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Preface

Welcome to the 2008 Annual Report of the New Zealand Paediatric Surveillance Unit (NZPSU).

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.

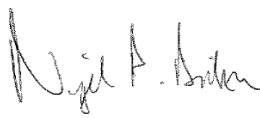
We would like to acknowledge the ongoing funding from the Ministry of Health.

New Zealand Paediatric Surveillance Unit
c/- Department of Women's & Children's Health
Dunedin School of Medicine
PO Box 913, Dunedin 9054

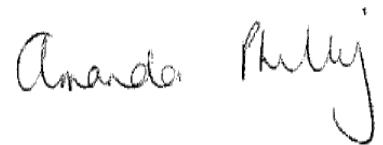
phone: +64 3 474 7825
e-mail: nzpsu@otago.ac.nz
website: www.otago.ac.nz/nzpsu



Barry Taylor



Nigel Dickson



Amanda Phillips



Introduction

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 in 2008 includes eight high-impact childhood conditions.

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

Key Events in 2008

- NZPSU undertook a brief survey of renal stones/unexplained renal failure in infants under 1 year of age to see if this occurred in New Zealand as the result of melamine. No evidence for this was found. A full report is on page 23.
- NZPSU developed it's own webpage. This can be found at: www.otago.ac.nz/nzpsu It contains all current and past questionnaires, protocols, annual reports and publications.

NZPSU liaised with the Canadian Paediatric Surveillance Unit over a similar Study conducted in Canada.

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 in most cases 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) considers the applications 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 2008 (SRP)

Member	Institution
Professor Barry Taylor	NZPSU, University of Otago, Dunedin
Associate Professor Nigel Dickson	NZPSU University of Otago, Dunedin
Dr Pat Tuohy	Ministry of Health*
Professor Elizabeth Elliot	Australian Paediatric Surveillance Unit
Dr Jeff Brown	Palmerston North Hospital
Professor Brian Darlow	University of Otago, Christchurch
Professor Diana Lennon	University of Auckland

** Replaced Dr Alison Roberts who was involved in the SRP since its inception in 1997.*

Surveillance Activities in 2008

In 2008, 210 clinicians participated in the system. The average response rate to the monthly report card/email was 95%. We are very pleased with the ongoing high response rate from the whole of the country.

Minimising the extra workload that the system imposes on paediatricians is a key factor for its success.

Table 2 shows the percentage of clinicians on the mailing list that

reported cases during 2007 and 2008. The table shows that in 2008, 155 of the participants did not report any cases, with two reporting five or more, the same as in 2007.

In 2008 the NZPSU monitored nine uncommon childhood conditions (*Table 3*). Some of the protocols and questionnaires used were adapted from those used by the Australian Paediatric Surveillance Unit.

Table 2: Respondents' Workload 2007 & 2008

Notifications	2007		2008	
	No.	%	No.	%
None	146	71.2	155	73.8
One	44	21.5	21	10
2-4	13	6.4	32	15.2
5 or more	2	0.9	2	0.9

Table 3: Conditions Under Surveillance in 2008

Condition	Surveillance Started	Surveillance Ended	Principal Investigators
Acute flaccid paralysis	October 1997	Ongoing	Dr Nigel Dickson
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	Ended December 2008	Professor Brian Darlow
Inborn errors of metabolism	January 2004	Ending December 2009	Dr Nikki Kerruish Dr Callum Wilson
Pneumococcal meningitis	April 2005	Ended May 2008	Professor Diana Lennon
Adverse Drug Reactions (ADR's)	May 2008	Ongoing	Dr Desiree Kunac
Acute Post Streptococcal Glomerulonephritis	October 07	Ending September 2009	Dr William Wong

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 2008

- There were seven cases notified to the NZPSU in 2008.
- Information has been obtained on all of these children including follow-up information two months after diagnosis.
- All seven were from the North Island.
- One female, six males.
- Age range 21 months to 14 years, median age 11 years.
- No seasonal variation.
- The overall incidence was 0.8 per 100,000 children < 15 years.
- A diagnosis of Guillain-Barré Syndrome (GBS) has been made in five of these cases, cauda equine syndrome in one and transverse myelitis in the remaining case.
- All seven 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 complete for four of the seven children, (57%).

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	4	57
2 stool samples, but one or both not within 14 days of onset of paralysis	0	0
1 stool sample	1	14
No stool samples	2	28

COMMENT

The system did not successfully capture the required rate of AFP in 2008. The rate of stool testing was 57% the WHO criteria is 80%. To try to improve the situation we have changed the name on the card to “Guillain Barre and other causes of acute flaccid paralysis”.

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.

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 2008.

HAEMOLYTIC URAEMIC SYNDROME (HUS)

