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Preface

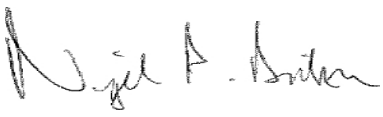
Welcome to the 2005 Annual Report of the New Zealand Paediatric Surveillance Unit (NZPSU). This is the eighth of its kind since the Unit was established in 1997.

The NZPSU was established with funding from the Ministry of Health in order to undertake surveillance of acute flaccid paralysis (AFP) for the Ministry of Health's National Certification Committee for the Eradication of Poliomyelitis (NCCEP). The opportunity was taken for the study of other uncommon high impact conditions, most of which has been undertaken by paediatricians with a particular research interest.

The ongoing success of the NZPSU is largely due to the high level of support from New Zealand paediatricians who have taken the time to provide information on the conditions under surveillance.

We would like to acknowledge the ongoing funding from the Ministry of Health.

Barry Taylor



Nigel Dickson

Amanda Phillips

Introduction

Surveillance is important to monitor both the incidence of emerging conditions and the effectiveness of prevention measures. The Paediatric Society of New Zealand (PSNZ) had for some years promoted the establishment of a unit that could regularly request specialist paediatricians to report on a number of conditions and this led to the establishment of the New Zealand Paediatric Surveillance Unit (NZPSU) in October 1997.

The aim of the NZPSU is to facilitate and improve the knowledge of uncommon high-impact childhood conditions in New Zealand. These conditions are of sufficiently low incidence or prevalence that case ascertainment on a national scale is needed to generate adequate numbers for meaningful study. The method was developed in the United Kingdom by the British Paediatric Surveillance Unit (BPSU) and has been used there since 1986. Subsequently, it has been introduced into several other countries, including Australia, and is used by some other specialist groups.

The core activities of the NZPSU are funded through a contract with the Ministry of Health to provide active surveillance of acute flaccid paralysis (AFP). The World Health Organisation (WHO), as part of the global eradication process, requires such surveillance to confirm New Zealand is free of poliomyelitis. Since the establishment of the NZPSU, the number of conditions under surveillance has increased and now includes eight high impact childhood conditions.

The NZPSU is a member of the International Organisation of Paediatric Surveillance Units (INoPSU).

Aims

The aims of the NZPSU are:

- To operate a system for monitoring acute flaccid paralysis, as part of the global certification of eradication of poliomyelitis, required by WHO.

- To facilitate national surveillance and improve the knowledge of uncommon high-impact childhood conditions in New Zealand.

How the Surveillance System Works

The method of surveillance is based on that developed in the United Kingdom in 1986 by the British Paediatric Surveillance Unit (BPSU). It has subsequently been used for the monitoring of rare childhood conditions in several other countries, including Australia, and also by other specialist groups.

Paediatricians in New Zealand gave their support to the surveillance system after the concept was discussed at several annual meetings of the Paediatric Society of New Zealand. A database of eligible clinicians, which included all paediatricians and other specialists working predominantly with children, was developed using the specialist register and the membership list of the Paediatric Society. All eligible clinicians were contacted and invited to participate. Those who agreed were provided with study protocols, which included definitions of the conditions under surveillance, specific reporting instructions, and a contact telephone number. Efforts are made to keep up-to-date with the paediatric specialist work force.

Every month participants are sent either a reply-paid card or an email (depending on their preferred method of reporting) to report whether in the previous month they have seen any cases of the conditions under surveillance. However, cases of AFP are also required to be reported immediately by phone to the NZPSU. When a case of any of the conditions is reported, the reporting clinician is sent a short questionnaire to complete on the case. The identity of the case remains anonymous. Duplicate notification is recognised by a code derived from the child's initials and date of birth.

Where possible, cases are regularly compared with other data sources such as hospital discharge data, notifications to the local Medical Officer of Health, and the New Zealand AIDS Epidemiology Group.

It is envisaged that some of the conditions under surveillance will be ongoing, while others will be for a finite period, usually two or three years.

Regular surveillance reports are made to the Ministry of Health specifically updating the progress with AFP surveillance.

Inclusion of New Conditions

A Scientific Review Panel (SRP) has been established primarily to consider the inclusion of new conditions into the scheme (see *Table 1* for details on members of the SRP). A study is eligible for consideration in the scheme if the condition of interest is:

- a relatively uncommon high-impact childhood condition (or an uncommon complication of a more common disease); and
- of such a low incidence or prevalence as to require ascertainment of cases on a national scale in order to generate sufficient numbers for study; and
- the SRP may also consider inclusion of short-term or geographically limited studies of comparatively more common conditions.

It is important for the success of the scheme that the workload of the mailing list is kept to a minimum. Accordingly, the SRP must be certain that studies conducted through the NZPSU are well designed and worthwhile. The SRP will take into consideration the scientific interest and public health importance of the proposed study, its methodology, and the suitability of the condition for ascertainment through the NZPSU scheme. Studies depending on immediate reporting and/or sample collection, or requiring the participation of other specialties, are less likely to be suitable.

***Table 1*: The Members of the NZPSU Scientific Review Panel (SRP)**

Member	Institution
Professor Barry Taylor	Dunedin School of Medicine
Dr Nigel Dickson	Dunedin School of Medicine
Dr Alison Roberts	Ministry of Health
Professor Elizabeth Elliot	Australian Paediatric Surveillance Unit
Dr Jeff Brown	Palmerston North Hospital
Professor Brian Darlow	Christchurch School of Medicine
Professor Diana Lennon	University of Auckland

Surveillance Activities in 2005

In 2005, 208 clinicians participated in the system. The average response rate to the monthly report card/email was 93%. We are very pleased with the ongoing high response rate from the whole of the country.

Respondent Workload
Minimising the extra workload that the system imposes on paediatricians is a key factor for its success. The range of conditions under surveillance and their incidence needs to be kept under review.

Table 2 shows the percentage of clinicians on the mailing list that reported cases during 2004 and 2005. The table shows that in 2005, 142 of the participants did not report any cases, with three reporting five or more, compared to four in 2004.

***Table 2*: Respondents' Workload 2004 & 2005**

Notifications	2004		2005	
	No.	%	No.	%
None	127	61	142	68
One	38	18	37	18
2-4	41	19	26	12.5
5 or more	4	2	3	1.5

In 2005 the NZPSU monitored 10 uncommon childhood conditions *(Table 3).* Some of the protocols and questionnaires used were adapted from those used by the Australian Paediatric Surveillance Unit.

Table 3: **Conditions Under Surveillance in 2005**

Condition	Surveillance Started	Surveillance Ended	Principal Investigators
Acute flaccid paralysis	October 1997	Ongoing	Dr Nigel Dickson Dr Paul Shillito
Haemolytic uraemic syndrome	January 1998	Ongoing	Dr William Wong
Congenital rubella syndrome	January 1998	Ongoing	Professor Diana Lennon
Perinatal HIV exposure	January 1998	Ongoing	Dr Nigel Dickson Dr Lesley Voss
Vitamin K deficiency bleeding	January 1998	Ongoing	Professor Brian Darlow
Prolonged infantile cholestasis	January 2002	December 2005	Dr Alison Wesley
Inborn errors of metabolism	January 2004	Ongoing	Dr Nikki Kerruish Dr Callum Wilson
Pertussis, hospitalised <12 months	July 2004	July 2005	Associate Professor Cameron Grant
Foregut and hindgut malformations	January 2004	August 2005	Dr Michael Sullivan
Pneumococcal meningitis	April 2005	Ongoing	Professor Diana Lennon

Brief reports on ongoing studies

ACUTE FLACCID PARALYSIS (AFP)

Dr Nigel Dickson

Ongoing study started in October 1997

INTRODUCTION

To confirm the absence of poliomyelitis WHO requires a surveillance system to be in place:

1. That captures an annual incidence of acute flaccid paralysis (AFP), not due to poliomyelitis, of at least one per 100,000 children < 15 years.

2. In which 80% of cases of AFP have two stool samples taken at least 24 hours apart, within 14 days of onset tested negative for wild polio virus in a WHO-accredited laboratory.

Telephone notification of all cases of AFP is required by the NZPSU to ensure that the necessary stool containers are dispatched in time to the notifying paediatrician.

KEY RESULTS FOR 2005

- There were nine cases notified to the NZPSU in 2005.
- Information has been obtained on all of these children including follow-up information two months after diagnosis.
- All nine AFP cases were from the North Island.
- Six males, three females
- Age range 1 month to 14 years, median age 6 years.
- No seasonal variation.
- The overall incidence was 1.1 per 100,000 children < 15 years.
- A diagnosis of Guillain-Barré Syndrome (GBS) has been made in five of these cases, Ewings Sarcoma in one, Spinal Muscular Atrophy in another, with the remaining two cases unknown, but not polio.
- All nine cases have been discounted as Polio by the National Certification Committee for the Eradication of Polio (NCCEP).
- Timely analysis (< 14 days after onset paralysis) of stool samples satisfying the WHO criteria was only complete for two of the nine children.

***Table 4*: Percentage of AFP cases with adequate stool samples (or otherwise)**

Category	Stool samples	
	No.	%
2 stool samples within 14 days of onset of paralysis	2	22
2 stool samples, but one or both not within 14 days of onset of paralysis	1	11
1 stool sample	2	22
No stool samples	4	44

COMMENT

The system successfully captured the required rate of AFP. However, the rate of stool testing (22%) is not meeting the WHO criteria (80%). The NZPSU appreciates the support from clinicians in making telephone notifications of AFP, and attempts to ensure that timely stool specimens are sent to ESR for appropriate testing.

Ongoing surveillance of AFP, even though the WHO believes Polio to have been eradicated from the Western Pacific region, is likely to be required for some years. This will require the continued telephone notification of all cases of AFP, including those with a definitive diagnosis such as Guillain-Barre syndrome etc. A challenge has always been to utilise a non-specific case definition – such as 'acute flaccid paralysis' – in a health system where a more definitive diagnosed for children with such symptoms is likely to be made.

