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HEALTH OF ABORIGINAL MOTHERS AND BABIES IN NSW

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SUMMARY

There are, on average, about 1,300 births to Aboriginal mothers in NSW each year – 1.5 per cent of all births in NSW. For Aboriginal mothers overall, pregnancy is characterised by fewer antenatal visits and higher rates of gestational diabetes, premature rupture of membranes and threatened premature labour compared with non-Aboriginal mothers. In 1982-90 the mortality rate among Aboriginal mothers was 47.3 per 100,000 births, which was 5.8 times higher than that of non-Aboriginal mothers. Aboriginal women are more likely to have a spontaneous onset of labour, and less likely to have an induction of labour, a forceps delivery or an elective caesarean section, than non-Aboriginal women. Aboriginal infants are more likely to be low birthweight or premature than non-Aboriginal infants, and are about twice as likely to die in the perinatal period.

METHODOLOGY

Data were obtained from several sources: the NSW Midwives Data Collection (MDC), the NSW Inpatient Statistics Collection, the Registry of Births, Deaths and Marriages, notifications of maternal deaths to the NSW Health Department, and Australian Bureau of Statistics (ABS) census data for 1986 and 1991.

The NSW Midwives Data Collection covers all births in NSW of at least 20 weeks gestation or 400 grams birthweight. The MDC for the years 1986-88 and 1990-91 provided information on demography, pregnancy profile, labour and delivery, and infant characteristics. Unless otherwise specified, analyses refer to these five years. The MDC also provided information on perinatal deaths according to the type of delivery and the mother's number of previous pregnancies (parity). However, the perinatal mortality rates based on these data may be under-estimates because the MDC form is completed before discharge or transfer of the infant, so some late neonatal deaths may not be recorded.

The NSW Inpatient Statistics Collection for the fiscal years 1989-90 and 1990-91 provided information on postpartum complications.

The NSW Registry of Births, Deaths and Marriages provided information on perinatal deaths (other than by type of delivery and parity) for the years 1987-1990. The perinatal mortality rates are based on year of birth, using Registry data for the numerator and MDC data for the denominator.

Aboriginal and non-Aboriginal resident populations as recorded by the ABS from the 1986 and 1991 Censuses were used as denominators to calculate fertility and crude birth rates. The resident population counts provided by the ABS for 1991 were preliminary.

Chi-square statistics were performed to test whether any apparent differences in proportions or rates between groups were statistically significant. A 5 per cent

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significance level was used. Only differences which are statistically significant are reported as differences.

DEMOGRAPHIC INFORMATION

On average, 1,274 births of Aboriginal infants were reported to the MDC each year. This represents 1.5 per cent of all births in NSW. Eighteen per cent of Aboriginal women giving birth were resident in a Sydney Health Area, while 69 per cent were resident in a rural Health Region.

In 1991 the crude birth rate for Aborigines in NSW was 20.2 livebirths per 1,000 population, which was 1.3 times higher than for the non-Aboriginal population. The general fertility rate for Aboriginal women in 1986 was 84.2 livebirths per 1,000 women (aged 15-44 years), which was also 1.3 times higher than for non-Aboriginal women. The highest age-specific fertility rate for Aboriginal women was in the 20-24 years age group (142.9 livebirths per 1,000 women), followed by the 15-19 age group (92.0 livebirths per 1,000 women) (Figure 1). In contrast, for non-Aboriginal women the highest age-specific fertility rate was in the 25-29 age group (113.4 livebirths per 1,000 women).

Aboriginal mothers tended to be younger than non-Aboriginal mothers: 27 per cent of Aboriginal women giving birth were teenagers compared with 5 per cent of non-Aboriginal women. A higher proportion of Aboriginal women giving birth in the Regions were teenagers (30 per cent) than Aboriginal women resident in the Areas (20 per cent). There were higher proportions of Aboriginal teenage mothers in the Orana and Far West and New England Regions than Aboriginal mothers elsewhere in NSW.

Aboriginal women giving birth were more likely to be single than non-Aboriginal women: 56 per cent of Aboriginal mothers were single compared with 11 per cent of non-Aboriginal mothers.

PREGNANCY PROFILE

Aboriginal women tended to be of higher parity (i.e. have had more previous pregnancies) than non-Aboriginal women: 5 per cent of Aboriginal women giving birth had at least five previous pregnancies, compared with 0.7 per cent of non-Aboriginal women. Thirty-four per cent of Aboriginal women gave birth for the first time, compared with 40 per cent of non-Aboriginal women.

Aboriginal women tend to have fewer antenatal visits than non-Aboriginal women: more than 6 per cent of Aboriginal women giving birth had fewer than two antenatal visits throughout their pregnancy in 1986-88 compared with less than 1 per cent of non-Aboriginal women.

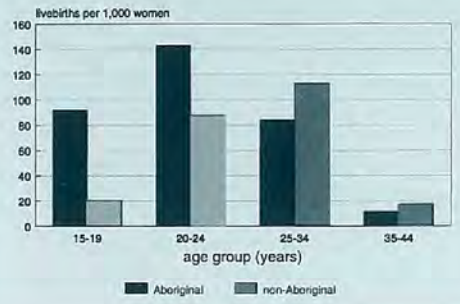
Over the same period, 27 per cent of Aboriginal women had their first antenatal visit after 20 weeks gestation, compared with 11 per cent of non-Aboriginal women. Less than half (49 per cent) of Aboriginal women had their first visit within the first trimester (12 weeks), compared with about two-thirds (65 per cent) of non-Aboriginal women.

The proportion of Aboriginal women having fewer than two antenatal visits during their pregnancy was higher:

- for young women (10 per cent of women aged less than 17 years compared with 4 per cent of women aged 30 years and above);
- in Sydney Areas compared with Regions (9 per cent compared with 6 per cent); and
- in the Central Sydney Area (12 per cent) compared with elsewhere in NSW.

FIGURE 1

AGE-SPECIFIC FERTILITY RATES BY ABORIGINALITY, NSW 1986



For the period 1986-88, general practitioners (GPs) were most commonly responsible for the antenatal care of Aboriginal women in the Regions, and public hospitals were most commonly responsible for their antenatal care in the Areas. Overall, GPs were the single most frequent provider of antenatal care for Aboriginal women, being responsible for the antenatal care of 50 per cent of Aboriginal women, compared with 19 per cent of non-Aboriginal women. Aboriginal women – in both the Areas and Regions – were much less likely than non-Aboriginal women to have antenatal care provided by specialist obstetricians (21 per cent compared with 53 per cent).