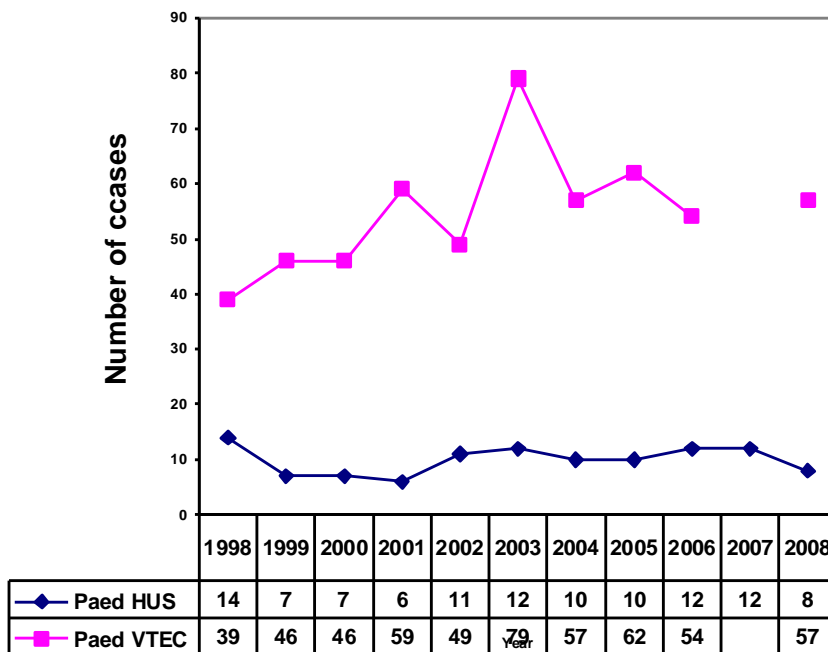
Dr William Wong

Ongoing study started in January 1998

KEY RESULTS FOR 2008

- 8 cases of HUS reported, 7 had a diarrhoeal prodrome (D+) and 1 was atypical (pneumococcal associated)
- Geographic distribution of D(+) HUS - 6 in North Island, 1 South Island
- Median age at presentation of D(+) HUS was 8.3 years, range 1.5 to 9.8 years
- 3/7 of the diarrhoeal group had E coli 0157H7 isolated from their stools
- 6/8 patients needed acute peritoneal dialysis for 5-24 days
- All patients regained renal function to come off dialysis.

Childhood haemolytic uraemic syndrome and VTEC isolates, 1998-2008



PERINATAL EXPOSURE TO HIV

Dr Nigel Dickson, Dr Lesley Voss

Ongoing study started January 1998

In 2008, there were 9 reports to the NZPSU of infants/children born to women infected with HIV. Of these:

- 1 was a perinatally infected child born overseas
- 8 were infants born in New Zealand in 2008 to women with HIV diagnosed prior to giving birth or during their pregnancy.
- Of the 8 infants born in New Zealand in 2008:
 - 5 were born in Auckland, 1 in Wellington, 1 in Christchurch and 1 in Dunedin.
 - 7 were born to mothers whose HIV had been diagnosed before her pregnancy and 1 was diagnosed during her pregnancy.
 - 4 of the mothers were African and 4 were Asian.
 - All of the mothers were given antiretroviral treatment during pregnancy; 2 gave birth by caesarean section and 6 gave birth vaginally; none of the babies were breastfed.
 - None of the children are believed to be infected with HIV

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

Ongoing study commenced January 2004

KEY RESULTS FOR 2008

Disorder	Age at diagnosis	Reason for diagnosis
Medium chain acyl dehydrogenase deficiency (MCAD)	< 1 month	Newborn Screening (NBS)
Isovaleric acidaemia	< 1 month	NBS
Isovaleric acidaemia	19 months	Brother of above Asymptomatic
MCAD	< 1 month	NBS
Benign citrullinemia	< 1 month	NBS
MCAD	< 1 month	NBS
MCAD	62 months*	Fasting hypoglycaemia and lethargy with vomiting illness
Hyperphenylalaninemia	< 1 month	NBS
Galactosaemia	< 1 month	NBS Poor feeding, vomiting, jaundice
MCAD	< 1 month	NBS
Phenylketonuria (PKU)	< 1 month	NBS
Benign citrullinemia	< 1 month	NBS
Hyperphenylalaninemia	< 1 month	NBS
Benign citrullinemia	< 1 month	NBS
3 Methylcrotonyl CoA carboxylase deficiency (3-MCC)	< 1 month	NBS
Very long chain acyl-CoA dehydrogenase deficiency (VLCAD)	< 1 month	NBS
PKU	< 1 month	NBS
Multiple Acyl-CoA Dehydrogenase Deficiency (MADD)	< 1 month	NBS
MCAD	< 1 month	NBS
Non-ketotic hyperglycinaemia	< 1 month	Neonatal encephalopathy Died age 5 days

* Born before expanded newborn screening introduced,

SERIOUS PAEDIATRIC ADVERSE DRUG REACTIONS (ADRs)

Dr Desiree Kunac, Dr Michael Tatley, A/Professor David Reith, Professor Keith Grimwood

Two year study, commenced August 2008.

KEY RESULTS FOR 2008

There were 12 notifications made to the NZPSU during 2008.

One notification was made in error and for 2 notifications, no further details were received.

For the remaining 9 cases, report summaries are provided below:

Suspect medicine(s)	Adverse drug reaction	Age	Sex	Seriousness / Outcome	Medical Warning
Fluoxetine	Facial tics	10 years	Male	Recovered	
Meningococcal B DTaP-IPV- HepB/Hib	Hypotonic Hyporesponsive Episode (HHE)	2 months	Female	Recovered	
Citalopram	Withdrawal syndrome Convulsions Respiratory disorder	1 day	Male	Not recovered at time of report Life threatening	
Triamcinalone	Injection site atrophy	5 years	Male	Not recovered at time of report Persisting disability	
Ibuprofen	Melaena	12 years	Female	recovered hospitalised	✓
Cefuroxime	Urticaria Erythema multiforme	Unknown	Unknown	Recovered	
Infliximab	Localised paraesthesia distal Numbness	15 years	Female	Recovered Hospitalised	
Ceftazadime	Urticaria	9 years	Male	Recovered	✓
Diclofenac	Interstitial nephritis Acute renal failure	15 years	Male	Not recovered at time of report Hospitalised	✓

Four of the 9 cases (which appear shaded in the table) are new reports that were not previously notified to CARM, highlighting the value of this active surveillance system. Importantly, two of these cases resulted in a medical warning being entered for the child in the NZ Health Information Service database, and all four cases are now entered into the CARM database to further enhance our understanding of serious ADRs in children.

ACUTE POST STREPTOCOCCAL GLOMERULONEPHRITIS (APSGN)

Dr William Wong, Dr Jocelyn Neutze, Professor Diana Lennon

Two year study, commenced September 2007

KEY RESULTS FOR 2008

In the first year of the study 65 cases (41 males) were reported. In year 2 of the study, a further 92 cases (63 males) were identified up until June 2009.

DEMOGRAPHIC FEATURE OF THE 2 YEAR GROUP

Mean age was 7.5years, median 6.8 years, range 1.4 -15.8 years.

Ethnic groups: Maori 44 (50.5%), Samoan 21 (24%), European 11/87(12.6%), Tongan 4(4.5%), and others 8 (9.2%).

A family history of renal disease was present in 6 (6.9%) patients.