HAEMOLYTIC URAEMIC SYNDROME (HUS)

Dr William Wong

Ongoing study started in January 1998

KEY RESULTS FOR 2005 COHORT

- 10 case of HUS (M=F) was reported, 8 had a diarrhoeal prodrome and 2 were atypical (1 pneumococcal associated, 1 ADAMST13 deficiency)
- Geographic distribution of cases ranged from Kaitaia to Dunedin
- Incidence is 0.9 per 100,000 < age 15 years
- Median age at presentation 2.2 years, range 0.5 to 4.4years
- 5/8 had E coli 0157 isolated from their stools
- 4/10 needed acute peritoneal dialysis for an average of 13 days
- All patients regained renal function to come off dialysis, but 2 patients on short term follow up have persistent urinary abnormalities or impaired glomerular filtration rate.

CONGENITAL RUBELLA SYNDROME (CRS)

Professor Diana Lennon

Ongoing study started in January 1998

We have not provided a report for Congenital Rubella as there were no cases reported in 2005.

VITAMIN K DEFICIENCY BLEEDING (VKBD) 2004 & 2005 REPORT

Professor Brian Darlow

Ongoing study started in January 1998

There were three notifications of VKDB received in 2004; one report was not valid, hence there were two valid cases, although both were only "probable".

- One cases involved an infant born at 39 weeks gestation at a Birthing Centre. The parents had declined vitamin K. At six hours of age the infant had an apnoeic spell and subsequently had further seizures. The infant was given i.m. vitamin K prior to transfer to hospital and coagulation studies being performed. An MRI scan showed both extracerebral haemorrhage and temporal lobe changes consistent with haemorrhagic infarction. At four months of age, neurological examination was normal but the infant was lost to further follow up.

- One case involved an infant born at 32 weeks gestation. Vitamin K was recorded as having been given i.m. following birth. At 8 days of age the infant suffered a massive intracranial haemorrhage involving one hemisphere. Coagulation studies were abnormal and interpreted as consistent with VKDB and normalized 12 hours after FFP and i.m. vitamin K. Liver function tests were normal. The infant died.

There were no notifications of VKDB received in 2005. This is the first year since surveillance began in 1998 that there have been no reported cases.

One abstract has been published after 5 years of surveillance (Darlow BA. Vitamin K deficiency bleeding (VKDB) in New Zealand infants: results of surveillance over five years (1998 to 2002). *Pediatr Res* 2004;56;474) and a full report is now being prepared.

PERINATAL EXPOSURE TO HIV

Dr Nigel Dickson, Dr Lesley Voss

Ongoing study started January 1998

In 2005, there were 20 reports to the NZPSU of infants/children born to women infected with HIV. Of these:

- 2 were of perinatally-infected child born overseas.
- 3 were of children infected with HIV born in New Zealand in 2005 to women whose HIV had not been diagnosed prior to delivery.
- 3 were of older siblings of these infected children
- 1 was of an uninfected child whose mother was diagnosed after giving birth
- 1 was of an uninfected child born in 2004

Therefore there were reports of 10 infants born in New Zealand in 2005 to women with HIV diagnosed prior to giving birth. Of these:

- 3 were born in Auckland, 5 in other North Island centres, and 2 in South Island centres.
- 5 were born to mothers whose HIV had been diagnosed before her pregnancy and 5 during her pregnancy.
- 8 of the mothers were of African and 2 of European ethnicity.
- All the mothers were given antiretroviral treatment during pregnancy, 7 gave birth by caesarean section, and none of the babies were breastfed.
- None of these 10 children are believed infected with HIV (although some are still awaiting final confirmation)

These figures show the success that can be achieved in the prevention of perinatal transmission of HIV when the infection is diagnosed in a mother prior to delivery. It would be expected to occur in about a quarter of affected pregnancies without specific measures (antiretroviral therapy, care with means of delivery, and avoidance of breast-feeding) being taken.

INBORN ERRORS OF METABOLISM (IEM)
(Urea cycle, amino acid, organic acid disorder or fatty acid oxidation defect).

Dr Nikki Kerruish, Dr Dianne Webster, Dr Callum Wilson, Dr Esko Wiltshire

3 year study commenced January 2004

There were 8 notifications during 2004 that fulfilled the protocol criteria and 1 year follow-up questionnaires have been returned for all cases. Brief details are given below.

Disorder	Age at diagnosis	Reason for diagnosis	1 year follow-up
Holocarboxylase synthetase deficiency	newborn	Family history, clinical symptoms	Died age 3 months. Metabolic decompensation.
Hyperphenylalaninaemia	<1 month	Newborn screening	Remains well. No therapy required.
PKU	<1 month	Newborn screening	Remains well. PKU formula, low protein diet.
Ornithine transcarbamylase deficiency	prenatal	Family history, prenatal molecular genetic testing	Died age 2 weeks. Group B Streptococcal septicaemia, metabolic decompensation.
Ornithine transcarbamylase deficiency	14 years	Clinical symptoms (acute confusional episode)	Remains well. 1 episode vomiting and lethargy. Sodium benzoate therapy.
PKU	< 1 month	Newborn screening	Remains well. PKU formula, low protein diet.
Beta ketothiolase deficiency	6 months	Clinical symptoms (severe ketosis, out of keeping with underlying mild infection)	Generally well. 3 admissions with URTI/LRTI requiring oral/IV fluid.
Multiple acyl Co A dehydrogenase deficiency (glutaric acidaemia type 2)	1 month	Clinical symptoms (neonatal encephalopathy)	Spastic quadraparesis, bulbar palsy, hydrocephalus, paroxysmal dystonia and visual impairment. Multiple hospital admissions.

There were 5 notifications that fulfilled the protocol criteria during 2005 and questionnaires have been returned for all cases. Brief details are given below.

Disorder	Date of birth	Age at diagnosis	Sex	Region	Reason for diagnosis
Maple syrup urine disease	14/2/05	2 weeks	f	Auckland	Admitted with encephalopathy, positive newborn screen result available next day
Non-ketotic hyperglycinaemia	16/8/05	1 week	f	Auckland	Lethargic from birth with seizures, hypotonia and apnoeas. Typical amino acid profile (plasma and CSF).
Glutaric aciduria type 1	25/11/04	5 months	f	Christchurch	Choreoathetosis and dystonia following febrile illness (enteroviral encephalitis). Typical organic acid profile.
Ornithine transcarbamylase deficiency	25/9/05	10 years	f	Hawkes Bay	Episode of confusion and hallucinations. Typical amino acid profile.
Non-ketotic hyperglycinaemia	12/9/05	1 week	f	Hamilton	Seizures, lethargy and apnoeas from birth. Typical amino acid profile (CSP and plasma).

A child from Hawkes Bay was diagnosed with PKU through the newborn screening programme in Dec 2005, and hence known to some of the investigators despite not being reported to the NZPSU.

Points of interest

There have been three new cases of PKU during the two year period (2004 and 2005). This is slightly less than the expected incidence of approximately 1 in 20,000 (National Testing Centre Report 1996).

There were no reports of medium-chain acyl Co-A dehydrogenase deficiency (MCAD) during the 2 year period. It is possible that this reflects under-diagnosis. MCAD is the most frequent inherited fatty acid oxidation disorder with an incidence of about 1 in 10,000–20,000. Onset of clinical signs and symptoms is usually during a child's first three years of life. There is no typical

presentation of MCAD but the most common features include vomiting and lethargy. Hypoglycemia, encephalopathy, respiratory arrest, hepatomegaly, seizures, coma, apnoea, cardiac arrest, and sudden death have also been documented. Long-term outcomes may include developmental and/or behavioural disabilities and at the initial presentation, the mortality risk is as high as 25%. Laboratory findings include hypoketotic hypoglycemia, with hyperammonaemia and abnormal liver function. Treatment of MCAD deficiency involves avoidance of fasting and ensuring adequate glucose intake during illnesses.

There were two cases of non ketotic hyperglycinaemia in 2005 (and a further two so far in 2006). This is unusual as the incidence typically approximates 1 in 100,000. One of these cases has been reported as an “instructive case” in the Journal of Paediatrics and Child Health. [1]

NB: All figures should be treated with caution due to the size of the birth cohorts (approximately 58,000pa) and relative rarity of these conditions.

REFERENCE

1. Wallace AH, Manikkam N, Maxwell F. Seizures and a hiccup in the diagnosis. J Paediatr Child Health 2004;40(12):707-8.

PNEUMOCOCCAL MENINGITIS IN NEW ZEALAND CHILDREN UNDER 15 YEARS OF AGE

Professor Diana Lennon, Dr Rachel Webb

2 year study started in May 2005

PRIMARY STUDY AIMS

- To determine the incidence of pneumococcal meningitis in New Zealand children
- To determine the frequency of different serotypes causing pneumococcal meningitis

SECONDARY STUDY AIMS

- To determine the pattern of referral of CSF isolates to ESR for sero-typing
- To gauge the burden of paediatric pneumococcal meningitis on the New Zealand health system, and therefore to contribute to an economic analysis to assess the cost-benefit of introducing 7-valent pneumococcal vaccine into the routine New Zealand Childhood Immunisation Schedule

CASE DEFINITION

The case definition for pneumococcal meningitis has been revised to take into account molecular methods (PCR) for the diagnosis of *Streptococcus pneumoniae* infection.

STUDY PROGRESS

Since data collection began on 1 May 2005, 34 notifications (excluding known duplicates) have been made to the NZPSU. Auckland clinicians made the highest number of notifications to NZPSU (16) but had a very low rate of completing questionnaires (3). To date 14 completed questionnaires have been returned of which 13 represent definite cases and 1 a possible case. Thus the available information on the 13 reported definite cases is likely to be a significant under-representation of the true number of cases.