The rate of gestational diabetes in 1990-91 for Aboriginal women was 15.4 per 1,000 confinements, which was 1.4 times higher than for non-Aboriginal women. The rates of premature rupture of membranes and threatened premature labour for Aboriginal women in 1986-91 were 22.9 and 21.4 per 1,000 confinements, respectively, which were both 1.5 times higher than for non-Aboriginal women.

Over the period 1982-90 five deaths of Aboriginal women were reported to the NSW Health Department which were directly or indirectly attributable to pregnancy or its management. This represents a maternal mortality rate of 47.3 per 100,000 births, which was 5.8 times higher than the rate for non-Aboriginal women. The rate for direct maternal deaths in Aboriginal women was 37.9 per 100,000 births, which was 7.4 times higher than for non-Aboriginal women.

LABOUR AND DELIVERY

Ten per cent of Aboriginal women giving birth were confined in Level 6 (special obstetric) hospitals, 62 per cent were confined in Level 4-5 (country base/metropolitan district) hospitals and 27 per cent were confined in Level 1-3 (local, small isolated and country district) hospitals. Aboriginal women were less likely than non-Aboriginal women to be confined in Level 6 and Level 4 hospitals, and more likely to be confined in Level 5, Level 3 and Level 2 hospitals.

The onset of labour was spontaneous for 80 per cent of Aboriginal births and induced for 14 per cent of Aboriginal births. Aboriginal women were more likely to have a spontaneous onset of labour and less likely to have an induction of labour than non-Aboriginal women.

FIGURE 2

BIRTHWEIGHT BY ABORIGINALITY, NSW 1986-91

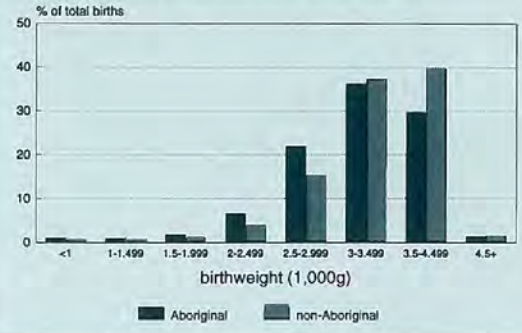
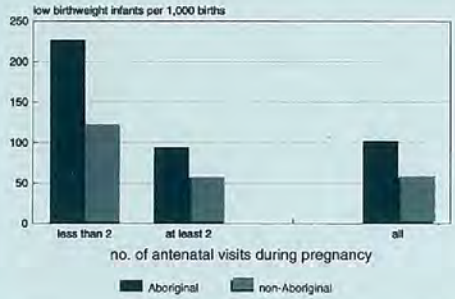


FIGURE 3

LOW BIRTHWEIGHT BY ABORIGINALITY AND ANTENATAL VISITS, NSW 1986-88



Seventy-seven per cent of Aboriginal births were by spontaneous cephalic delivery, 7 per cent were delivered by forceps and 14 per cent were delivered by caesarean sections (elective: 6 per cent, emergency: 8 per cent). Overall, there were no significant differences in types of delivery of Aboriginal infants between the Areas and Regions. However, compared with non-Aborigines, Aboriginal infants were more likely to have spontaneous cephalic deliveries, and less likely to be delivered by forceps and elective caesarean sections. That Aboriginal women were of higher average parity than non-Aboriginal women does not explain the differences in type of delivery between the two groups.

Breech presentations were less common for Aboriginal births than non-Aboriginal births, the rates being 33.9 and 43.4 per 1,000 births respectively. Aboriginal births with breech presentations, however, were more likely to have a vaginal delivery than non-Aboriginal births.

The rate of major puerperal infection for Aboriginal women in 1989-90 and 1990-91 was 11.8 per 1,000 confinements, which was nearly four times higher than for non-Aboriginal women.

INFANT CHARACTERISTICS

The birthweight distribution of Aboriginal infants is skewed towards lower birthweights compared with non-Aboriginal infants (Figure 2). Ten per cent of Aboriginal births were of low birthweight (less than 2,500 grams), which was 1.8 times higher than for non-Aboriginal infants.

The gestational age distribution of Aboriginal infants is also skewed towards lower gestational ages compared with non-Aboriginal infants. Ten per cent of Aboriginal births were premature (less than 37 weeks gestation), which was 1.4 times higher than for non-Aboriginal births.

More than 4 per cent of Aboriginal infants were born at term but of low birthweight (i.e. at least 37 weeks gestation but less than 2,500 grams), which was 2.1 times higher than for non-Aboriginal births.

The majority of both Aboriginal (58 per cent) and non-Aboriginal (65 per cent) low birthweight infants were premature (rather than term low birthweight) births.

The risk of low birthweight for Aboriginal infants was highest for:

- teenage mothers (12 per cent);
- mothers aged 40 years and above (12 per cent);
- women having their first child (12 per cent);
- women who had at least five previous pregnancies (13 per cent); and
- women who had fewer than two antenatal visits during their pregnancy (23 per cent) (Figure 3).

The risk of prematurity for Aboriginal births was highest for:

- teenage mothers (12 per cent);
- mothers aged 40 years and above (12 per cent);
- women having their first child (11 per cent); and
- women who had fewer than two antenatal visits during their pregnancy (21 per cent).

The rates of low birthweight, prematurity and term low birthweight for Aboriginal births were higher than for non-Aboriginal births, even after differences in maternal age and parity and number of antenatal visits were taken into account. This suggests that differences in these factors between Aboriginal and non-Aboriginal mothers, alone, cannot adequately explain the excess risk of poor perinatal outcomes for Aboriginal infants. For example, Figure 3 shows that the risk of low birthweight for infants born to women who had had fewer than two antenatal visits during their pregnancy was about two times higher for Aboriginal than non-Aboriginal infants.

PERINATAL MORTALITY

In 1987-90 there were 120 perinatal deaths among Aboriginal births, of which 75 (63 per cent) died before delivery (stillbirths), and 45 (37 per cent) died within 28 days of birth (neonatal deaths). This represents a perinatal mortality rate of 23.5 per 1,000 births, which was 1.9 times higher than for non-Aboriginal births. The stillbirth rate for Aboriginal infants was 14.7 per 1,000 and the neonatal death rate was 8.8 per 1,000, which were 2.0 and 1.8 times higher respectively than for non-Aboriginal births. Overall, the perinatal death rates for Aboriginal infants were not significantly different between the Areas and Regions.