CLINICAL HISTORY

A history of a sore throat was elicited in 48, (51%), (2 had cervical adenitis only), skin infection in 30 (32%) and both in 10 (10.8%). 62 patients had the time to onset of PSGN recorded, the mean of which was 11.4 (8.9-13.6 days 95%CI), median 9.5 days.

77 patients had information recorded about whether an antibiotic was given for the sore throat or skin infection, 37/77 (48%) were prescribed an antibiotic. Only 1 child had another sibling who also had APSGN at the same time as the index case.

Gross haematuria was present in 85% of patients with 9 patients having microscopic haematuria only. Oedema was observed in 49/92 (51%), oliguria in 43/88 (49%) and anuria in 2 patients. Hypertension was recorded in 67/92 (70%), Eight had encephalopathic features with 1 having seizures at presentation. 19 patients did not have C3 measured and 17 of 92 did not have streptococcal serology tested at presentation, thus rendering their true diagnosis unclear. 46/90 (51%) had significantly raised initial ASOT (>480), 43/90 (48%) antiDNAase B elevated >680. 31 children had both elevation of both ASOT and antiDNAase B titres.

Of the 92 patients, 47 did not have a throat swab recorded as having been done, 15 patients had Group A streptococcal grown, 22 had normal flora, 2 had no growth and 1 patient each grew a staphylococcus species and a Group G streptococcus.

TREATMENT

Of the 92 patients, 79 (85%) were hospitalized for a mean of 5.3 ± 4.3 days. Four patients had severe nephritis and required a renal biopsy to exclude other treatable causes of severe acute glomerulonephritis. 42/92 children were treated with frusemide diuretic because of either hypertension and or volume overload. Almost all children were treated with a course of penicillin following the diagnosis of APSGN.

CONCLUSION

The majority of APSGN cases were Maori, and Samoan children followed by New Zealand Europeans. When all the cases have been collected, it is projected that the final number will be 165-170 cases. Of the 157 cases reported so far, 64(41%) live in Counties Manukau District Health Board.

It is a significant concern that many children following an episode, fail to attend follow up children's outpatient clinics to document that their glomerulonephritis has resolved without residual sequelae.

Final Reports

VITAMIN K DEFICIENCY BLEEDING (VKDB): Final Report

Professor Brian Darlow

Study commenced January 1998 and ended December 2008

BACKGROUND

Although records are incomplete, vitamin K prophylaxis for haemorrhagic disease of the newborn (now called vitamin K deficiency bleeding – VKDB) was probably given by intramuscular injection at birth to most New Zealand infants in the early 1990s.¹ However in 1992, after a British study suggested a possible association between intramuscular vitamin K and an increased incidence of childhood cancer², an expert committee of the Paediatric Society of New Zealand (PSNZ) recommended oral vitamin K as routine prophylaxis in other than high risk situations.³ This recommendation was similar to that in many other countries but partly because the only preparation of vitamin K available in New Zealand, Konakion[®], was not licensed for oral use, the Department of Health continued to recommend intramuscular prophylaxis.⁴

Oral administration is complicated by the need for repeat doses and compliance with this regime in New Zealand was poor.⁵ Even with good compliance there was evidence the oral route might not be as successful at preventing late-onset VKDB.⁶ By 1995 further epidemiological studies had failed to confirm a link between intramuscular vitamin K and childhood cancer.^{7,8} That year, the PSNZ issued a revised statement recommending that all newborns should have vitamin K prophylaxis and that the preferred route of administration was intramuscular.⁹

The majority of New Zealand births are attended by midwives who were sometimes unwilling to give oral Konakion if the parents declined an intramuscular injection. A new preparation, Konakion MM[®], which was designed for oral or intramuscular use, became available in October 1999 and was the only licensed preparation from 2001. A consensus statement on vitamin K prophylaxis from organisations representing paediatricians, midwives, nurses, general practitioners and obstetricians was published in Feb 2001.¹⁰ This again recommended all babies receive vitamin K; intramuscular injection was the preferred route, but repeat 2mg oral doses, at birth, 3-5 days and 4-6 weeks, were recommended for parents who declined the intramuscular dose.

The first year of surveillance of VKDB was 1998. Following the introduction of the new vitamin K preparation in 2001 and continuing uncertainties surrounding acceptance of intramuscular vitamin K and delivery by the alternative route, surveillance was continued until 2008.

METHODS

Prospective surveillance by the New Zealand Paediatric Surveillance Unit (NZPSU) from January 1st 1998 to December 31st 2008. Paediatricians notifying a case of VKDB were sent a standard two page questionnaire.

CASE DEFINITION

Vitamin K Deficiency Bleeding was defined as: “spontaneous bruising or bleeding associated with prolonged clotting time, but not due to an inherited coagulopathy or DIC, in an infant <6 months old”.

Cases were classified as:

Early – bleeding occurring on the first day of life;

Classic – bleeding occurring from the 2nd to the 7th day of life

Late-onset– bleeding occurring from one week to six months of age.

Definite cases had evidence of bleeding plus a raised prothrombin time (PT), or in some cases a raised INR, together with normal fibrinogen level and normal or raised platelet count, and rapid correction of the PT after administration of vitamin K.

Probable cases conformed to type but lacked key information such as a coagulation profile pre-treatment with vitamin K.

Possible cases were atypical and lacked key diagnostic information.

RESULTS

Over the 11 years of the study the response rate to the survey card, including notification that none of the listed conditions had been seen, averaged 94.5% of the mailing list per annum.

There were 35 notifications of VKDB; one questionnaire was not returned (rate of return 97%), four notifications were not valid and there were five double reports, leaving a total of 25 cases. Three cases were classified as **Early**, 10 as **Classic** and 12 as **Late-onset**. For confirmed cases (see below) the male to female ratio was 2:1.

Early cases: Three cases but all classified as possible only.

Classic cases: Ten cases: 8 classified as definite, 2 as probable. (See Table 1)

None of the 8 definite cases received vitamin K; in five cases consent was withheld and in three cases an intramuscular injection was agreed to but omitted in error. Seven infants were exclusively breast-fed and one received no oral feeding. In six cases the initial presentation was gastro-intestinal bleeding and in two was oozing from the umbilicus or heel-prick sites. All infants fully recovered.