A second mail-out of questionnaires to clinicians who have not returned their original questionnaire is currently underway. Arrangements are also underway for the questionnaire to be placed on the NZPSU / Paediatric Society website. Information from the ESR Invasive Pathogens Laboratory with regards to serotypes of invasive isolates which have been referred to ESR is also awaited. At the end of the data collection period (1st May 2007) information on hospital discharge and mortality data across the study period will be sought for cross-referencing purposes.

INFORMATION ON NOTIFIED CASES

Of the 13 reported definite cases, 4 came from Capital and Coast DHB, 3 from Auckland DHB, 2 from Hutt Valley DHB, and 1 case each from Canterbury, Lakes District, Counties Manukau and Waikato DHBs.

All but one of the 13 definite cases was under two years. 3 children were aged 0 – 5months, 4 children aged 6 – 11months, 5 children aged 12 – 23months, and 1 child was aged 2 –5years. Ethnicity data was available on all 13 cases: 8 children were NZ European, 4 NZ Maori and 1 a Pacific Islander. Length of hospital stay among reported cases ranged from 4 – 28 days. The median length of stay was 10 days.

Two out of 13 children had underlying medical conditions which may have predisposed them to invasive pneumococcal disease (1 = CSF leak secondary to skull fracture, 1 = cerebral palsy and recurrent chest infections) The complication rate was high, with SIADH reported in 4 out of 13 cases. 3 children were admitted to ICU or PICU. 1 child required neurosurgical intervention and the same child died .1 of the surviving children has ongoing neuro-developmental problems.

Audiology screening has been performed in 7 of the 12 surviving children with no cases of sensori-neural hearing loss reported among these 7 children.

REVISED ESTIMATE OF CASE NUMBERS AFTER REVIEW OF LABORATORY DATA FROM AUCKLAND AND MIDDLEMORE HOSPITALS

In an attempt to clarify the true number of cases coming from the Auckland region, information on positive CSF and Blood cultures was sought from Auckland and Middlemore hospital laboratories and cross-referenced with the NZPSU notifications.

This resulted in a total of 24 cases nationwide – a significantly higher number of total cases over the same time-period. This number is likely to under-represent the true number of cases as a number of questionnaires from other centres have not yet been returned.

Maori and Pacific children were over-represented with 6 NZ Maori, 4 Tongan and 2 Samoan children along with 12 NZ European children making up the 24 cases. 22 of the 24 cases were aged under two years. 12 children came from the greater Auckland region (6 from Auckland and 6 from Counties Manukau), 7 from the greater Wellington region (5 from Capital and Coast and 2 from Hutt Valley), with 1 each from Lakes, Waikato and Bay of Plenty DHBs. Only 2 reported cases came from the South Island (Canterbury DHB).

There were significant numbers of complications, with 6 children developing SIADH. 4 children were admitted to PICU / ICU and 4 underwent neurosurgical intervention. 2 children died. 2 surviving children have developmental problems. Audiology information was available on 14 children and 3 out of 14 have some degree of sensori-neural hearing loss.

CONCLUSIONS

Despite the current limitations with regards to incomplete data, the results from this study to date confirm that pneumococcal meningitis is a significant problem in New Zealand children, with major attendant morbidity and mortality. Information from ESR with regards to the serotypes of isolates is awaited. It is anticipated that the currently licensed (but not yet routinely scheduled) 7 valent conjugate pneumococcal vaccine (Prevenar) would cover most of the serotypes occurring in pneumococcal meningitis cases in New Zealand and hence routine vaccination would be expected to greatly reduce the incidence of meningitis and other forms of invasive pneumococcal disease, as has been the case in other countries where routine vaccination is already undertaken.

Final Reports for Completed Studies

1. PROLONGED INFANTILE CHOLESTASIS

Dr Alison Wesley, Simon Chin, Jason Yap, Helen Evans
Department of Paediatric Gastroenterology, Starship Children's Hospital, Auckland

Study commenced 2002 Completed 2005

AIMS
To determine the incidence of infantile cholestasis and its causes in New Zealand and to compare with other parts of the world.

METHODS
Using the NZPSU system paediatricians were asked to notify cases of infant cholestasis (2 weeks post term to 6 months of age) during the years 2004 and 2005, and asked to complete a questionnaire to provide information about the diagnoses, clinical presentation, and diagnostic tests undertaken.

RESULTS
A total of 94 notifications were made, but after allowing for duplicate notifications and removing infants – all preterm at diagnosis - who did not satisfy criteria,54 returned questionnaires were able to be analysed. Overall, 59% (32/54) of the notified infants were male.

The aetiology of the cholestasis was extra hepatic biliary atresia (EHBA) 31% (17/54), parenteral nutrition related 20% (11/54), sepsis 5.5% (3/54), bile duct paucity 5.5% (3/34), metabolic/genetic 5.5% (3) and idiopathic 31% (17). 59% of notified infants were male.

Idiopathic included diagnoses reported as 'neonatal hepatitis', inspissated bile, gallstones, idiopathic hepatitis and intrahepatic cholestasis. Paucity of intrahepatic ducts was a histological diagnosis, and none of these infants had other clinical features which would have enabled identification as Alagilles Syndrome. Only one child was identified as having Alpha-1-Antitrypsin deficiency.

16 infants with EHBA underwent portoenterostomy at an average age of 67 days (9-113). One infant seen for the first time at age 5.5 months did not undergo surgery because of the late presentation. Of these children, one has died of severe gastrointestinal bleeding while awaiting transplant. 8 have received liver transplants at a mean age of 18.5 months (0.5 to 2.8yrs) and 8

remain well. One child with a genetic diagnosis was considered for transplant but because of sepsis could not be listed and has died.

Overall, 68% of infants had hida scans as part of their investigations and 55% had liver biopsies. Biopsies were rarely performed when the diagnosis was parenteral nutrition related but frequent for EHBA and less commonly for idiopathic causes.

Infections which were tested for included Toxoplasmosis (68%). CMV (80%), EBV(37%), Hepatitis A (53%), Hepatitis B (68%), Syphilis (15%) and Herpes (30%). Evidence of CMV infection was determined in 5 infants, but no other congenital infections were detected. Of these 5 infants, 1 also had EHBA, 1 a group B streptococcal infection, I a diagnosis of idiopathic neonatal hepatitis and two were diagnosed with a CMV hepatitis.

CONCLUSIONS

The commonest causes of Infantile Cholestasis were EHBA (31%) and idiopathic (31%). It is possible that more definitive diagnoses may have been possible if liver biopsies had been performed more often in the idiopathic group and follow up of these infants may help in determining whether more specific diagnoses were made at a later date. Metabolic/genetic diagnoses are less common than reported from Australia (5.5% *v* 23%).[2] Parenteral nutritional related cholestasis, as in Australia was a common cause of cholestasis, in both countries accounting to 20% of cases. Congenital infection was an uncommon cause of infantile cholestasis (9.5%).

Infants in New Zealand with biliary atresia are receiving their Kasai operation at a later age than infants in the UK (67 days *v* 54 days)[1].

There was no difference in the ethnicity of infants with either EHBA, or Idiopathic neonatal liver disease.A considerably longer study would be needed to obtain a true incidence of metabolic or genetic liver disease.

REFERENCES

1. McKiernan P.J. et al. Lancet (2000) 355: 25-29.
2. Storman et al, J.Pediatr Child Health(2001) 37, 47-50.

Table 1:1 Ethnicity and investigation of infants with Infantile Cholestasis

	EHBA	Idiopathic	Parenteral nutrition related	Sepsis	Bile Duct Paucity	Metabolic/genetic
Number	17	17	11	3	3	3
Percentage	31	31	20	5.5	5.5	5.5
% Polynesian	47	47	27	100	33	66
% Liver biopsy	82	53	9	66	100	66
% Pale stools	82	29	72	100	66	66
% *Hida* scan	94	64	36	66	100	100

2. INFLAMMATORY BOWEL DISEASE (IBD)

Dr A Wesley, Dr S Mouat, Dr J Yap, Dr S Chin

Starship Children's Hospital, Auckland

3 year study, completed December 2003

AIMS

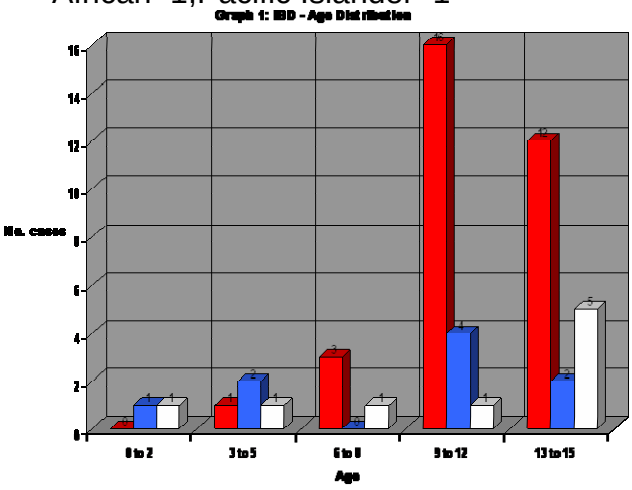
The aims of the study were to investigate the incidence, presentation and initial management of paediatric inflammatory bowel disease in New Zealand

RESULTS

There were 52 reported cases during the 24 month period of study, 21 in 2002 and 31 in 2003, and overall incidence of 2.9 cases per 100,000 per year.