The risk of perinatal death is higher for infants with lower birthweights. Fifteen per cent of Aboriginal low birthweight infants died in the perinatal period, which was the same proportion as for non-Aboriginal low birthweight infants. This suggests that much of the excess risk of perinatal death for Aboriginal infants is due to the fact that Aboriginal infants are more likely to be born with a low birthweight.

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BOOM IN DEMAND FOR GENETICS SERVICES IN NSW

Jennifer Blackwell, Executive Officer,
NSW Genetics Service, NSW Health Department

Genetic disorders and birth defects impose a heavy physical, emotional and financial burden on individuals, families and the community. Advances in science and technology are expanding knowledge of the role of genes in disease and are leading to better methods of preventing, diagnosing and treating genetic disorders and diseases of multiple causes, including genetic factors. Developments in screening programs, prenatal diagnosis and community education are increasing the availability of, and demand for, genetics services.

A major challenge is to implement new genetic technology in the most beneficial way, to ensure high-quality care and to ensure access to services. A new five-year plan, 1993-1998, provides a framework for the ethical and effective development of genetics services until 1998. Maintaining the principle of an integrated approach encompassing clinical, counselling, educational and laboratory services – centrally coordinated through the NSW Genetics Service Advisory Committee – is seen as the most effective and economical way of achieving the goals of the NSW Genetics Service. The major goals include:

- ensuring the provision of, and access to, genetics services appropriate to the needs of the people of NSW;
- improving the use of information and services relating to genetic disorders and birth defects; and
- reducing the impact of genetic disorders and birth defects on the sufferers, individuals at risk and their families.

Genes contain the information that determines how we grow, what we look like and how well our bodies work. Information in a gene is called the genetic code. Genes are part of chromosomes which are in all the cells of our bodies. We each have 46 chromosomes which contain between 50,000 and 100,000 genes. Chromosomes and genes are made of DNA. Our body's health and growth will be changed if the genetic code is changed or if there are extra or missing genes.

There has been an explosion of knowledge about the processes of heredity and their implications for human health. The international Human Genome Project aims to identify all the 50,000 to 100,000 genes in humans by the year 2005.

Using both molecular biology techniques and family studies, nearly 5,000 disorders have been identified as being caused by a mutation in a single gene. The number of disorders susceptible to molecular genetic or DNA diagnosis is rapidly increasing. In recent months, genes have been located for Huntington disease, motor neurone disease, melanoma and osteoporosis.

Many medical problems evident at birth, or soon after birth, are primarily genetic in origin or include a genetic factor. Other genetic disorders appear later in life. Genetic disorders include: Down's syndrome, neural tube defect, cystic fibrosis, diabetes, fragile X syndrome, muscular dystrophies, spinal muscular atrophies, haemophilia, hearing disorders, neurofibromatosis, polycystic kidney disease, thalassaemias, phenylketonuria, galactosaemia and Huntington disease.

They also include some forms of heart disease, cancer, asthma, epilepsy, visual disorders, short stature syndromes, intellectual disability and hypothyroidism.

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The risk of perinatal death is higher for infants born prematurely. Seventeen per cent of Aboriginal infants born prematurely died in the perinatal period. This was 1.4 times higher than for non-Aboriginal premature births. However, excluding infants born at less than 25 weeks gestation, Aboriginal preterm births were no more at risk of perinatal death than non-Aboriginal preterm births. This suggests that at least some of the excess risk of perinatal death for Aboriginal infants is due to the fact that Aboriginal infants are more likely to be born prematurely.

The risk of perinatal death for Aboriginal births was highest for teenage mothers (30.3 per 1,000 births) (Figure 4), women having their first child (34.0 per 1,000 births), and vaginal breech deliveries (310.3 per 1,000 births).

Extreme prematurity was the single most common cause of perinatal death for Aboriginal infants, accounting for 19 per cent of perinatal deaths. In contrast, extreme prematurity accounted for 12 per cent of non-Aboriginal perinatal deaths. The rate of perinatal death due to extreme prematurity for Aboriginal births was 4.5 per 1,000 births, which was 3.0 times higher than for non-Aboriginal births.

'Maternal conditions which may be unrelated to pregnancy' were associated with 17 per cent of Aboriginal and 10 per cent of non-Aboriginal perinatal deaths. Most of these maternal conditions were hypertensive disorders (high blood pressure). The perinatal death rate associated with maternal hypertensive disorders for Aboriginal births was 2.2 per 1,000 births, which was 3.1 times higher than for non-Aboriginal births.

'Other placental separation and haemorrhage' (i.e. other than placenta praevia) and 'other and unspecified morphological and functional abnormalities of the placenta' were both associated with 12 per cent of Aboriginal perinatal deaths. The perinatal death rates associated with these conditions for Aboriginal births were 2.1 and 3.6 times higher, respectively, than for non-Aboriginal births.

FIGURE 4

PERINATAL MORTALITY BY ABORIGINALITY AND MATERNAL AGE, NSW 1987-90

At least one in 20 people will experience a gene-related disability by the age of 25. In the average lifetime, more than half the population will have a disease with a genetic component. Many chronic diseases of middle and old age appear to be due to multiple causes, including genetic factors. Up to half the admissions to paediatric hospitals, and 12 per cent of adult admissions to general hospitals, are for genetic disorders and about 28 per cent of infant deaths are due to birth defects.

Like newborn screening for phenylketonuria, cystic fibrosis, galactosaemia and hypothyroidism, early detection means effective treatments can be introduced both to relieve suffering and reduce the high costs associated with the long-term care of individuals with chronic disability.

Information and counselling can enable couples at genetic risk to have healthy children through informed choices about reproductive options, including prenatal diagnosis. Many congenital malformations, chromosomal abnormalities and metabolic errors can be diagnosed early in pregnancy. Prenatal testing is widely available for two of the most common disorders, Down's syndrome (among women of advanced maternal age) and neural tube defect.

