Dr Anthony Morosini
Dr Brian Morris
A/Prof Michael Nissen
Dr Trevor Olsen
Dr Tat-Hin Ong
Dr Peter O'Regan
Dr Brian Patten
Dr Donald Perry-Keene
Dr Jose Prado
Dr Jeffrey Prebble
Dr Dorothy Radford
Dr David Rogers
Dr Peter Roper
Dr D Clark Ryan
Dr Christpher Ryan
Dr Patrick J Ryan
Dr Geoffrey Seet
Dr Wei Seto
Dr DC Shelton
Dr CY Skellern
Dr AJ Slater
Dr H Stalewski
Dr SL Stathis
Dr Mark Stretton
Dr Kerry Sullivan
Dr Ram Suppiah
Dr Michael J Thomsett
Dr Fiona Thomson
Dr Susan Thornton
DR DK True
Dr David Tudehope
Dr J Van Haeringen
Dr Rosslyn Walker
Dr Cameron Ward
Dr Timothy Warnock
Dr Kerri-Lyn Webb
Dr R Westmoreland
Dr Jasper Westhuyzen
Dr Neil R Wigg
Dr Michael Williams
Dr Sue Wilson
Dr Paul G Woodgate
Dr NF Woolfield
A/Prof Neil Wigg

SA

Dr Philips Adams
Dr George P Blake
Dr Hilary Boucaut
Dr R Burnell
Dr Richard Cockington
Dr Brian Coppin
Dr David G Cortis
Dr Jenny Couper
A/Prof Geoffrey Davidson
Dr Terrence Donald
Dr Philip Egan
Dr David S Everett
Mr WDA Ford
Prof Kevin Forsyth
Dr Andrew W Grieve
Dr Eric Haan
Dr TTS Han
Dr Bevan Headley
Dr Paul Henning
Dr Malcolm Higgins
Dr David JS Hill
Dr Anthony Hoby
Dr Anthony R Israel
Dr Judith Jaensch
Dr Diana Jolly
Dr Kenneth F Jureidini
Dr Jon Jureidini
Dr JD Kennedy
Dr David Ketteridge
Dr Maria Kirby
Dr Margaret A Kummerow
Dr Margeret Kyrkou
Dr Christopher Lamb
Prof David Lines
Dr Peter Marshall
Dr Victor Nossar
Dr Josie Nozza
Dr Maree O'Keefe
Dr Christopher Pearson
Dr Peter Petek
Dr Robert Pollinitz
Dr NK Poplawski
Dr Terence Pouras
Dr Michael Rice
Dr Malcolm Richardson

Congratulations to the highlighted winners of the 2004 wine prize.

Clinicians returning 100% (all months) of cards in 2004

Prof Don Robertson
Dr RN Russo
Dr A Sabato
Dr BR Saxon
Dr Michael Smiley
Dr Anthony Sparnon
Dr Billy S Tao
Dr Mark A Thesinger
Dr Andrew Tidemann
Dr Deirdre White

TAS

Dr Kenneth Armstrong
Dr Christopher Bailey
Dr Edmond Fenton
Dr Patrick Fernando
Dr Peter J Flett
Dr Evelyn Funk
Dr Elizabeth Hallam
Dr SJ Parsons
Dr Mark Pascoe
Dr Margaret Phelan
Dr AW Shugg
Dr Ian Stewart
Dr Michelle Williams