Demographic characteristics

- 30 males, 22 females
- Age range at diagnosis: 0.25-15.4 years, mean 11.0 years
- Ethnicity: European=39, Maori=2, Indian=2, Middle Eastern=2, African=1,Pacific Islander=1



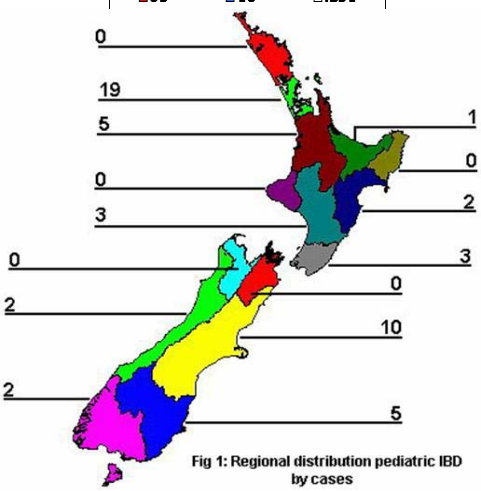


Fig 1: Regional distribution pediatric IBD by cases

Of the 52 children notified, 65% (34/52) had Crohn's disease(CD), 17% (9/52) ulcerative colitis(UC), and the remaining 17% (9/52) had chronic inflammation of the colon, without small bowel involvement and no definitive histological evidence to favour CD or UC, and were included in an undetermined category.

1. Crohn's disease (CD) (n=34):

Estimated incidence of CD was 1.9 cases per 100,000 per year

Mean age of diagnosis 11.8 years and mean time from onset of symptoms to diagnosis 7.2 months

Symptoms at presentation

- abdominal pain (74%)
- weight loss (50%)
- rectal bleeding (47%)
- diarrhea (41%)
- 5 cases had perianal disease at presentation

Growth

Mean weight Z score = -1.02
Mean height Z score = -0.36

Laboratory results (mean values)

- Haemoglobin 106gm/L
- Platelet count 524
- ESR 32.5mm/hr
- Albumin 31gm/L.

Investigations

- Colonoscopy was undertaken on 91%, 76% also underwent upper endoscopy.
- Barium contrast studies were done on 68%

Distribution of disease

- ileocolonic-38%
- colonic-50%
- 70% also had upper GI tract involvement

Drug treatment

- 5 ASA preparation- 85%
- Steroids- 73%
- Azathioprine-32%
- Antibiotics- 18%

Surgical intervention
7 cases required surgery for perianal involvement, bowel obstruction, or intractability

2. Ulcerative colitis (UC) (n=9)

Estimated incidence of UC was 0.5 cases per 100,000 per year

Mean age at diagnosis 9 years, range 0.25-15.4 years and mean time from onset of symptoms to diagnosis 4.8 months

Presenting Symptoms
Bloody diarrhoea was the most common symptom (66%)

Growth
Mean weight Z score = +0.02
Mean height Z score = -0.1

Laboratory results (mean values)
- Haemoglobin 110gm/L
- Platelet count 491
- ESR 37mm/hr
- CRP 1.8
- Albumin 38gm/L.

Distribution of disease
78% had pancolitis
22% had (L) sided disease

Drug treatment
- 5 ASA drugs 100%
- Steroids 44%
- Azathioprine 11%

No cases required subsequent surgery

3. Undetermined (n=9)

Estimated incidence of undetermined IBD was 0.5 cases per 100,000 per year

The mean age was 9 years (youngest = 3 months) and the mean delay from presentation to diagnosis 1.2 years.

Growth
Mean weight Z score = +0.8.

Laboratory results (mean values)
- Haemoglobin 119 gm/L

- Platelet count 433
- ESR 21 mm/hr
- CRP 16
- Albumin 38gm/L.

Distribution of disease
- 67% had pancolitis.

Drug treatment
- 5 ASA drugs 89%
- 1 child was started on an elemental diet

SUMMARY OF REPORT

The overall incidence of IBD of 2.9 cases per 100,000 per year is lower than other countries such as Australia (5.4 cases per 100,000 per year). This may reflect in part the ethnic background of the population of New Zealand and in particular the North Island, as no patients with Crohn's disease or ulcerative colitis were Maori or Pacific Island ethnicity. The incidence of IBD on the other hand in the South Island (5.1 per 100,000 per year) was similar to Australia, North America and Europe.

The majority of patients had Crohn's disease (34/54). The peak age group for CD (9-12 years) and the mean delay from onset of symptoms to diagnosis (8.4 months) was comparable to overseas data. Clinical presentation appeared similar to overseas studies. Initial treatment with 5- ASA drugs(88%), steroids (75%) were preferred to enteral nutrition treatment (12%). 32% subsequently went onto azathioprine. 21% required surgery mainly for perianal disease.

The mean age of diagnosis for ulcerative colitis was similar(9 years), with a shorter mean delay from onset of symptoms to diagnosis (4.8 months). Most patients with UC had pancolitis (78%). All were treated with 5-ASA drugs and 44% were also treated with steroids.

The group with undetermined inflammatory bowel disease had a mean delay from presentation to diagnosis of 1.2 years, and were treated mainly with 5-ASA drugs.

ACKNOWLEDGMENTS

We would like thank all paediatricians who participated in providing data for this study.

3. PERTUSSIS, HOSPITALISED < 12 months

Associate Professor Cameron Grant, Dr Rebecca Somerville
Starship Children's Hospital, Auckland

1 Year Study, commenced July 2004, completed July 2005

This is a summary of the completed study. The full report can be viewed on the Paedatric Society Website: www.paediatrics.org.nz and follow the NZPSU link.

INTRODUCTION

Pertussis in infants continues to be a difficult disease to manage. In comparison with other developed countries New Zealand has a large pertussis disease burden. These comparisons are based upon data collected by passive surveillance. Passive surveillance underestimates pertussis disease incidence. The primary aim of this study was to use active surveillance to determine the burden of disease among infants hospitalised with pertussis in New Zealand, with particular emphasis on duration of hospitalisation, need for intensive care, death and disability.

METHODS

By utilising the notification system of the New Zealand Paediatric Surveillance Unit active clinical and laboratory surveillance was established in 18 of the 21 District Health Board (DHB) regions in New Zealand. These DHBs provided health care for 93% of the live births in New Zealand in 2001. Ethical and managerial approval was obtained for 12 months of active surveillance from 1st August 2004 to 31st July 2005. The case definition used to define study eligibility was any infant (less than 12 months of age) admitted to hospital in the previous month with a diagnosis of pertussis, based on either laboratory confirmation or clinical features.

RESULTS

i. 110 infants were identified, equivalent to a hospitalisation rate of 196 per 100,000. The median age was 61 days. Twenty four percent of the infants had a co-morbidity.
ii. The hospitalisation rate (per 100,000) varied with ethnicity being 296 for Maori, 358 for Pacific and 117 for infants of European and other ethnic groups.
iii. The age of infants hospitalised with pertussis varied with ethnicity with a larger of the Pacific infants (46%) compared with European/other infants (13%) being less than six weeks of age. After adjustment for age the risk of hospitalisation remained increased for both Pacific and for Maori infants.
iv. Forty-four percent of the hospitalised infants were from households in the most socially deprived quintile. A larger proportion of both Pacific and Maori infants were from more socially deprived households. After

adjustment for social deprivation, the risk of hospitalisation remained increased for Maori but not for Pacific infants.

v. Thirty eight (46%) of the 82 infants old enough to be immunised had received no immunisations. Despite being more likely to be immunised Pacific children had a higher hospitalisation rate with this being due to a larger proportion of them acquiring pertussis prior to six weeks of age.
vi. Apnoea and cyanosis were key case defining symptoms particularly in those infants three months of age and younger.
vii. Among the 110 cases there was one death, 10 readmissions, eight infants admitted to intensive care units and six infants who experienced a disease complication. A total of 683 inpatient days including 51 intensive care unit days were utilised in caring for these 110 infants.
viii. Eighty two percent of the 92 infants for whom this information was available, had contact with someone with cough. Ninety two percent of these were household contacts. Frequently there were multiple household contacts. Approximately half of the contacts were adults.
ix. Laboratory confirmation of diagnosis was achieved for 81%. Culture had a sensitivity of 41% and polymerase chain reaction (PCR) a sensitivity of 65%. The PCR results were available after a median of three days compared with six days for culture.
x. Based upon a capture-recapture analysis that compared the 110 cases identified by this active surveillance with the 106 cases identified by passive notification we estimated that there were between 126 and 136 infants hospitalised with pertussis during this time interval.

CONCLUSIONS

i. Infant pertussis hospitalisation rates in New Zealand are high in comparison with other developed countries. One quarter of those hospitalised are too young to have received any immunisations. This age factor was a particular issue for Pacific infants and accounts for much of the excessive disease burden that occurs in Pacific infants despite them being more completely immunised. Other household members with cough are present in the majority of households where these infants live with approximately half of these contacts being adults.
ii. These two factors, young age of infants when pertussis is acquired and large proportion of coughing contacts being adults, indicates a need for immunisation of adults that is especially aimed at improving immunity during child bearing years.
iii. Apnoea and cyanosis are key case defining symptoms especially in those three months of age and younger. Apnoea should remain a case defining symptom. Cyanosis should become a case defining symptom.
iv. Based upon a capture-recapture analysis under reporting of the number of infants hospitalised with pertussis appears modest.

4. IDIOPATHIC NEPHROTHIC SYNDROME (INS) IN NEW ZEALND CHILDREN, DEMOGRAPHIC, CLINICAL FEATURES,INITAIL MANAGEMENT AND OUTCOME AFTER 12 MONTHS FOLLOW UP:RESULTS OF A 3-YEAR NATIONAL SURVEILLANCE SURVEY

Dr William Wong

3 year study, completed 2003

ABSTRACT

Aim
To describe the demographic, clinical features, steroid response, histopathology and complications of all children diagnosed with idiopathic nephrotic syndrome (INS) in New Zealand over a 3 year period.

Methods
A questionnaire seeking relevant clinical information was sent to all paediatricians who reported a new case of nephrotic syndrome to the New Zealand Paediatric Surveillance Unit. A follow up questionnaire was sent to reporting paediatricians after the first 12 months of follow up.

Results
The incidence was 1.9 children per 100,000 under age 15years. There was no significant difference in INS between ethnic groups. 80.4% were steroid responsive with median time to response of 8.4 days and mean time to relapse was 15.1+/-12.1 weeks (10.1-19.8 95%CI). Follow up at 12 months after diagnosis showed that two thirds were either steroid dependent or frequent relapsers. Steroid resistance patients had a more variable course with some developing chronic renal failure and other remaining persistently nephrotic.