One probable case (case 12) had a typical history; consent for vitamin K was withheld, the infant was exclusively breast-fed and there was oozing from the umbilicus on day two, which resolved after intramuscular vitamin K. Because of the rural location of the birth, no coagulation studies were carried out before vitamin K was administered.

In the other probable case (case 13), there was a verbal assertion that intramuscular vitamin K had been given but no written record and this baby had received a small amount of formula

feeds. Coagulation studies and response to intramuscular vitamin K were compatible with a diagnosis of VKDB.

Table 1: Details of cases of Classic VKDB – New Zealand surveillance 1998-2008

Case No. (year)	Gest/Sex	Vit K	Age (d)	Presentation	Feeding	Diagnosis Confirmed*	Outcome
4 (1999)	39 / M	None – error	3	Heel-prick ooze	Solely breast	Yes	Healthy
5 (2002)	42 / M	None - withheld	3	Heel-prick ooze	Solely breast	Yes	Healthy
6 (2002)	32 / M	None - error	2	GIT	Solely breast	Yes	Healthy
7 (2003)	41 / F	None – error	5	GIT† in baby with MAS‡	Not fed	Yes	Healthy
8 (2003)	38 / F	None – error	2	Heel-prick ooze / GIT	Solely breast	Yes	Healthy
9 (2007)	40 / M	None – withheld	2	GIT	Solely breast	Yes	Healthy
10 (2007)	40 / M	None – withheld	3	GIT	Solely breast	Yes	Healthy
11 (2007)	38 / M	None – withheld	7	GIT	Solely breast	Yes	Healthy
12 (2003)	41 / F	None – withheld	2	Umbilicus bleed	Solely breast	Probable case only	Unknown
13 (2006)	35 / F	Unclear	2	GIT	Formula but small amounts	Probable case only	Healthy

* Diagnosis confirmed by appropriate coagulation studies and prompt response to vitamin K

† GIT : gastro-intestinal haemorrhage

‡MAS : Meconium aspiration syndrome

Late-onset cases:

Twelve cases: 9 classified as definite (one atypical), 3 as possible (See Table 2). None of the 8 typical cases received vitamin K; in four cases consent was withheld, in two no vitamin K was given on professional advice, and in two cases the reason for omission was unclear. Seven of these infants were exclusively breast-fed and one predominantly so. In all cases bleeding occurred in the first or second month of life; 4 infants suffered an intracerebral bleed, three had bruising and one bleeding from the umbilicus and nose. Five infants had deranged liver function tests. One infant died, three have developmental delay (in one case probably from an underlying condition) and only three infants had full recovery.

The one atypical case (case 19) was an infant born at term, who received intramuscular vitamin K. The infant was exclusively breast-fed and presented with nose bleeds at 101 days of age. The INR was >10, APPT 107 secs, fibrinogen 4.8 g/L and platelets “normal”. The

bleeding resolved and the coagulation screen normalised after intravenous vitamin K. The liver function was grossly abnormal with a hepatitis picture of unknown aetiology. A further dose of intramuscular vitamin K was required two weeks later.

Table 2: Details of cases of Late-onset VKDB – New Zealand surveillance 1998-2008

Case No. (Year)	Gest/Sex	Vit K	Age (d)	Presentation	Feeding	Liver disease	Diagnosis Confirmed*	Outcome
14 (1998)	34 / M	None – unclear#	37	Bruising / ICH	Solely breast	No	Yes	Healthy
15 (1998)	39 / F	None- unclear#	30	Bruising / VP ooze	Solely breast	Yes\$	Yes	Delayed development
16 (1999)	39 / F	None - withheld	11	GIT / ICH	Mostly breast	Yes	Yes	Died
17 (1999)	38 / F	None – withheld	13	Umbilical / nose bleeds	Solely breast	Yes - mild	Yes	Healthy
18 (2000)	40 / M	None – withheld	14	Umbilical / GIT	Solely breast	Yes	Yes	Healthy
19 (2001)	40 / M	I.M. at birth	101	Nose bleeds	Solely breast	Yes	Yes	Unknown
20 (2002)	40 / M	None – withheld	30	ICH	Solely breast	No	Yes	Delayed development
21 (2002)	40 / M	None – withheld	19	ICH	Solely breast	Yes	Yes	Delayed development
22 (2008)	38 / M	None – withheld	11	Bruising	Solely breast	No	Yes	Healthy
23 (2000)	40 / M	None – withheld	3 rd wk	GIT	Solely breast	Not known	No – possible	Unknown
24 (2004)	32 / F	I.M. at birth	9	ICH	Mostly formula	No	No - possible	Died
25 (2007)	29 / M	I.M. at birth	32	GIT	TPN / breast	No	No - possible	Unknown

GIT : gastrointestinal haemorrhage

ICH : Intracranial Haemorrhage

I.M. intramuscular

VP : venepuncture

Reason for not receiving vitamin K is uncertain

* Diagnosis confirmed by appropriate coagulation studies and prompt response to vitamin K

\$ Peroxisomal disorder

Of the three possible cases, one infant was born at term and two were premature. The term infant (case 23) did not receive vitamin K after consent was withheld, was exclusively breast-fed and had gastrointestinal bleeding in the third week of life. Results of a coagulation screen were not documented but the symptoms reportedly responded to intramuscular vitamin K.

Both preterm infants received intramuscular vitamin K at birth. A 32 week gestation infant (case 24) was predominantly formula fed and suffered an intracranial haemorrhage on day 9. The INR was 2.5, APPT 71 secs, fibrinogen 2.3 g/L falling to 1.7 g/L and platelets 203×10^9 . Intramuscular vitamin K was given but the infant died. And a 29 week gestation infant (case 25) received several days of parenteral nutrition and then fortified breast milk. Rectal bleeding occurred at 32 days of age. Liver function tests were normal. The coagulation tests were marginally abnormal; APTT 51 secs (normal 32-46), prothrombin ratio 1.3 (normal 0.8-1.2), fibrinogen 3.0 g/L and platelets 480×10^9 , with normalisation after 1mg oral vitamin K.