VIC

Dr Roger Allen
Dr David Amor
Dr David S Armstrong
Dr A Auldist
Dr Gordon Baikie
Dr E Bajraszewski
Dr David Bannister
Dr Charles Barfield
Prof Graeme Barnes
Dr Noel Bennett
Dr John M Bishop
Dr Fiona Brown
Dr Mary Brown
Dr William Capell
Dr J Carapetis
Dr David Carolane
Dr Elizabeth Carse
Dr Bronwyn Cathels
Dr C Chandran
Dr Caroline Clarke
Dr Chris Cooper
Dr s Costello
Dr John Court
Dr Noel Cranswick
Dr D Cutting
Dr Anita D'Aprano
Dr Margot Davey
Dr Peter Davis
Dr Martin Delatycki
Dr P Dewan
A/Prof Lex Doyle
Dr John Drew
Dr J John D'Souza
Dr Karen Leslie Dunn
Dr Kevin Dunne
Dr Peter J Eastaugh
Dr Maurice Easton
Dr Daryl Efron
Dr James Elder
Dr Adrien Elderhurst
Dr Lance Fong
Dr Peter Forrest
Dr J Freeman
A/Prof Nicholas Freezer
Dr Vanessa Gabriel
Dr Danny E Garrick
Dr Desmond Guppy
Dr C Hamilton
Dr Simon Harvey
Dr Richard Haslam
Dr Simon Hauser
Dr Sari Hayllar
Dr Katie L Heathershaw
Dr Robert Henning
Dr David J Hill
Dr Nigel W Hocking
Dr G Hogg
Dr James Holberton
Dr A Hopper
Dr Sian Hughes
Dr Ian Humphrey
Dr John Hunter
Dr F Jarman
Dr B Jenner
Dr David Johnson
Dr Hugh Kelso
Dr S Khosrowpanah
Dr Annette Knoches
Dr AJ Kornberg
Dr Renata Kukuruzovic
Dr Teresa Lazzaro
Dr Anthony Lewis
Dr Edwin Lowther
Dr Lionel Lubitz
Dr Catherine Marraffa
Dr R John Massie
Dr Catherine McAdam
Dr Brendan McCann
Dr Ian P Mcntyre
Dr KL McKay
Dr D McLaren
Dr James A McLellan
Dr Kathryn McMahon
Dr RB McNeill
Dr Joseph Mel
Dr JF Mills
A/Prof PT Monagle
Dr Anne Moulden
Dr Margot Nash
Dr MR Oliver
Dr Anne O'Neill
Dr Greg Pallas
Dr Chris Pappas
Dr Julian H Paxton
Dr Roderic Phillips
A/Prof D Reddihough
Dr Charles Richardson
Dr P Robinson
Dr Christine Rodda
Dr Katherine S Rowe
Dr R Neil Roy
Dr Luke Sammartino
Dr R Savarirayan
Dr Leslie J Sheffield
Dr Ian J Skelton
Dr Susan Skull
Dr Anne Smith
Dr Andrew Smith
Dr C Smith
Dr John Spensley
Dr TG Stubberfield
Dr R Stunden
Dr Joseph Tam
Dr Mimi Tang
Dr Russell Taylor
Dr Nick Thies
Dr Karin Tiedeman
Dr Brian J Timms
Dr Jacinta Tobin
Dr SC Treleaven
DR FCM Veit
Dr Rowan Walker
Dr Amanda Walker
Dr Keith Waters
Dr Robert Weintraub
Dr Anthony Weldon
Mr S Wivkramasinghe
Dr Martin Wright
Dr Harry Zehnwirth

WA

Dr AM Bullock
Dr Lesley Callingham
Dr Gervase Chaney
Dr Peter J L Chauvel
Dr Richard Christie
Dr Hock Leng Chua
Dr Barry Clements
Dr Charles Crompton
Dr Luigi D'Orsogna
Dr Harry Dumbell
Dr IJ Everitt
Dr Annette Finn
Dr Noel French
Dr Gary Geelhoed
Dr Elizabeth Green
Prof Sasson Gubbay
Dr Linda Harris
Dr Richard Hill
Dr Lawrence Hu
Dr CA Jeffries-Stokes
Dr Kay H Johnston
Dr Timothy Jones
Dr Bradley Jongeling
Dr C Kikiros
Dr Kay Johnston
Dr Barbara A King
Dr Geoffrey Knight
Dr Rolland Kohan
Dr Hement Kulkarni
Dr Peter N Le Souef
Dr JM Lesslie
Dr Cherry Martin
Dr Catherine Mews
Dr Mark Parker
Dr Aida Partridge
Dr Marianne Phillips
Dr Jacqueline Scurlock
Dr Jeffrey Stevens
Dr Jeffrey Tompkins
Dr Russell Troedson
Dr Kenneth Whitin

Thank you to all clinicians for your ongoing involvement and contribution to the APSU. We appreciate your support.

Congratulations to the highlighted winners of the 2004 wine prize.

We apologise to anyone inadvertently left off the list

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