Conclusion
The incidence and outcome of children with INS is similar to overseas studies. A large variety of steroid treatment regimens were noted.Current evidenced based guidelines to treat INS were used infrequently.

The incidence of idiopathic nephrotic syndrome (INS) in New Zealand children is not known. Of the overseas incidence studies (US [1,2] , UK [3,4]), only one was population based[1]. In the 1950s, the annual incidence of nephrotic syndrome in children aged below 16 years in the USA, derived from the Erie County Survey was approximately 2 per 100,000 children and the cumulative prevalence was about 16 per 100,000[1]. The incidence was lower in white children (1.9 per 100,000) than in non-white children (2.8 per 100,000) though

the numbers of non-white children were small and the incidence was higher in lower socio-economic groups. Similar results were obtained in the Ohio Study[2], where case ascertainment was by questionnaire requesting data on hospitalised cases seen between 1944 and 1953. A retrospective study of hospital records in Birmingham, UK[3] revealed that the annual incidence of nephrotic syndrome was 2.6 per 100,000 children for European children, 3.4 for Afro-Caribbean children and 16.9 for Asian children from the Indian subcontinent. This study assumed that all children with nephrotic syndrome (NS) were treated in the major hospitals. A study from Leicestershire, published at about the same time found similar results[4]

There is no information on the epidemiology or management of INS in New Zealand children. In 1998, Simpson et al[5], published a biopsy series of children with INS and highlighted some of the differences between his group compared with overseas studies. A prospective study between 1st July 2001 and 1st July 2004 was undertaken to define demography, clinical features, complications and treatment in children newly diagnosed with INS in New Zealand.

THE AIMS OF THE PRESENT STUDY WERE

1. To compare the age, sex, and ethnicity of children with steroid responsive and steroid resistant INS
2. To describe renal histo-pathology seen in children with steroid resistant NS
3. To describe the distribution of infrequent relapsers, frequent relapsers and steroid dependence amongst steroid responsive INS
4. To ascertain the steroid regimens to treat the first presentation of INS
5. To describe the frequency and type of complications associated INS.
6. To describe the one year outcome of children with INS

METHODS

New Zealand paediatricians were asked to report to the New Zealand Paediatric Surveillance Unit, any child with newly diagnosed INS satisfying the case definition between 1st July 2001 and 1st July 2004. The definition for INS was any child between the ages of 3 month and 15 years presenting with oedema, hypo-albuminaemia with normal renal function and in the absence of a systemic illness causing nephrotic syndrome. Infants with congenital INS and those with INS presenting after age 15 years were excluded. An initial questionnaire was sent to reporting paediatricians requesting demographic and clinical information. A follow up questionnaire was sent to the patient's primary paediatrician 12 months later requesting information on response to steroid treatment, number of relapses, vaccination status, biopsy result and complications of INS. Ethnicity of the index case was determined from the reported ethnicity of the parents on the hospital admission data. Three children had parents from 2 differing ethnic groups, for example Indian mother and a Maori father. The study was approved by the Auckland Research Ethics Committee. The ethnic group Asian included peoples from all Asian countries and those from the Indian subcontinent The 2001 Statistics New Zealand Population Census was used for population based comparisons.

STASTICAL ANALYSIS
Software Graphpad Instat 3 and Graphpad Prism 4, Graphpad Software Inc San Diego were used. Unpaired t test and chi square test were used to analyse for statistical significance.

Definitions of steroid response and relapse
Remission – albustix showing 0 to trace protein for 3 consecutive days
Relapse – albustix showing 3+ or more on 3 consecutive days
Infrequent relapser – 1 relapse in 6 months or less than 4 per year
Frequent relaper – 2 or more relapses per 6 months or more than 4 per year
Steroid dependence – 2 consecutive relapses during reducing or alternate day steroids or within 14 days of cessation of steroids
Steroid resistance – no response to steroids after 4-6 weeks of daily high dose therapy

Over the 3 year initial reporting period, 95.8% of the questionnaires were returned to the surveillance unit. Duplicate reported cases were excluded, and cases that were initially diagnosed overseas. The dataset was validated with hospital discharges for nephrotic syndrome in children under age 15 years for July 2001- July 2004 collected by the New Zealand Health Information System

RESULTS
Fifty-one children were reported to have INS; of which 49 were diagnosed in New Zealand with two children diagnosed overseas and were thus excluded from further study. The annual prevalence of INS was 16.3 per 100,000 (11.1-21.5, 95% CI) children under age 15 years. The incidence was 1.9 per 100,000.
The demographics and clinical features shown in Table 4:1 and 4:2 respectively.

TABLE 4:1 **Demographic features at presentation**

Gender *	**Males 35, females 14**
Mean/median age (SD)	6.1/4.9(3.8)
Age distribution(%)	
<12months	0
1-5years	26(53)
6-10	14(29)
>11years	9(18)

Comparison of age at presentation between male and females (p= 0.2, unpaired t test)

TABLE 4:2 **Clinical features at presentation n(%)**

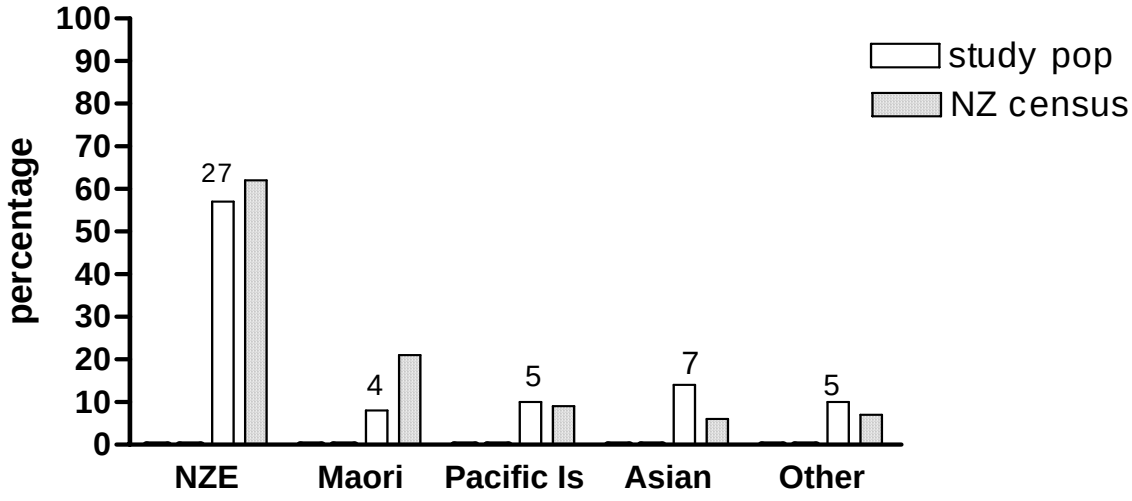
Micro-haematuria	35(71)
Gross haematuria	1*
Elevated blood pressure(>95th centile)	15(31)
Normal renal function	49(100)

- *biopsy showed MCD with weak C1q immunofluorescence*

Ethnicity
Figure 1 shows the ethnic composition of the group.

There was no significant difference in the proportion of ethnic groups represented in the study compared with the general population of New Zealand under age 15 years.(chi square p=0.3). Steroid resistant NS was not more common in Maori and Pacific Island ethnic groups than Caucasians (6 of 9 steroid patients were European).

Fig 1. Ethnicity of study population compared with New Zealand population <15years age



NZE NZ European 592,053, Maori 196,480, Pacific Is 90,144, Asian 56,280, Other 7,434

Number on each bar represent number of patients in that ethnic group. Chi square test comparing study group and general New Zealand population <15years, p=0.297

Initial steroid treatment and subsequent response
All 49 patients were given an empirical trial of prednisone. Steroid responsiveness was observed in 80.4% of the group, with a mean time to response of 12.1 days (8.9-15.4 95% CI), median of 8.4 days, range 3-54 days. Mean and median time to relapse was 15.1 weeks (10.1-19.8 95%CI) and 15 weeks respectively. The initial dosing prednisone was variable, ranging from 1.1-2mg/kg/day with 70% of patients receiving 2mg/kg/day and 82% receiving their therapy in a single daily dose

A wide variety of steroid regimens were utilised after the initial treatment with 25 different regimens recorded. Duration of steroid therapy following initial remission varied from 1-8 weeks of daily treatment. Nine were treated with 2-8 weeks of daily prednisone with reduction, 15 treated for 1-7 weeks of alternate day prednisone without reduction and 26 treated with 1-16 weeks of alternate day therapy with progressive reduction. The median duration of total steroid treatment was 12 weeks (range 2-19 weeks)

Renal Biopsy
Twenty three of 49 (46%) had biopsies done in the first 12 months. Indications for biopsy were steroid resistance (8), frequent relapsing or steroid dependence (13), and unusual presentation in 2, (gross haematuria and post bone marrow transplant). Of the 8 children biopsied for steroid resistance, 4 had focal segmental glomerulosclerosis (FSGS), 2 minimal change disease (MCD), and 2 diffuse mesangial proliferation (DMP). All patients with frequent relapsing or steroid dependent nephrotic syndrome had MCD.

ADJUNCTIVE THERAPIES
Antibiotic prophylaxis and pneumococcal vaccination
Antibiotic prophylaxis was administered to 33/46 children, with oral pencillin V being prescribed in the majority. Sixty percent of patient were administered the pneumococcal vaccine during the 12 months of follow up.

Diuretics and albumin infusions
Twenty six percent (13) patients received diuretics as part of their initial therapy with most (78%) also receiving infusions of concentrated albumin for hypovolemia or severe oedema. The reason for albumin administration was not requested in the initial questionnaire. There was no obvious difference between those who were given diuretics with or without albumin in terms of the frequency of hypertension or age at presentation.