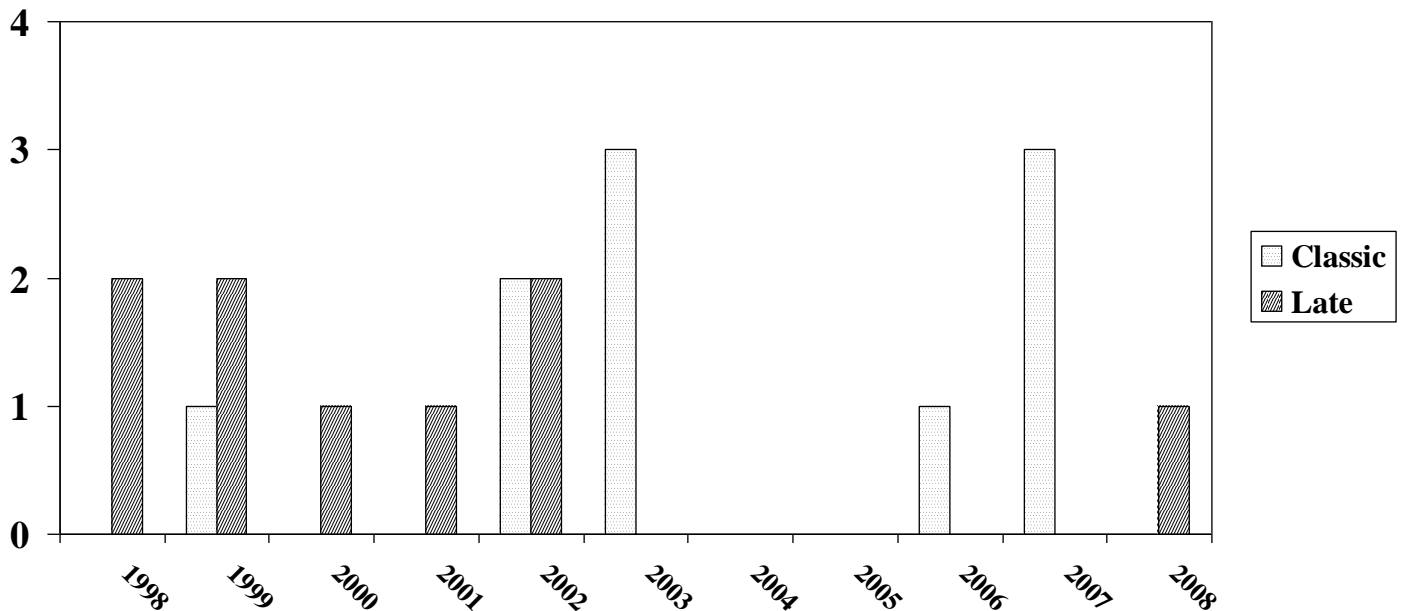


Figure 1: Cases of Classic and late-onset VKDB in New Zealand by year of presentation

All cases definite except two Classic cases classified as probable, one in 2003 and one in 2006.

Discussion

VKDB in New Zealand is virtually confined to fully breast-fed infants not given vitamin K at birth. At least 4 cases (25% of definite cases not given vitamin K), were due to error. One case resulted from mis-communication between professionals where vitamin K was signed as given in anticipation of this by a colleague, and in the other cases there were communication errors with parents. In at least two further cases, parents withheld consent on the advice of a health professional.

The incidence of definite Classic VKDB (8 cases) was 1.25 per 100,000 births (and 1.6 per 100,000 births if the two probable cases are included).

Classic cases were spread over the study years (*Figure 1*). Reports of the incidence of classic VKDB from overseas populations are complicated by differences in the chosen denominator, but recent figures range from 0.08 to 5.4 per 100,000.^{11,12} A meaningful denominator is births where no vitamin K was given, which is unknown for New Zealand. In Christchurch Women's Hospital over the period June 2002 to December 2008 (33,228 births) 95.9 % received intramuscular vitamin K, 2.3% oral vitamin K and 1.8% none, with a consistent pattern across the years. If these figures applied to New Zealand over the 11 years of the study (total New Zealand births varying from 54,000 to 65,000 per annum), the incidence of Classic cases would be 69 per 100,000 births or 1 in 1439 births. This incidence is lower than the historical suggested incidence of 1 in 60 to 1 in 400 breast-fed babies not given vitamin K prophylaxis.

The incidence of definite Late-onset VKDB (9 cases) was 1.4 per 100, 000 births. There was only one definite Late-onset case after 2002 and that had a Classic pattern with delayed presentation. Excluding this case, the incidence from 1998 to 2002 was 2.85 per 100,000 births.

The reported incidence of Late-onset VKDB from developed countries ranges from 0 to 7.2 per 100,000.^{13,14} Konakion MM[®] became available in 1999 and was the only preparation from 2001. Although data are lacking it seems probable that a greater proportion of New Zealand babies were receiving oral vitamin K in the early years of the study.⁵ In other countries Late-onset bleeding has been seen more often with oral regimes but this has not been the case in New Zealand. It is unclear why there have been few cases after 2002 but, as has been suggested following a similar trend in the UK¹⁵, one possibility is greater vigilance and investigation of babies with prolonged jaundice. Two-thirds of Late-onset cases were associated with liver disease.

A recent review¹³ has highlighted the value of tests for Proteins Induced by Vitamin K Absence (PIVKAs), which can point to a diagnosis of VKDB even when performed after administration of vitamin K. This test was not routine in New Zealand hospital laboratories during the study period and often laboratory tests were lacking in rigour, making diagnosis uncertain in a number of cases.

Conclusions

- Cases of VKDB have been virtually confined to fully breast-fed infants who have not received vitamin K prophylaxis.
- New Zealand has had a low incidence of VKDB over an eleven year period, despite much debate about both the route of administration and appropriateness of any prophylaxis in the early years of the study.
- Since 2001 New Zealand has had a clear consensus statement supported by all key professional groups and the Department of Health, and only a single preparation of vitamin K suitable for either intramuscular or oral administration, which has been helpful.

Recommendations

- The 2001 consensus statement should be updated, taking account of these results.
- When a diagnosis of VKDB is entertained, laboratories should be asked to undertake PIVKA tests.
- Further research should be undertaken to ascertain knowledge of prospective parents and of care-givers about the evidence for recommending vitamin K at birth and with respect to reasons for infants not having vitamin K by any route.