Prenatal molecular genetic diagnosis is available for couples where the pregnancies are at high risk for disorders such as cystic fibrosis, Duchenne/Becker muscular dystrophy, myotonic muscular dystrophy, polycystic kidney disease, spinal muscular atrophy, Huntington disease, haemophilia, thalassaemia and fragile X syndrome. Long-term social and financial benefits can be achieved through prenatal diagnosis. Carrier testing and presymptomatic diagnosis are also available.

Screening for genetic predisposition could achieve early intervention or prevention of some disorders, reducing distress and costs associated with later complications. There is a genetic component in the cause of many common conditions such as coronary artery disease, diabetes and some cancers, including melanoma, breast and bowel cancer. Dietary or lifestyle changes can be effective. For example, those identified at high risk of lung cancer could reduce the likelihood of developing the disease by avoidance of cigarette smoking. Identification of those at increased cardiovascular risk means dietary and lifestyle modifications can be adopted to reduce risk. Where parents are affected, early identification and intervention will benefit their offspring.

Public education is vital so people are aware of the options available to them to reduce disability.

Pregnancy often provides the first occasion when couples consider reproductive risks related to family history or age, so awareness by general practitioners and obstetricians is especially important. Clients at risk for a genetic disorder or at risk of having a child with a birth defect or genetic disorder can be referred to specialist genetics services for evaluation and counselling.

Clinical services operate principally through four major clinical genetics units: Prince of Wales Children's Hospital (POWCH), Royal Alexandra Hospital for Children (RAHC), Westmead Hospital and in the Hunter Area. These units provide a Statewide network of services to metropolitan and country centres and at prenatal diagnosis clinics in level 5/6 maternity hospitals. In NSW there are nine clinical geneticist positions and four training posts.

The value of non-medical genetic counsellors in making services more accessible has been recognised with the employment of 22 (15 full-time equivalent) counsellors, in the major units and at the Royal Hospital for Women, Royal North Shore Hospital, Wollongong, Gosford, Lismore, Coffs Harbour, Port Macquarie, Tamworth, Broken Hill, Dubbo, Goulburn and Wagga. Genetics clinics are held at King George V, Liverpool and Nepean hospitals. Clinical geneticists and genetic counsellors also hold clinics in other country towns.

A limited number of laboratories provides a Statewide service. Cytogenetics laboratories are associated with each clinical genetics unit at POWCH, RAHC, Westmead and in the Hunter Area. Molecular genetics services operate on a one disorder/one laboratory consortium model where each disorder is studied in only one laboratory in NSW. Statewide services are provided from three laboratory groups: POWCH/Concord, RPAH/RNSH and Hunter. Biochemical genetics and the newborn screening program are at the Oliver Latham Laboratory.

The Statewide genetic education program is based at Royal North Shore Hospital.

The NSW Genetics Service Advisory Committee, in line with international guidelines, recommends a ratio of one geneticist per 300,000 population. To achieve this standard, a further 14 geneticists will need to be trained and employed over the next five years. Based on demographic and geographic factors in NSW, it is recommended that by 1997 three additional clinical genetics units be established in the Northern Sydney Area, Liverpool and Royal Prince Alfred/King George V (RPAH/KGV) hospitals, making a total of six units (assuming Westmead and RAHC become one unit from 1996). A clinical geneticist position is recommended for Wentworth Area with joint appointment at a major unit.

The employment of additional genetic counsellors throughout the State will improve access to information and make services more effective and more economical. Development of services is desirable in Wentworth, South Western Sydney, Southern Sydney, Central Sydney and in Western Sydney.

Expansion of cytogenetics laboratory staff will be required to handle increasing demand for prenatal studies, and particularly at RNSH to support the new clinical genetics unit. Clinical genetics units at Liverpool and RPAH will use existing services for the term of this five-year plan.

Another molecular genetics collaborative group is planned for RAHC/Westmead/Liverpool by 1996-97. The number of hospital scientists will need to rise to manage demand for DNA testing.

Newborn screening is a single Statewide service and would be most appropriately attached to a major children's hospital. This will be achieved with the relocation of Oliver Latham Laboratory to RAHC, with the biochemical genetics services provided by that laboratory.

The Statewide genetic education program will continue to promote community awareness of genetic health issues and of preventive services. The program will develop resources for incorporation into school curricula and community education services. The program also is responsible for keeping health professionals informed of developments and new services.

The increasing importance of DNA technology, diagnosis and potential treatment should encourage Health Areas and Districts to enhance services for their preventive, therapeutic and counselling values. Investment in genetics services is offset in the long term by the reduction in the incidence of chronic conditions and that cumulative savings will be in the interests of health services and the communities they serve.

THE AUSTRALIAN PAEDIATRIC SURVEILLANCE UNIT

Kerry Chant, Research Assistant, APSU, Public Health Officer
Elizabeth Elliott, Co-ordinator, APSU, Senior Lecturer, University of Sydney. Consultant Paediatrician

The Australian Paediatric Surveillance Unit (APSU) in May 1993 began the active surveillance of eight rare paediatric conditions – AIDS, HIV infection and neonatal exposure to HIV, childhood dementia, congenital rubella, drowning/near drowning episodes, extrahepatic biliary atresia, haemorrhagic disease of the newborn, Kawasaki disease and Rett syndrome.

The APSU was formed in June 1992 and became a unit of the Australian College of Paediatrics (ACP) in May 1993. It is modelled on the British Paediatric Surveillance Unit which has been operating in the United Kingdom for eight years.

The aim of the APSU is to facilitate and improve the surveillance of rare paediatric conditions or rare complications of common paediatric conditions. Australia is particularly suited to such a scheme as its low population, large size and separate State health systems have made it difficult to obtain information on the basic epidemiology of many rare conditions.

While a rare condition affects only a small number of children, it may be associated with significant mortality and/or morbidity, result in high utilisation of health care services, and/or have significant social, emotional and financial implications for the child and family. In the case of infectious diseases there may exist the potential for wider community involvement.

Surveillance and case ascertainment, in addition to providing information on the epidemiology of rare conditions, may provide information on the aetiology, management, outcomes and health and support-related needs of the child and family. This information can be used in developing prevention strategies, identifying optimal management approaches and informing decisions about the provision of health and family support services.

The surveillance of cases experiencing a rare complication of a common condition will allow information to be gathered on a subgroup of this population. This may represent a subgroup with greater disease severity, for example, children admitted to an intensive care unit with a diagnosis of asthma. This subgroup may benefit the most from prevention and optimal medical management.