Complications of nephrotic syndrome
The complications of nephrotic syndrome and prophylaxis of thrombo-embolism are shown in table 4:3.

TABLE 4:3 Complications of childhood nephrotic syndrome

Invasive sepsis	**n**	timing of complication
Pneumococcal peritonitis	**1**	at diagnosis
Pneumococcal bacteremia	**1**	Pneumococcal bacteremia

Non invasive		
Stahylococcal skin abscess	1	8months after

All episodes of sepsis occurred whilst the patients were nephrotic. No patients were recorded as having invasive sepsis whilst on antibiotic prophylaxis. There was no relationship between the use of aspirin and the need for diuretic therapy or albumin infusions (4 of the aspirin group had diuretics and or albumin and 4 did not). One patient presented with a sinus venous thrombosis of the central cerebral sagittal vein and was subsequently diagnosed to have steroid sensitive nephrotic syndrome.

Status of patients at 12 months

Of the original 49 patients, 46 were available for follow up at 12 months and their outcome is shown in Table 4.4

TABLE 4:4 Outcome of children at 12 months follow up (n=46)

Clinical Category	**n(%)**
Steroid responsive	37(80.4)
Steroid dependent	11
Frequent relapsing	10
Infrequent relapsing	7
Nil relapse	6
Unclassified	3
Steroid resistant biopsies	9 (19.6)
FSGS	4
MCD	2
DMP	2
Not biopsied	1
Thrombotic complication and prophylaxis	
Aspirin therapy	8
Venous thrombosis(central)	1 at 1st presentation

FSGS focal segmental glomerulosclerosis, MCD minimal change disease, DMP diffuse mesangial proliferation

Three were either lost to follow up or had left the country. All were alive at the 12 months follow up questionnaire but those with steroid resistance (n=9) had varying degrees of impaired health. One patient with FSGS progressed to end stage renal failure and had commenced peritoneal dialysis, 4 were hypertensive because of FSGS or steroid dependent nephrotic syndrome. Eight patients were being treated with alternative immunological therapy, (cyclosporin A, levamisole, cyclophosphamide) because of steroid resistance or dependence. Recent follow up of those with FSGS showed they were developing renal insufficiency and the 2 patients with DMP remain persistently and severely nephrotic after failing treatment with cyclosporin.

DISCUSSION

This is the first prospective study of childhood nephrotic syndrome in New Zealand. New Zealand has a population of 4.1 million people, with a unique demography including a large number of migrants from the Pacific Islands and more recently an influx from China and South-east Asia. A previous study from New Zealand had suggested that glomerular diseases were over represented in adults whose ethnic groups originated from the Pacific Islands[7]. That study showed that Maori and Pacific Island adults had a higher incidence of nephrotic syndrome, however, Simpson[5] in a 10 year retrospective study of renal biopsies for childhood nephrotic syndrome, was unable to confirm this pattern for the paediatric age group. The present population-based study was also unable confirm the results of the earlier adult study. This may be due to small patient numbers in each ethnic group. The incidence of INS in New Zealand children, 1.9 per 100,000 children under age 15 years, is very similar to international published studies[1,2,3]. Studies also indicate there is a higher incidence in low socio-economic groups and non Caucasian ethnic groups[1], however this relationship could not be investigated in this current study as in 30% of the patients, information on the occupational status of either parent was not available. The also author acknowledges the difficulties of eliciting assignment of ethnicity. The small numbers in each ethnic group limits the statistical power to detect significant differences in the study population.

A relatively high proportion (46%) of the group had a renal biopsy. It has been standard practice in our unit to perform a biopsy in patients with frequently relapsing or steroid dependent NS prior to commencing alternative immunosuppressive therapy such as cyclophosphamide or cyclosporin A. This biopsy protocol remains our current practice in spite a number of studies indicating that steroid responsiveness was more accurate in predicting outcome than exact histological diagnosis.[8,9,10] Our unit believes that a histological diagnosis assists the nephrologist in providing the most appropriate treatment and guide to prognosis. Nine of 46 patients were steroid resistant which is lower than the 66% in the earlier biopsy based report by Simpson[5]. Analysis of the renal biopsy group showed that 74% were minimal change and 17% were FSGS. The proportion of patients with MCD is comparable to that reported in the ISKDC[11] study, however, a higher incidence of FSGS (17%) is observed in the present study. This figure is identical to that observed in the renal biopsy study by Simpson. Earlier series

in the 1960s and 1970s showed that FSGS composed of 7-10% of children with nephrotic syndrome. More recent series show 20-60% of patients with this pathological lesion with a higher frequency in African American compared with Caucasians.[12,13,14]

Sixty six percent of our steroid responsive group had frequent relapses or steroid dependence with a mean of 5.3 relapses per year. This rate of relapse is consistent with published data[15]. Although the median duration of total steroids was 12 weeks, many patients were given relatively short courses of prednisone. Recent reviews and meta-analyses of published controlled clinical have recommended that initial presentation of steroid sensitive nephrotic syndrome be treated with prolonged course of prednisone of between 3 to 7 months duration. This would result in fewer relapses without a significant increase in steroid toxicity.[16,17] One review suggested that increased dose of steroids as well as prolonged duration was important in reducing the risk of relapse[17]. Our current study provides an opportunity for the education of paediatricians in the optimal steroid treatment of INS based on current evidence based guidelines.

Serious infection complications arising from NS were relatively uncommon, with only 3 episodes of invasive pneumococcal sepsis. All cases occurred at the time of diagnosis and not were preventable. A recent review by McIntyre[18] highlighted that there were no controlled trials on the use of penicilin prophylaxis or pneumococcal vaccination in childhood NS.

One quarter of our patients were given frusemide and 20% salt poor albumin during their initial presentation. Published evidence based recommendations for albumin infusions remain are lacking. The author's policy has been to restrict the use of albumin and diuretics to patients with significant abdominal pain secondary to intra-vascular volume depletion and severe genital oedema. It was unclear from the results of the survey how many of the patients receiving albumin had significant volume depletion or severe oedema as most of the patients were under the care of general paediatricians in other centres. The dangers of albumin infusions have been reported in a number of studies. Haws[19] showed that albumin infusions caused hypertension in 46% of the treatment courses and addition hypokalemia, hypernatremia was observed in 40% and 17% of treatment courses respectively. Yoshimura et[20] al indicated that albumin administration might delay the response to steroid therapy and induce more frequent relapses after remissions. The present study was unable to confirm a link between albumin infusions and relapse frequency with only 10/29 relapsers having had albumin compared with 19/29 who did not have albumin infusions. Although the study did not seek specific details of complications of albumin therapy, none were reported to the investigator. The author recommends 0.5-1.0gm/kg of 20% albumin solution given over 2-3 hours followed by 1mg/kg of intravenous frusemide. We have observed occasional episodes of transient hypertension which were easily managed with antihypertensive medication.

In conclusion, the present study confirms the incidence of paediatric INS is similar to that in other countries but could not confirm that non Caucasian

races had a higher incidence of nephrotic syndrome. There was a wide variety of steroid treatment regimens indicating possible confusion to the most satisfactory regimen based on current evidenced based guidelines. Results of this study form a useful basis for developing guidelines on the management of childhood nephrotic syndrome

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5. SUBDURAL HAEMORRHAGE IN CHILDREN UNDER 2 YEARS OF AGE

Dr Patrick Kelly, Dr Bridget Farrant
Starship Children's Hospital

2 year study, completed 2002

INTRODUCTION

Infantile subdural haemorrhage (SDH) can result from non-traumatic causes or from accidental trauma, but many cases are associated with child abuse. Such cases may be described as Non-Accidental Head Injury (NAHI) or Inflicted Traumatic Brain Injury (ITBI).

Our aim was to describe the incidence, demographics and characteristics of infantile SDH in New Zealand. This might assist in guiding further research into the prevention, diagnosis and treatment of these injuries in the New Zealand population.

MATERIALS AND METHODS

SDH in infants under 2 years (whatever the cause) was included on the NZPSU report card from 2000 to 2002. Neurosurgeons were included on the distribution list in 2002. A questionnaire was sent out covering basic demographics, history, medical findings, diagnosis, social history and outcome at one week.

In July 2005, we searched NZHIS data (whether from death certificates or discharge codes) using ICD10 codes. We were able to match individuals from the two datasets, and thus to avoid duplication. Data obtained from the NZHIS was limited, so the data could be merged only for analysis of incidence, age, gender and ethnicity.

We recorded NZPSU cases as abuse if they were diagnosed as such by the notifier, and NZHIS cases as abuse if there was a child maltreatment ICD code. In cases without such a code, we did not know whether child abuse had been considered. We recorded NZHIS cases as not abuse if codes provided an apparently consistent mechanism of injury.

This left a number of infants with traumatic SDH, no diagnosis (or code) of child abuse, yet no history (or code) of significant accidental trauma. To identify cases best regarded as indeterminate, we applied the algorithm proposed in 1992 by Dr Duhaime[i]. Given the limitations of the data to which the algorithm could be applied, we did not use the algorithm to change any diagnosis from accident to abuse.

RESULTS

Table 5.1 shows the number of cases of intracranial haemorrhage identified. Eighty-six cases were identified from NZHIS data alone. Thirty-seven of these had the nonspecific perinatal code P528 and probably did not have SDH. This left 49 cases of SDH not detected by the NZPSU method, only 2 of which were deaths. When duplicates were removed, the NZPSU detected 77 cases, giving an overall total for SDH of 126.