Acknowledgements

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REPORT ON STUDY UNDERTAKEN BY NZPSU TO EXPLORE POSSIBLE MELAMINE TOXICITY AMONG NEW ZEALAND INFANTS

Dr Nigel Dickson, Dr Tonya Kara

SUMMARY

New Zealand paediatricians were surveyed using the New Zealand Paediatric Surveillance (NZPSU) network to ascertain whether in the 12 month period to the end of September 2008 they had seen any child under the age of one year with unexplained renal stones or acute renal failure that might have been attributable to melamine intoxication. None were reported, confirming the expectation that this was not a discernible problem in this country.

BACKGROUND

In September 2008, news broke about the contamination of infant milk formula in China with melamine, a substance that when added to milk increases the nitrogen concentration, falsely suggesting an increased protein content.ⁱ Melamine has low oral acute toxicity but excessive exposure in animals causes renal stones.ⁱⁱ In China, this resulted in a small number of infant deaths, and a larger number of cases of renal stones and acute renal failure in young children who had been fed on contaminated milk formula. Subsequently, dozens of countries banned the sale or imports of dairy products from China, the UN issued a worldwide alert, and the European Union banned China-made baby-food.ⁱⁱⁱ There was considerable public interest in the situation in New Zealand fuelled by the fact that the Sanlu, the major Chinese company implicated, was 43% owned by the New Zealand company Fonterra.

Information on the clinical features and diagnostic criteria for melamine associated renal stones and acute renal failure have recently been published by the WHO. The stones are normally radiolucent and have a negative image on urinary tract x-ray. This feature differentiates the stones from those of radiopaque stones of calcium oxalate and calcium phosphate.

Although in Chinese infant milk formula is not imported into New Zealand, in view of the interest and as there are a relatively large number of Chinese families living in New Zealand who might have visited China, the New Zealand Paediatric Surveillance Unit (NZPSU) discussed the situation with the Ministry of Health, and it was decided to undertake a study of possible cases of recent melamine damage among infants in New Zealand.

The specific aim was to determine if there has been any child or children under the age of one year who in the past 12 months had suffered from renal stones and/or acute renal failure that might have been due to melamine damage.

METHOD

The study utilised the network established by the NZPSU to undertake surveillance of acute flaccid paralysis in New Zealand as part of the worldwide strategy to monitor the polio

eradicate programme. This is also used to study a number of other uncommon but important childhood conditions.

On the NZPSU Report Card that was sent out at the beginning of October 2008 paediatricians were asked to report whether or not in the previous 12 months they have cared for any child under the age of one year with “renal stones and/or acute renal failure”. Those that reported affirmatively were sent a short questionnaire, asking for the basis on which the diagnosis was made, the results of standard investigations for stones or ARF, the likely cause, and known exposure to milk and milk products (Appendix 3)

Originally we had planned to ask about “unexplained renal stones and/or acute renal failure”, but opted to remove “unexplained” so as to receive information on all infants with renal stones seen by specialist paediatricians. However the removal of “unexplained” resulted in some comments on the Report Cards from paediatricians who had seen infants with acute renal failure clearly due to another cause such as perinatal asphyxia, congenital urethral valves, cardiac surgery. Hence we communicated through the Paediatric Society list server after the Cards had been sent out that we only required reports of “renal stones and/or unexplained acute renal failure”.

The study was given ethical approval by the Multi-centre Ethics Committee.

RESULTS

Of the 210 Report Cards sent out 194, a response rate of 92%.

Initially there were seven positive reports. Three reports were of the same child with renal stones (Case 1 below); one report was of a child who had presented at age 18 months (hence over our upper age limit of 12 months) with a proteus urinary tract infection with staghorn calculi; one was of a child with congenital dysplastic kidneys who went into acute renal failure; one was of an infant with urethral valves who went into acute renal failure; and the remaining report was of an 11 month old child with renal stones (Case 2 below). We were also informed that some other young children had developed acute renal failure due to perinatal asphyxia and also post operatively, but details of these individual cases were not provided or sought.

Hence, overall there were reports of 2 infants under the age of 12 months with renal stones and none with explained renal failure. Both these infants were male and of European ethnicity. Both of the infants had had a urinary tract infection.

1. One infant with Trisomy 21 was diagnosed at age 4 weeks while on parenteral nutrition post cardiac surgery. He had been born prematurely at 33 weeks, and had been on diuretics since birth for heart failure. He had hypercalcaemia, nephrocalcinosis on ultrasound examination and calcification on abdominal x-ray examination. He also had hypercalciuria which was thought to be secondary to his diuretic therapy and which resolved with a change in diuretic. He had had formula feeds with Karicare only.

2. The other presented at age 11 months with a pyrexial illness and found to have a pyonephrosis, and a “7.5mm stone seen in the left VU junction on US and plain film”. The infant was considered to have “VUJ obstruction with hydronephrosis, renal stasis, infection and stone formation”. The infant was reported “to have had some exposure to milk formula”. A calcium phosphate stone was removed at surgery. While a 24 hour urine collection was not undertaken a random sample suggested a high calcium/creatinine ratio. Serum calcium was normal. This stone was probably due to stasis at the VUJ junction in combination with hypercalciuria, a known complication of VUJ obstruction.

There were 64,540 births in the year to the end of September 2008. With an 86% response, assuming the non-responders are representative of paediatricians throughout the country, the incidence rate was 0/55,000. The upper 95% confidence for this zero incidence is approximately 6.7/100,000 or 1 per 15,000.

DISCUSSION

This study revealed just two infants with diagnosed renal stone nationally in New Zealand in the twelve month period up to the end of September 2008. For both of these infants there was a clear evidence to exclude it being due to melamine. In both cases the stones were radio opaque (melamine-associated stones are radio translucent), and associated with Hypercalciuria. For case 1 the stones were associated with diuretic therapy, and Case 2 with urinary stasis. In this period there was no infant reported with unexplained renal failure. Given this zero incidence the plausible upper limit for the incidence rate of serious renal stones was approximately 1 per 15,000 infants under the age of 1 year.