Rare condition surveillance may also be used to evaluate and review prevention strategies. The number of cases of congenital rubella, haemophillus influenzae type b infection in under five-year-olds, and drowning/near drowning episodes in under five-year-olds occurring in a specified period can be used as outcome measures to monitor the effectiveness of prevention strategies.

Rare disease surveillance will also facilitate and promote collaborative research on rare conditions and, by increasing awareness among clinicians, may prompt early diagnoses of these conditions.

A variety of methods is used to monitor the occurrence of rare conditions. These methods can be categorised as follows:

- statutory requirement to notify a condition to an organisation (e.g. NSW Infectious Diseases Surveillance System);
- voluntary notification to an organisation (e.g. British Paediatric Surveillance Unit, NSW Birth Defects Registry);
- death certification; and
- hospital databases (e.g. NSW Inpatient Statistics Collection).

The APSU is an example of a national active case surveillance system reliant on the voluntary notification of selected conditions by participating clinicians. It is described as "active" because each month clinicians are prompted to report selected conditions by being sent a report card to complete.

All paediatricians and other clinicians working predominantly with children in Australia are sent a monthly report card. They are asked to complete the reply-paid report card by indicating the number of cases of the listed surveillance conditions they have seen in the previous month or by ticking the "nothing to report" box.

The APSU maintains a database which records the clinicians' response to the monthly report card. The APSU notifies the investigator responsible for the condition on receipt of a case report. The investigator then forwards a brief questionnaire to the clinician requesting further demographic and clinical details. Identifying patient details are not requested. The first two letters of the first name and surname, date of birth and postcode are used to detect duplicate reports.

Investigators are required to provide feedback to the APSU and reporting clinicians. The investigator advises the APSU of the number of notifications meeting the case definition and the number of duplicate reports and reporting errors. The investigator also provides a brief summary of the study findings for inclusion in the APSU annual report.

The mailing list was derived from lists of paediatricians and related specialists held by the Royal Australian College of Physicians (RACP), ACP, the Health Insurance Commission and paediatric sub-specialist groups. The list is updated regularly.

Investigators complete an application form in which they outline the case definition, aims and objectives, research questions, rationale, ethical issues and funding arrangements for the study. Applications are reviewed by the Scientific Review Panel which consists of several paediatricians and a public health clinician. Applications are occasionally sent for external review by a clinician with expertise in the specialty area of the study.

In the event of an epidemiological emergency the APSU can promptly add a new condition to the card and institute telephone reporting if necessary. This situation may arise in the case of a newly described condition, where there are new concerns about a side effect from a vaccine or other treatment, or where a change in public health policy is to be adopted and the effect of this change needs to be monitored.

Investigators are required to obtain ethics approval from their institution before a condition can be listed on the card.

The response rate for the first five months of the APSU's operation has been about 85 per cent. While we aim for a response rate of more than 90 per cent we are pleased with the progress and appreciate the cooperation of clinicians.

The Australian Paediatric Surveillance Unit can be contacted at The Children's Hospital, PO Box 34, Camperdown NSW 2050.

PUBLIC HEALTH ABSTRACTS

Professor James S. Lawson, Professor and Head of the School of Health Services Management at the University of NSW, has prepared the following public health items from the literature.

SLEEPING POSITION AND SUDDEN INFANT DEATH

Sudden infant death syndrome (SIDS) is the leading cause of death in western communities for infants aged four weeks to one year. Epidemiological studies suggest that prone sleeping (sleeping on the stomach) increases the risk of sudden infant death syndrome. But there have been no physiological data to support the results of these epidemiological studies. This gap has partly been filled by an American study. There are some methodological problems with the study, however, it suggests the prone sleeping position may be related to SIDS because it allows other risk factors, such as overheating, to become active. This may explain why sleeping prone has such a strong relation to the rates of SIDS in Tasmania and New Zealand, where the use of soft mattresses and sheepskins is relatively common, but seems to have little influence in the United States, where the rate of SIDS is much lower even though the proportion of infants sleeping prone is almost twice as high.

But these epidemiological data cannot explain why thousands of babies sleep face down on soft mattresses in overheated rooms and do not die, whereas some who sleep on their backs on hard mattresses in cool rooms do die. It seems a gasping reflex is necessary to maintain breathing in mammals, including humans. It has been postulated that the separation of babies from their mothers in western society removes the intermittent arousal present in those societies where the mother and baby sleep together. This may explain the almost absence of SIDS in, for example, Hong Kong, as compared to western societies. The study by Ponsonby, Dwyer, Gibbons et al was conducted in Tasmania.

Poets CF and Southall DP. Prone sleeping position and sudden infant death. *New Engl J Med*, 1993; 329:6:425-426.
Ponsonby A, Dwyer T, Gibbons LE et al. Factors potentiating the risk of sudden infant death syndrome associated with the prone position. *New Engl J Med*, 1993; 329:6:377-382.

UNIVERSAL HEPATITIS B VACCINATION SUGGESTED

Paul Goldwater of Adelaide has presented a strong case for the universal offering of hepatitis B vaccine to all children in Australia. His argument is based on the following factors:

- Possibly 22,000 cases of hepatitis B occur each year in Australia, and 500-600 of these patients have to be placed in hospital.
- There has been a marked increase in hepatitis B in injecting drug users.
- The cost of immunisation is now low, at $3-$5 for the full paediatric dose.
- Hepatitis B vaccines are very safe and of proven efficacy.

Goldwater PN. History of hepatitis B vaccination in New Zealand: lessons for Australia? *Aust J Public Health*, 1993; 17:221-5.

ADVANCES IN PROTECTION FROM THE SUN

In Australia, where the bronzed Aussie image is still held as aesthetically ideal, malignant melanoma is the most common invasive tumour in adults between 20 and 40 years. About 2 per cent of the total population suffers from malignant melanoma. There have been significant improvements in knowledge about the danger of exposure to the sun and in Queensland nearly 90 per cent of children know that too much sun causes skin cancer. But knowledge is rarely sufficient for an increase in health-promoting behaviours. For example, only 30 per cent of adolescents in NSW use protective measures when out in the sun. A study in Newcastle, NSW, has indicated that the use of sunscreen creams has risen to nearly 70 per cent of beachgoers, but less than 17 per cent are using appropriate hats, shade and shirts. Perhaps a key direction for the future is the provision on beaches and elsewhere of shade by the use of trees or the construction of low-cost sun shelters.