Table 5:1 **Basic demographics: all accidents versus all abuse**

Demographic		Abuse N = 48	Accident N = 28	Odds Ratio
Gender	Male	33 (68.8%)	15 (53.6%)	1.0
	Female	15 (31.3%)	13 (46.4%)	0.5 (0.2, 1.4)
Ethnicity	European	14 (29.2%)	15 (53.6%)	1.00
	Maori	27 (56.3%)	6 (21.4%)	4.8 (1.6, 14.8)
	Pacific	6 (12.5%)	6 (21.4%)	1.1 (0.3, 4.0)
	Other	1 (2.1%)	1 (3.6%)	1.1 (0.1, 11.3)
Age	< 1	35 (72.9%)	19 (67.9%)	1.3 (0.5, 3.5)
	1 - 2	13 (27.1%)	9 (32.1%)	1.00

Table 5:2 **Population-based incidence of non-accidental subdural haemorrhage**

Age	**Ethnicity**	**Rates per 100,000**			
		"Minimum"	95% CI	"Maximum"	95% CI
< 2 years	All	14.7	10.8 – 19.4	19.6	15.1 – 25.0
	Non-Maori	8.6	5.3 – 13.2	13.1	9.0 – 18.5
	Maori	32.5	21.4 – 47.3	38.5	26.3 – 54.4
1 – 2 years	All	7.3	3.8 – 12.9	8.0	4.2 – 13.6
	Non-Maori	4.9	1.8 – 10.7	5.7	2.3 – 11.8
	Maori	14.6	5.3 – 31.8	14.6	5.3 – 31.8
< 1 year	All	22.0	15.4 – 30.4	31.1	23.1 – 40.9
	Non-Maori	12.3	6.9 – 20.3	20.5	13.3 – 30.3
	Maori	50.0	31.0 – 76.4	61.9	40.5 – 90.7

There were 12 cases of SDH from birth trauma, of whom 3 died, and 49 cases of "Other intracranial bleeding" in the neonatal period. None of these died. Most had the non-specific ICD code P528, for conditions such as intra-ventricular haemorrhage. Seven cases of SDH in the neonatal period, not ascribed to birth trauma, were reported to the NZPSU. Six of these had apparent contributing factors. None had left hospital. No cases of SDH from antenatal massage were described.

Outside the neonatal period, there were 24 infants with non-traumatic SDH. None died. Diagnoses included arterio-venous malformation, haemophilia, hydrocephalus, malignancy, meningitis and septicaemia. No case of glutaric aciduria was reported.

There were 28 cases of SDH resulting from accidental trauma, none of whom died. Sixteen of these cases were notified to the NZPSU, but 12 (43%) were not. The NZHIS data on mechanism was limited to E codes. Acknowledging the limitations of E codes, we accepted NZHIS codes for motor vehicle accidents, falls on stairs, falls being carried and falls "one level to another" as plausible mechanisms.

There were 16 cases of SDH resulting from "dubious" accidental trauma (16). One died. Seven of these were notified to the NZPSU, but 9 (56%) were not. Of these 9 cases, 7 had SDH and skull fractures, and 2 had SDH plus other serious injury, with E codes for trivial or unspecified mechanisms. Among the 7 NZPSU cases, there were 3 that, using Duhaime's criteria, were presumptive for abuse, and 4 which were indeterminate. One of these was an 8 month infant who was notified to the NZPSU as "no definite conclusion", and not reported to the statutory authorities. Two months later, he was found dead at home from multiple blows to the head, and returned to the study on a death certificate via NZHIS, this time identified with a child maltreatment code.

There were 48 cases of SDH resulting from abuse, of whom 6 died. Thirty-seven were notified to the NZPSU, but 11 (23%) were not. Table 1 compares infants with SDH from abuse and those with SDH from accidents, excluding the indeterminate. The only significant difference is the over-representation of Maori in the abused group. When the NZPSU data is analysed separately, the findings remain essentially the same. Table 2 shows the "minimum" and "maximum" population-based incidence from this data.

All subsequent analysis was perforce restricted to the NZPSU dataset.

In the 37 cases of abuse, there was no history of injury at all in 14 cases. In 15 cases, a fall < 1m was reported (in two of these, a caregiver later confessed). In 3 cases, it was reported that the infant had fallen 1- 3 m. There were no reports of falls > 3 m. There was one other confession. A number of other mechanisms of injury were suggested.

In contrast, there was a history in all 16 accidental cases. There were 3 motor vehicle accidents, 3 falls > 3m and 6 falls 1-3 m. There were 4 falls < 1m: one with momentum, one from a sibling's arms striking the occiput on a wooden floor and presenting immediately, one fall from a mattress onto a concrete fire-hearth, and one witnessed fall onto a concrete floor. Two of these infants had bruising to the head at site of impact. None of the 4 had injuries outside the head, and all were apparently neurologically normal at 7 days after admission.

All 16 accidental cases, and 35 of the 37 cases of abuse, had a CT head. One case of abuse was dead on arrival, so did not. One case of abuse had no CT because the SDH was discovered on outpatient MRI performed for 6th nerve palsy. In all, 26/37 (70.3%) abusive cases and 5/16 (31.3%) accidental cases had brain MRI. A neurosurgical procedure to evacuate subdural blood was perfomed in 3 (18.8%) accidental cases, and in 9 (24.3%) cases of abuse. Injuries to the head were the only injuries found in 14 (87.5%) of the accidental cases, and in 24 (64.9%) of the cases of abuse.

External bruising away from the head was noted in 1 of 14 (7%) of accidental cases, and in 7 (19.4%) cases of abuse. A skeletal survey was performed in 5 (31.3%) of accidental cases, and in 33 (89.2%) of cases of abuse. Bone scan was performed in no accidental cases, but in 5 (13.5%) of cases of abuse. Fractures outside the head were seen only in cases of abuse, in 8 infants (21.6%). Rib fractures were found in 2 infants (5.4%), both of whom also had metaphyseal fractures, and one of whom had subperiosteal bleeding of one tibia. Metaphyseal fractures (without other skeletal fractures) were found in 2 other infants, and isolated long bone fractures in 4 others.

Data on family demographics was poor, and is therefore not presented. Whether the family was known to the Department of Child Youth and Family Services is documented in 28 cases of abuse (24 were not), and in 7 cases of accidental injury (5 were not).

In 23 cases of abusive injury (62%), the identification of the perpetrator was unknown at the time of notification. In 8 cases the perpetrator was thought to be the father (in 3 cases, because he confessed), in 3 cases the mother (including 1 confession), and on 3 occasions an unrelated caregiver.

Neurological status at one week was normal in 12 of 16 accidental cases (75%), but in only 12 of 37 cases of abuse (32%). There were no deaths in the accidental group, but the mortality in the abusive group was 5/37 (13.5%)

DISCUSSION

International population-based studies of NAHI show relatively consistent results. The rate in South Wales and South-West England was between 10.1 (5.3 – 19.2) and 12.45 (6.4 – 24.1) per 100,000 infants under 2 years[ii]. A more recent study

through the British PSU for the United Kingdom obtained an overall rate of "SDH / effusion" of 12.5 (10.3 – 14.6) under 2 years. The rate was 24.1 (20.9 – 28.2) in infants under 1 year, and 1.3 in infants between 1 and 2 years. However, the rate of NAHI was lower: 7.1 (6.4 – 9.3) under 2 years, and 14.2 (12.1 – 17.8) in infants less than 1[iii]. In Scotland the rate was 24.6 (95% CI, 14.9 – 38.5) cases per 100,000 under 1 year[iv]. In North Carolina the rate was 17.0 (95% CI, 13.3 – 20.7) per 100,000 under 2 years. The rate was 29.7 (22.9 – 36.7) in infants 1 year or less, and 3.8 (1.3 – 6.4) in infants 1 - 2 years old[v].

There is no single completely reliable diagnostic test for abusive head injury. Not all forms of abusive head injury result in SDH, and the presence of SDH in an infant (while a matter of grave concern) does not by itself necessarily prove that the infant was abused. Responsible practice in child protection with regard to head injury will always yield cases where we can be sure that the injury was accidental, cases where we can be sure it was abusive, and cases where we just don't know. The use of "minimum" and "maximum" figures in our analysis reflects the reality of clinical practise. Even if one takes only the "minimum" figures obtained in this study, our incidence of SBS is similar to overseas data. In the Maori population, our incidence is arguably the highest in the world. The Maori rates are extremely concerning, particularly as the same phenomenon was seen in an earlier, retrospective, study[vi]. The present study, unfortunately, provides no more information as to why this should be so, and further research is urgently needed.

The use of "minimum" and "maximum" figures was also forced upon us by the surprisingly large number of cases not reported through the NZPSU. Other NZPSU studies have not encountered this problem, which is open to a variety of interpretations. Some paediatricians may have been too busy to complete the questionnaire. Paediatricians may have assumed that the study was only interested in traumatic SDH where NAHI was in the differential. Accidental or equivocal cases may have come through trauma services and not been referred to a Paediatrician. This last possibility is the most worrying, as this group comprised 20% of all traumatic infantile SDH, probably including cases of unrecognized child abuse. It is possible that inexperienced clinicians may have been falsely reassured by the absence of injuries to other parts of the body. However, as this study shows, abusive injury (like accidental injury) may be confined to the head alone. Apart from corroborated major trauma, it is our opinion that all cases of traumatic head injury in an infant should be reviewed by a Paediatrician with expertise in child protection.

Our data on the mortality and morbidity of NAHI is limited, but consistent with international literature showing a high mortality, and a high risk of long-term disability resulting in a very significant cumulative burden on the community.

It was disappointing, but not unexpected, that the demographic information available through the NZPSU was so poor. Again, this replicates our earlier study

on the limited content of medical records in these cases. A strong case can be made that all infants admitted with traumatic SDH (whatever the cause) become part of an ongoing clinical research project, which would collect not only complete demographic information and data about the original injury, but ongoing neurodevelopmental data.

The burden of death or permanent brain damage is an extremely high price for infants, their families and society to pay for the effects of a brief episode of uncontrolled rage. It would be far better to try and prevent these injuries. One paper suggests that focused education of both parents in the neonatal period may be effective, for an estimated cost of $US10.00 per infant[vii]. This evidence is derived from a dedicated continuing program of nurse-led parent education, not a one-hit media campaign. It is time for New Zealand to consider such an intervention, which should be prospectively evaluated.