The strengths of this study is that it actively surveyed all specialist paediatricians using a well recognised process, with a high response rate. It is anticipated that all infants with persistent severe symptoms of renal stones and with acute renal failure would be referred to a paediatrician, and investigations would be undertaken that would reveal the cause of these. The weaknesses of this study is that infants with milder symptoms would probably not be seen by paediatricians, and as the melamine-related stones are radio translucent might not be diagnosed without an USS.

The conclusion is that there is no evidence of serious problem of renal stones or acute renal failure among infants in New Zealand over the past 12 months that could be attributed to melamine toxicity.

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Conditions Ever Monitored by NZPSU

All conditions ever monitored by the NZPSU.

Condition	Commenced	Concluded
Acute flaccid paralysis	October 1997	Ongoing
Haemolytic nephritic syndrome	January 1998	Ongoing
Congenital rubella syndrome	January 1998	Ongoing
Perinatal HIV exposure	January 1998	Ongoing
Vitamin K deficiency bleeding	January 1998	Ongoing
Fetal Alcohol Syndrome	July 1999	December 2001
Subdural haemorrhage (<2 years)	January 1999	December 2002
Retinopathy of prematurity (stage III)	January 1999	December 2000
Diabetes mellitus	January 1999	December 2000
Fetal alcohol syndrome	July 1999	December 2001
Kawasaki disease	January 2001	December 2002
Bronchiectasis	January 2001	December 2002
Idiopathic Nephritic syndrome	July 2001	July 2003
Inflammatory bowel disease	January 2002	December 2003
Prolonged Infantile Cholestasis	January 2004	December 2005
Foregut and Hindgut Malformations	January 2004	December 2005
Pertussis	July 2004	July 2005
Inborn Errors of Metabolism	January 2004	Ongoing
Pneumococcal Meningitis	April 2005	May 2008
Adverse Drug Reactions(ADR's)	May 2008	Ongoing

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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, Lisbon, Portugal in 2004 and Munich Germany 2008. A/P Nigel Dickson has attended the meetings in Canada, England and Portugal.

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 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; and
- 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

Table 6: Members of INoPSUINoPSU Website: www.inopsu.com

Country	Unit	Email	Website
Australia	APSU	apsu@chw.edu.au	www.apsu.org.au
Britain	BPSU	helen.friend@rcpch.ac.uk	www.bpsu.inopsu.com
Canada	CPSP	cpsp@cps.ca	www.cps.ca/cpsp
Germany	ESPED	Prof.von.kries@gmx.de	www.esped.uni-duesseldorf.de
Ireland	IPSU	robert.cunney@malix.hse.ie	
Latvia	LPSU	aspedlat@com.latnet.lv	
Malaysia	MPSU	jho@pc.jaring.my	
Netherlands	NSCK	rob.rodriquespereira@tno.nl	www.nvk.pedianef.nl
New Zealand	NZPSU	nzpsu@otago.ac.nz	www.otago.ac.nz/nzpsu
Papua New Guinea	PNGPSU	hopepng@datec.com.pg	
Portugal	PPSU	uvp-spp@ptnetbiz.pt	www.spp.pf/ingl/index_17.html
Switzerland	SPSU	hans-peter.zimmermann@bag.admin.ch	www.bag.admin.ch/infekt/melde/spsu/d/index/.htm(German)
Wales	WPSU	John.Morgan@eglam-tr.wales.nhs.uk	www.link-wales.org.uk
Trinidad and Tobago	T &TPSU	Prof MIA Omer	
Greece and Cyprus	GCPSU	xhatzi@med.uth.gr	

Table 7: Characteristics of the Paediatric Surveillance Units

Country	Population (x10⁶<15years)	Established	Approx number of respondents
Australia	3.98	1992	1000
Britain/Eire	12.8	1986	2500
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	750
Papua New Guinea	1.92	1996	40
Portugal	1.67	2000	1506
New Zealand	0.83	1997	210
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 2008
Clinicians who had a 100% return rate in both 2007 and 2008 are underlined

Thank you to those clinicians who returned all of their cards in 2008!

<u>Aftimos</u>	<u>Salim</u>	<u>Drake</u>	<u>Ross</u>
<u>Aho</u>	<u>George</u>	<u>Edwards</u>	<u>Liz</u>
<u>Aiken</u>	<u>Richard</u>	<u>Elder</u>	<u>Dawn</u>
<u>Asher</u>	<u>Innes</u>	<u>Evans</u>	<u>Juliana</u>
<u>Baker</u>	<u>Nicholas</u>	Evans	Helen
Barker	David	<u>Farrell</u>	<u>Alan</u>
<u>Bates</u>	<u>Giles</u>	<u>Ford</u>	<u>Rodney</u>
<u>Battin</u>	<u>Malcolm</u>	<u>Forster</u>	<u>Richard</u>
<u>Best</u>	<u>Emma</u>	Gangakhedhar	Arun
<u>Bhatia</u>	<u>Sat</u>	<u>Gavin</u>	<u>Raewyn</u>
Nikki	Blair	Grangaard	Eric
<u>Bourchier</u>	<u>David</u>	<u>Gapes</u>	<u>Stephanie</u>
Bowkett	Brendon	<u>Gentles</u>	<u>Tom</u>
<u>Bradley</u>	<u>Stephen</u>	<u>Goldsmith</u>	<u>John</u>
<u>Broadbent</u>	<u>Roland</u>	<u>Grant</u>	<u>Cameron</u>
<u>Broomfield</u>	<u>Guy</u>	Graham	Dave
<u>Brooks</u>	<u>Jeanine</u>	Grangaard	Erik
<u>Broomfield</u>	<u>Frank</u>	<u>Gunn</u>	<u>Alistair</u>
<u>Brown</u>	<u>Jeff</u>	Hall	Anganette
Brynes	Cass	<u>Hall</u>	<u>Kate</u>
<u>Buchanan</u>	<u>Leo</u>	<u>Hewson</u>	<u>Michael</u>
<u>Buckley</u>	<u>David</u>	<u>Harding</u>	<u>Jane</u>
<u>Campanella</u>	<u>Silvana</u>	<u>Hoare</u>	<u>Simon</u>
<u>Caseley</u>	<u>Terry</u>	<u>Hoare</u>	<u>Simon</u>
<u>Clarkson</u>	<u>John</u>	<u>Hofman</u>	<u>Paul</u>
<u>Cole</u>	<u>Nyree</u>	<u>Heron</u>	<u>Peter</u>
<u>Corban</u>	<u>Jenny</u>	<u>Hornung</u>	<u>Tim</u>
<u>Coulter</u>	<u>Belinda</u>	<u>Hunter</u>	<u>Warwick</u>
<u>Cunningham</u>	<u>Vicky</u>	<u>Hunter</u>	<u>Wendy</u>
<u>Dalton</u>	<u>Marguerite</u>	Hector –Taylor	James
<u>Daniel</u>	<u>Alison</u>	<u>Jackson</u>	<u>Pam</u>
<u>Darlow</u>	<u>Brian</u>	<u>Jankowitz</u>	<u>Peter</u>
<u>De Sylva</u>	<u>Tony</u>	<u>Jellyman</u>	<u>Timothy</u>
<u>Drage</u>	<u>Alan</u>	<u>Kelly</u>	<u>Andrew</u>
<u>Denny</u>	<u>Simon</u>	<u>Doran</u>	<u>John</u>
<u>Dickson</u>	<u>Cameron</u>	<u>Leadbitter</u>	<u>Philip</u>
<u>Dixon</u>	<u>Joanne</u>	<u>Lees</u>	<u>Hugh</u>
<u>Doocey</u>	<u>Clare</u>	<u>Lennon</u>	<u>Diana</u>