Lowe JB, Balanda KP, Gillespie AM et al. Sun-related attitudes and beliefs among Queensland school children. Aust J Public Health, 1993; 17:202-8.
Foot G, Girgis A, Boyle CA et al. Solar protection behaviours: a study of beachgoers. *Aust J Public Health*, 1993; 17:209-14.

SMOKING WORSENS CORONARY HEART DISEASE

Population-based studies in Newcastle, NSW, have again demonstrated that men who smoke are 2.9 times more likely than non-smokers to have a first myocardial infarction or fatal heart attack, and for women the equivalent figure is 3.5 times. The combination of atherosclerotic coronary heart disease, hypertension and tobacco smoking deserves continuing attention.

Chun BY, Dobson AJ and Heller RF. Smoking and the incidence of coronary heart disease in an Australian population. *Med J Aust*, 1993; 159:508-512.

ADMINISTRATIVE COSTS IN HOSPITALS

A United States study has shown that one dollar in every four spent by American hospitals goes on administrative costs. This appears to be twice as high as in Canada. While administration is a necessity, such vast allocation to administration in American hospitals means the money is not available for the clinical care of patients.

Woolhandler S, Himmelstein DU, Lewontin JP. Administrative costs in US hospitals. *New Engl J Med*, 1993; 329:6:400-403.

METHADONE PRESCRIBING IN AUSTRALIA INCREASES

Methadone prescription is a major treatment for opiate dependence in Australia and elsewhere. Participation in a methadone program can reduce or eliminate illicit opiate use and the health and social problems associated with opiate dependence. Accordingly, methadone prescription should be given high priority as a public health activity. Since 1985 methadone prescription rates have remained relatively stable in all States with the exception of NSW and Victoria, where the rate has risen by a factor of 500 per cent. There has been an expansion in both the public and private sector in NSW and Victoria, but the greatest expansion has been in the private sector which, in NSW, increased from 50 in 1985 to 2,917 in 1990. In the absence of knowledge about the prevalence of injecting opiate use, it is not known whether the provision of methadone is adequate.

Gaughwin M, Kliewer E, Ali R et al. The prescription of methadone for opiate dependence in Australia, 1985-1991. *Med J Aust*, 1993; 159:107-108.

INFECTIOUS DISEASES

NOTIFICATIONS

PERTUSSIS (WHOOPING COUGH)

The pertussis notification rate for NSW for the period January 1 to December 31, 1993, was 22.2/100,000 population. This compares with a rate of 23.6 for the first 11 months of the year. South Western Sydney Public Health Unit received notifications at a rate of 32.8/100,000 population.

A total of 1,346 notifications for pertussis was received in 1993. This is more than six times the number of notifications received for 1992.

Immunisation providers are asked to consider the consequences of not offering whooping cough vaccine to infants and children when there is documented evidence of high levels of *Bordatella pertussis* throughout the State.

MEASLES

The annual notification rate for the State was 38.5/100,000 population. This compares with a rate of 40.6 for the first 11 months of 1993.

Western Sydney Public Health Unit received notifications at a rate of 109.3/100,000 population.

Measles notifications in Western Sydney peaked in epiweek 44.

The mean age for notifications was 9.5 years (range one month to 69 years). Nine per cent of notifications were for neonates and infants. Seventy-two per cent were for children over the age of five; 28 per cent of cases were for people over the age of 12 years.

HEPATITIS E

A 23-year-old woman presented to a local hospital with a history of diarrhoeal illness and jaundice. Six weeks before onset of symptoms the patient had travelled in India and then middle Europe (Portugal and Spain). While in Spain she had sought medical advice for gastroenteritis but did not respond to treatment.

Serology results from Fairfield Hospital in Melbourne indicated Hepatitis E virus EIA antibody positive and past infection with Hepatitis B virus with probable immunity. Epidemics consistent with Hepatitis E virus have been identified in the Indian subcontinent (Benenson:211).

NEW YELLOW FEVER CLINIC

Dr Joe McGirr
Accident and Emergency Department
Wagga Wagga Base Hospital
Docker Street
WAGGA WAGGA NSW 2650

NON-NOTIFIABLE STD SURVEILLANCE

The term non-gonococcal urethritis (NGU) is usually used to describe sexually transmitted urethritis in males where *Neisseria gonorroeae* cannot be isolated. In North America and Europe the incidence of NGU has overtaken that of gonococcal urethritis in the past decade, partly because of decreases in the incidence of gonorrhoeal infection. A total of 323 cases of gonorrhoea was notified in NSW for 1993 by laboratories, and 1,050 cases of NGU by sexual health clinics. The most frequent known causes are *Chlamydia trachomatis* (30-50 per cent), *Ureaplasma urealyticum* (10-40 per cent), and rarely, herpes simplex virus (HSV), *Trichomonas vaginalis* and others. As with *N. gonorrhoeae,* asymptomatic infections are common. *N. gonorrhoea, C. trachomatis* and HSV are the usual causative agents of sexually transmitted urethritis in females, and a small number of cases of NGU was reported in females in 1993.

TABLE 1

NOTIFICATIONS OF NON-NOTIFIABLE SEXUALLY TRANSMITTED DISEASES JANUARY-DECEMBER 1993
(Diagnoses from sexual health centres unless otherwise stated in footnote)

AHS Infection		CSA[1]	SSA[2]	ESA[3]	SWS[3]	WSA[4] + WEN	NSA[1]	CCA[1]	ILL[5]	HUN[3]	NCR[1]	NER[1]	OFR[3]	CWR[6]	SWR[7]	SER[8]
Chlamydia trachomatis	Male	3	4	64	3	23	3	–	8	11	2	4	13	–	12	
	Female	1	5	52	6	16	1	1	4	32	2	14	13	–	27	
	Total	**4**	**9**	**116**	**9**	**39**	**4**	**1**	**12**	**43**	**4**	**18**	**26**	–	**39**	**4**
Donovanosis	Male	–	–	–	–	–	–	–	–	–	–	–	–	–	–	
	Female	–	–	–	–	–	–	–	–	–	–	–	–	–	–	
	Total	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
*Genital herpes	Male	10	13	222	3	35	12	7	7	21	5	3	3	–	3	
	Female	10	10	143	2	18	3	9	8	24	6	6	5	–	17	
	Total	**20**	**23**	**365**	**5**	**53**	**15**	**16**	**15**	**45**	**11**	**9**	**8**	–	**20**	**3**
*Genital warts	Male	47	77	490	57	155	33	31	62	93	41	16	20	–	2	
	Female	27	60	214	24	65	19	14	25	37	23	22	15	–	1	
	Total	**74**	**137**	**704**	**81**	**220**	**52**	**45**	**87**	**130**	**64**	**38**	**35**	–	**3**	**15**
Nongonococcal urethritis	Male	10	14	525	11	279	14	15	52	69	20	6	13	–	1	
	Female	2	–	–	3	3	4	5	–	–	4	–	1	–	–	
	Total	**12**	**14**	**525**	**14**	**282**	**18**	**20**	**52**	**69**	**24**	**6**	**14**	–	**1**	–
Lymphogranuloma venereum	Male	–	–	–	–	–	–	–	–	–	–	–	–	–	–	
	Female	–	–	–	–	–	–	–	–	–	–	–	–	–	–	
	Total	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–