ACKNOWLEDGEMENTS

We gratefully acknowledge the support of the staff of the NZPSU (Dr Nigel Dickson, Melissa Carter and Professor Barry Taylor), without whom this study would not have been possible. We thank all the Paediatricians and Neurosurgeons who notified cases and filled in questionnaires, for adding yet another task to their busy schedules. We thank Chris Lewis of NZHIS for his help, Sue Guthrie from the Clinical Records Department of the Auckland District Health Board for her assistance with coding issues, and Dr John Thompson of the Department of Paediatrics, University of Auckland Faculty of Medicine and Health Sciences, for his advice on statistical analysis.

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Conditions Ever Monitored by NZPSU

Table 6: **All conditions ever monitored by the NZPSU**

Condition	Abb.	Commenced	Concluded
Acute flaccid paralysis	AFP	October 1997	Ongoing
Haemolytic nephritic syndrome	HUS	January 1998	Ongoing
Congenital rubella syndrome	CRS	January 1998	Ongoing
Perinatal HIV exposure	HIV	January 1998	Ongoing
Vitamin K deficiency bleeding	Vit K	January 1998	Ongoing
Neonatal herpes simplex infection	HSV	January 1998	December 2000
Subdural haemorrhage (<2 years)	SDH	January 1999	December 2002
Retinopathy of prematurity (stage III)	ROP	January 1999	December 2000
Diabetes mellitus	DM	January 1999	December 2000
Fetal alcohol syndrome	FAS	July 1999	December 2001
Kawasaki disease	KD	January 2001	December 2002
Bronchiectasis	BE	January 2001	December 2002
Idiopathic Nephritic syndrome	INS	July 2001	July 2003
Inflammatory bowel disease	IBD	January 2002	December 2003
Prolonged Infantile Cholestasis	PIC	January 2004	December 2005
Foregut and Hindgut Malformations	FHM	January 2004	December 2005
Pertussis	Pert	July 2004	July 2005
Inborn Errors of Metabolism	IEM	January 2004	Ongoing
Pneumococcal Meningitis	Pneu Meng	April 2005	Ongoing

International Network of Paediatric Surveillance Units (INoPSU)

Establishment of INoPSU

The network was formed in August 1998 at a meeting of 10 Paediatric Surveillance Units expressing a desire to link with each other. This took place at the 22nd International Congress of Paediatrics in Amsterdam, The Netherlands. The first INoPSU conference was held in 2000 in Canada and was attended by representatives of the existing units. Subsequent meetings have been held in York, England in 2002 and Lisbon, Portugal in 2004. Dr Nigel Dickson has attended these recent meetings.

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

- facilitating communication and cooperation between existing national paediatric surveillance units;
- to assist in the development of new units;
- to facilitate sharing information and collaboration between researchers from different nations and scientific disciplines;
- to share information on current,past and anticipated studies and their protocols, and on conditions that have been nominated for surveillance but are not selected;
- 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 school 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 co-operative surveys 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.

Members of INoPSU

Founding members:

- Australian Paediatric Surveillance Unit (APSU)
- British Paediatric Surveillance Unit (BPSU)
- Canadian Paediatric Surveillance Programme (CPSP)
- German Paediatric Surveillance Unit (ESPED)
- Latvian Paediatric Surveillance Unit (LPSU)
- Malaysian Paediatric Surveillance Unit (MPSU)
- Netherlands Paediatric Surveillance Unit (NSCK)
- New Zealand Paediatric Surveillance Programme (NZPSU)
- Papua-New Guinea Paediatric Surveillance Unit (PNGSU)
- Swiss Paediatric Surveillance Unit (SPSU)

Additional Members

Welsh Paediatric Surveillance Unit (2000)
Portuguese Paediatric Surveillance Unit (2001)
Irish Paediatric Surveillance Unit (2001)
Greece and Cyprus Paediatric Surveillance Unit (2004)

Associate Members

Trinidad and Tobago Paediatric Surveillance Unit (2004)
British Ophthalmological Surveillance Unit

Administration of the Association

In order to carry out the aims and direct the activities of INoPSU a secretariat has been set up. From 2004 Professor Rudi von Kries (ESPED) has acted as convenor, Dr R Pereira (NSCK) has acted as deputy convenor and Richard Lynn (BPSU) has acted as communications liaison.

International Collaboration

New Zealand paediatricians who are interested in undertaking international studies, or compare the rates of uncommon disease between countries, are encouraged to consider using INoPSU for this purpose. Please contact Nigel Dickson for further information.

Table 7: **Members of INoPSU** INoPSU Website: www.inopsu.com

Country	Unit	Email	Website
Australia	APSU	apsu@chw.edu.au	http://apsu.inopsu.com
Britain	BPSU	bsu@rcpch.ac.uk	http://bpsu.inopsu.com
Canada	CPSP	cpsp@cps.ca	www.cps.ca/english/cpsp
Germany	ESPED	Prof.von.kries@gmx.de	www.esped.uni-duesseldorf.de
Ireland	IPSU	gilld@iol.ie	
Latvia	LPSU	aspedlat@com.latnet.lv	
Malaysia	MPSU	jho@pc.jaring.my	
Netherlands	NSCK	r.pereira@pg.tno.nl	www.nvk.pedianef.nl
New Zealand	NZPSU	nzpsu@otago.ac.nz	www.paediatrics.org.nz
Papua New Guinea	PNGPSU	hopepng@datec.com.pg	www.hopeww.org/where/png/png5.htm
Portugal	PPSU	ana.moreira@sb.com	www.bag.admin.ch/infekt/melde/spsu/dindex.htm
Switzerland	SPSU	hans-peter.zimmermann@bag.admin.ch	
Wales	WPSU	John.Morgan@eglam-tr.wales.nhs.uk	
Trinidad and Tobago	T &TPSU		
Greece and Cyprus			n.persianis@cytanet.com.ay

Table 8: **Characteristics of the Paediatric Surveillance Units**

Country	Population ($x10^6$<15years)	Established	Approx. no respondents
Australia	3.9	1992	1000
Britain/Eire	12.8	1986	2200
Canada	7.5	1996	2400
Germany	12.0	1992	460*
Greece and Cyprus	1.6	2001	
Ireland	1.3	1996	150
Latvia	0.4	1996	22
Malaysia	7.6	1994	400
Netherlands	3.0	1992	640
Papua New Guinea	1.9	1996	40
Portugal	1.6	2000	300
New Zealand	0.83	1997	200
Switzerland	1.3	1995	250
Trinidad & Tobago	0.5	2005	
Wales	0.65	1994	135*

* Heads of Paediatric Centres

List of Clinicians with 100% Return Rate 2005 (& 2004)

Clinicians who had a 100% return rate in both 2004 and 2005 are underlined

Thank you to those clinicians who returned all of their cards in 2005!

Aftimos	Salim	Hassall	Ian	Rowley	Simon
Aho	George	Hofman	Paul	Richardson	Vaughan
Aiken	Richard	Hoare	Simon	Reith	David
Asher	Innes	Heron	Peter	Rudge	Susan
Baker	Nicholas	Hornung	Tim	Russell	Glynn
Barry	John	Hunter	Warwick	Selby	Robyn
Bates	Giles	Jackson	Pam	Shaw	Ian
Battin	Malcolm	Jacquemard	Raimond	Shaw	Robyn
Bhatia	Sat	Jankowitz	Peter	Sinclair	Jan
Bourchier	David	Jefferies	Craig	Skeen	Jane
Bowkett	Brendon	Kelly	Andrew	Skinner	Jon
Bradley	Stephen	Lees	Hugh	Shillito	Paul
Broadbent	Roland	Leversha	Alison	Stanley	Thorsten
Broomfield	Frank	Liang	Allen	Steinmann	Kai
Brown	Jeff	Longchamp	Daniele	Stonehouse	Mary
Buchanan	Leo	Lourens	Ralph	Taylor	Barry
Buckley	David	McArthur	John	Teague	Lochie
Byrnes	Cass	McFarlene	Scott	Tomlinson	Paul
Calder	Louise	McIlroy	Peter	Tuck	Roger
Campanella	Silvana	Maikoo	Rajesh	Vogel	Alison
Caseley	Terry	Marshall	Andrew	Walker	Wendy
Clarkson	John	Manikkam	Noel	Watkins	Nicholas
Corban	Jenny	Maxwell	Fraser	Webb	Alan
Coulter	Belinda	Marks	Rosemary	Webster	Diane
Dalton	Marguerite	Meates-Dennis	Maud	Wills	Russell
Daniel	Alison	Meyer	Michael	Wilson	Callum

Darlow	Brian	Mildenhall	Lindsay	Wilson	Nigel
Denny	Simon	Mitic	Schumann	Wiltshire	Esko
Doran	John	Mitchell	Anne	Wilson	Ross
Drake	Ross	Mitchell	Ed	Wong	Maisie
Edwards	Liz	Morrison	Philip	Wong	William
Elder	Dawn	Mullane	Michelle		
Farrell	Alan	Nagel	Fred		
Ferguson	Stuart	Newman	David		
Ford	Rodney	Nicholson	Ross		
Forster	Richard	Nicolls	Wayne		
Gavin	Raewyn	Nutthall	Gabrielle		
Gapes	Stephanie	Moore	Philip		
Gentles	Tom	O'Donnell	Clare		
Goldsmith	John	Palmer	Penny		
Grant	Cameron	Parsons	Alan		
Grimwood	Keith	Pattemore	Philip		
Hall	Kate	Pereira	Nicola		
Hassall	Ian	Pinnock	Ralph		
Harris	Mark	Pringle	Kevin		
Gunn	Alistair	Robertson	Steven		
Hewson	Michael	Radcliffe	Marlon		
Harding	Jane	Ramadas	Ram		

Congratulations to Jane Skeen who was selected to win a $50 book token to be presented at the ASM of the Paediatric Society of New Zealand.