<u>Leversha</u>	<u>Alison</u>	<u>Rowley</u>	<u>Simon</u>
<u>Liang</u>	<u>Allen</u>	<u>Sadler</u>	<u>Lynette</u>
<u>Longchamp</u>	<u>Daniele</u>	<u>Sadowsky</u>	<u>Joel</u>
<u>Lourens</u>	<u>Ralph</u>	<u>Sanders</u>	<u>John</u>
<u>McArthur</u>	<u>John</u>	<u>Schmiti-Uli</u>	<u>Meia</u>
<u>Maikoo</u>	<u>Rajesh</u>	<u>Shaw</u>	<u>Robyn</u>
<u>Marks</u>	<u>Rosemary</u>	<u>Shillito</u>	<u>Paul</u>
<u>Manikkam</u>	<u>Noel</u>	<u>Skeen</u>	<u>Jane</u>
<u>Marshall</u>	<u>Andrew</u>	<u>Skinner</u>	<u>Jon</u>
<u>Matas</u>	<u>Richard</u>	<u>Stanley</u>	<u>Thorsten</u>
<u>Maxwell</u>	<u>Fraser</u>	<u>Smith</u>	<u>David</u>
<u>McCarthy</u>	<u>Karen</u>	<u>Smith</u>	<u>Warwick</u>
<u>McCay</u>	<u>Hamish</u>	<u>Spooner</u>	<u>Claire</u>
<u>McFarlene</u>	<u>Scott</u>	<u>Russell</u>	<u>Glynn</u>
<u>McIlroy</u>	<u>Peter</u>	<u>Selby</u>	<u>Robyn</u>
<u>Meates- Dennis</u>	<u>Maude</u>	<u>Shaw</u>	<u>Ian</u>
<u>Meyer</u>	<u>Michael</u>	<u>Steinmann</u>	<u>Kai</u>
<u>Mitchell</u>	<u>Ed</u>	<u>Stonehouse</u>	<u>Mary</u>
<u>Mitic</u>	<u>Schuman</u>	<u>Swan</u>	<u>Catherine</u>
<u>Moore</u>	<u>Philip</u>	<u>Taylor</u>	<u>Barry</u>
<u>Morreau</u>	<u>Johan</u>	<u>Taylor</u>	<u>Paul</u>
<u>Morris</u>	<u>Max</u>	<u>Tomlinson</u>	<u>Paul</u>
<u>Morrison</u>	<u>Philip</u>	<u>Teague</u>	<u>Lochie</u>
<u>Moyes</u>	<u>Chris</u>	<u>Tuck</u>	<u>Roger</u>
<u>Nagel</u>	<u>Fred</u>	<u>Twiss</u>	<u>Jacob</u>
<u>Nair</u>	<u>Arun</u>	<u>Van der Walt</u>	<u>Stephan</u>
<u>Neas</u>	<u>Katherine</u>	<u>Vogel</u>	<u>Alison</u>
<u>Nel</u>	<u>Jaco</u>	<u>Wendy</u>	<u>Walker</u>
<u>Neutze</u>	<u>Jocelyn</u>	<u>Warner</u>	<u>Todd</u>
<u>Newman</u>	<u>David</u>	<u>Wallace</u>	<u>Alexandra</u>
<u>Nicholson</u>	<u>Ross</u>	<u>West</u>	<u>Calre</u>
<u>Nobbs</u>	<u>Peter</u>	<u>Wills</u>	<u>Russell</u>
<u>Nutthall</u>	<u>Gabrielle</u>	<u>Wilson</u>	<u>Nigel</u>
<u>Palmer</u>	<u>Penny</u>	<u>Wilson</u>	<u>Ross</u>
<u>Parsons</u>	<u>Alan</u>	<u>Wilson</u>	<u>Callum</u>
<u>Pattimore</u>	<u>Philip</u>	<u>Wiltshire</u>	<u>Esko</u>
<u>Percival</u>	<u>Teuila</u>	<u>Wilson</u>	<u>Toni</u>
<u>Pereira</u>	<u>Nicola</u>	<u>Wong</u>	<u>Maisie</u>
<u>Pinnock</u>	<u>Ralph</u>	<u>Wong</u>	<u>William</u>
<u>Pringle</u>	<u>Kevin</u>	<u>Wong</u>	<u>Sharon</u>
<u>Ramadas</u>	<u>Ram</u>		
<u>Reith</u>	<u>David</u>		
<u>Richardson</u>	<u>Vaughan</u>		
<u>Robertson</u>	<u>Stephen</u>		

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