* First diagnosis; 1. 01/01/93-30/11/93; 2. 01/01/93-31/10/93; 3. 01/01/93-31/08/93; 4. 01/01/93-31/07/93; 5. 01/01/93-30/06/93;
6. No SHC in Region; 7. Laboratory and SHC data 01/01/93-30/11/93; 8. No SHC in Region. Data from GP network 01/01/93-31/10/93.

TABLE 2

FOODBORNE INFECTIOUS DISEASE NOTIFICATIONS BY PUBLIC HEALTH UNIT, CUMULATIVE 1993

Condition	CSA	SSA	ESA	SWS	WSA	WEN	NSA	CCA	ILL	HUN	NCR	NER	OFR	CWR	SWR	SER	Total
Foodborne Illness (NOS)	7	4	–	24	24	10	–	3	6	3	–	2	15	14	5	–	117
Gastroenteritis (Instit)	80	6	–	19	16	29	1	21	–	114	–	17	4	20	32	–	359
Hepatitis A – Acute Viral	48	22	42	55	116	20	52	12	19	16	56	72	6	5	16	6	563
Listeriosis	2	–	1	3	2	–	1	–	–	4	–	–	–	–	–	–	13
Salmonella (NOS)	27	58	56	77	31	10	64	28	12	73	79	49	31	7	17	13	632
Salmonella bovis morbificans	1	5	2	2	2	–	3	–	–	11	–	–	–	1	2	–	29
Salmonella typhimurium	18	27	21	20	18	13	20	4	3	22	11	11	18	4	14	10	234
Typhoid and paratyphoid	1	2	4	3	2	2	6	–	–	1	2	–	–	3	–	–	26

TABLE 3

SUMMARY OF NSW INFECTIOUS DISEASE NOTIFICATIONS DECEMBER 1993

Condition	Number of cases notified			
	Period		Cumulative	
	Dec 1992	Dec 1993	Dec 1992	Dec 1993
Adverse reaction	–	1	31	29
AIDS	16	21	311	372
Arboviral infection	9	8	344	647
Brucellosis	1	–	4	4
Cholera	–	1	–	1
Diphtheria	–	–	–	–
Foodborne illness (NOS)	10	2	193	117
Gastroenteritis (instit.)	4	4	418	359
Gonorrhoea	37	14	504	326
H influenzae epiglottitis	7	3	56	34
H influenzae B – meningitis	9	1	107	54
H influenzae B – septicaemia	3	2	28	25
H influenzae infection (NOS)	–	–	32	14
Hepatitis A	48	17	983	563
Hepatitis B	204	109	3,286	3,599
Hepatitis C	314	276	4,295	6,280
Hepatitis D	1	–	8	11
Hepatitis E	N/A	–	N/A	1
Hepatitis, acute viral (NOS)	–	–	17	6
HIV infection	31	18	683	512
Hydatid disease	–	–	5	3
Legionnaires' disease	10	2	103	61
Leprosy	–	–	5	3
Leptospirosis	3	1	22	16
Listeriosis	1	1	16	13
Malaria*	20	4	164	159
Measles	125	237	830	2,269
Meningococcal meningitis	9	8	91	96
Meningococcal septicaemia	–	3	17	42
Meningococcal infection (NOS)	–	–	12	11
Mumps	2	2	23	12
Mycobacterial tuberculosis	35	9	424	330
Mycobacterial – atypical	27	–	371	265
Mycobacterial infection (NOS)	2	3	38	74
Pertussis	30	87	222	1,346
Plague	–	–	–	–
Poliomyelitis	–	–	–	–
Q fever	15	9	216	366
Rubella	61	15	340	683
Salmonella infection (NOS)	60	29	860	895
Syphilis	47	29	948	710
Tetanus	–	–	2	5
Typhoid and paratyphoid	2	–	29	26
Typhus	–	–	–	–
Viral haemorrhagic fevers	–	–	–	–
Yellow fever	–	–	–	–

* from Malaria Register

TABLE 4

INFECTIOUS DISEASE NOTIFICATIONS BY SELECTED MONTH OF ONSET FOR 1993

Condition	Month				
	Sep	Oct	Nov	Dec	Total
Adverse event after immunisation	8	1	–	1	10
AIDS	33	36	22	21	112
Arboviral infection	6	15	15	8	44
Brucellosis	1	–	–	–	1
Cholera	–	–	–	1	1
Foodborne illness (NOS)	16	2	11	2	31
Gastroenteritis (instit.)	24	24	62	4	114
Gonorrhoea	14	28	25	14	81
H influenzae epiglottitis	–	1	–	3	4
H influenzae meningitis	3	2	1	1	7
H influenzae septicaemia	1	2	–	2	5
H influenzae infection (NOS)	3	1	–	–	4
Hepatitis A – acute viral	43	46	37	17	143
Hepatitis B – acute viral	8	3	8	1	20
Hepatitis B – unspecified	361	344	355	108	1,168
Hepatitis C – acute viral	2	3	5	–	10
Hepatitis C – unspecified	621	626	740	276	2,263
Hepatitis D – unspecified	1	2	–	–	3
HIV infection	39	33	34	18	124
Hydatid disease	–	–	2	–	2
Legionnaires' disease	5	4	5	2	16
Leprosy	1	–	–	–	1
Leptospirosis	1	2	1	1	5
Listeriosis	–	5	1	1	7
Malaria	16	3	7	4	30
Measles	378	491	583	237	1,689
Meningococcal meningitis	18	17	12	8	55
Meningococcal septicaemia	3	4	5	3	15
Meningococcal infection (NOS)	1	2	–	–	3
Mumps	4	1	3	2	10
Mycobacterial – atypical	15	7	7	–	29
Mycobacterial tuberculosis	25	22	14	9	70
Mycobacterial infection (NOS)	12	16	10	3	41
Pertussis	205	292	281	87	865
Q fever	31	28	25	9	93
Rubella	124	143	116	15	398
Salmonella (NOS)	21	36	87	22	166
Salmonella bovis morbificans	1	1	1	1	4
Salmonella typhimurium	15	16	10	6	47
Syphilis	50	55	80	29	214
Typhoid and paratyphoid	3	5	1	–	9
Total	2,113	2,319	2,566	916	7,918

Continued on page 12 ▶

Abbreviations used in this Bulletin:
CSA Central Sydney Health Area, SSA Southern Sydney Health Area, ESA Eastern Sydney Health Area, SWS South Western Sydney Health Area, WSA Western Sydney Health Area, WEN Wentworth Health Area, NSA Northern Sydney Health Area, CCA Central Coast Health Area, ILL Illawarra Health Area, HUN Hunter Health Area, NCR North Coast Health Region, NER New England Health Region, OFR Orana and Far West Health Region, CWR Central West Health Region, SWR South West Health Region, SER South East Health Region, OTH Interstate/Overseas, U/K Unknown, NOS Not Otherwise Stated.

Please note that the data contained in this Bulletin are provisional and subject to change because of late reports or changes in case classification. Data are tabulated where possible by area of residence and by the disease onset date and not simply the date of notification or receipt of such notification.

IMPROVING THE QUALITY OF HIV DATA

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[2] Eastern Sydney Public Health Unit

It is universally agreed that provision of timely and accurate epidemiological data on newly diagnosed cases of human immunodeficiency virus (HIV) infection is essential for the control of this disease. It would be reasonable to expect that information obtained from HIV testing laboratories, especially that concerning patients in the early stages of the disease, would be valuable in tracking and influencing the course of the epidemic.

In NSW, where the vast majority of this country's cases occur, confirmatory testing of positive HIV screening tests is performed in only four laboratories. Since the advent of HIV testing these laboratories have been the major source of data on all confirmed "new diagnoses" of HIV infection, and there has been an expectation that this would allow ready access to all laboratory data pertaining to confirmed "new diagnoses". However, despite this apparent advantage and the generous allocation of resources for HIV/AIDS surveillance to the NSW Health Department, the quality of data collected has been poor. A previous NSW Health Minister described it as "woeful".

In early 1992, when we reviewed the situation, we found an intolerable lack of basic and important information had been collected on the more than 15,000 "new diagnoses" of HIV contained in the National HIV Database. For example, in more than 18 per cent of the NSW cases the sex of the patient was unknown; nationally, age was not recorded in 24 per cent of cases, and in more than 40 per cent exposure category was not stated. At that time the information supplied to the NSW Health Department and in turn to the National HIV Database by laboratories was derived solely from information given by requesting practitioners on the HIV request form.

An examination of 400 request forms received by the Prince of Wales HIV Reference Laboratory in 1991 revealed a lack of information similar to that contained in the National HIV Database. Moreover, only one third of the request forms contained unique 2+2 patient identifiers (i.e. at least the first two letters of each of the given name and surname), a further 20 per cent were identified by a clinic code number and the remainder were given simply a private code by the doctor or allocated a laboratory accession number. The paucity of identifying information made disaggregation of data virtually impossible by either the Reference Laboratory or NSW Health Department and cast doubt on the validity of the number of "new diagnoses" stated to be in the National HIV Database.

IMPLEMENTATION OF THE CALL-BACK SYSTEM

To improve the quality of data, a system of "call-back" to referring practitioners by mail/telephone was introduced from April 22, 1992, for patients whose specimens tested positive for the first time in the Prince of Wales Hospital HIV Reference Laboratory and who were not known to have been tested as HIV antibody positive elsewhere. Where the specimen had been sent by another laboratory, it was necessary to obtain the name and address of the original requesting medical officer to whom the questionnaire was to be sent and the patient identifier used by that doctor. In such cases, this initial task could be carried out only by telephoning the referring laboratory and was the most time-consuming part of the call-back system.

Once the medical officer's details were obtained, a letter over the signature of the Medical Officer of Health for the Eastern Sydney Area Health Service was sent by certified mail to the referring doctor. The letter outlined the laboratory's obligation to supply information under the Public Health Act 1991 and sought the doctor's cooperation in obtaining this information. Enclosed with the letter was a notification form containing the 11 fields to be completed, with a reply-paid envelope addressed to the Medical Officer of Health. If no reply was received within one month a gentle reminder with another notification form and reply-paid envelope was sent to the referring practitioner. All correspondence was contained in envelopes marked "Confidential" and return envelopes were delivered unopened to one of the senior officers in the HIV Reference Laboratory who entered the data and checked for possible duplication and inconsistencies. If no reply was received to the second letter after a further month had elapsed, the Director of the Eastern Sydney Public Health Unit telephoned and reminded the doctor to complete the form.

The data obtained were entered onto a database program designed for the purpose. This program is part of a PC network with strictly limited access.

RESULTS

In the 18 months from April 22, 1992 to October 31, 1993, 286 new diagnoses of HIV infection were confirmed in the HIV Reference Laboratory, Prince of Wales Hospital. In 13 cases all the fields were completed on the request form by the referring doctor and no further action was needed. In the remaining 273 cases it was necessary to initiate the call-back procedure. The overall response to the call-back by referring practitioners was excellent, with a 100 per cent compliance with the scheme. Two hundred and forty-five (89.7 per cent) of the referring doctors returned the completed forms in response to the first letter, 19 (7 per cent) responded to the reminder letter while only nine needed to be prompted with a telephone call.

There were 11 fields requiring completion by the referring doctor and despite this expanded questionnaire the response was highly satisfactory. The fields least likely to be completed were the unique patient identifiers and the patient's home postcode (each 96.9 per cent complete) while the percentage completion of all other fields was 98 per cent or better. Table 9 compares the completeness of data supplied on the request forms sampled from 1991 and those obtained by call-back in 1992-93.

DISCUSSION

The successful call-back system was implemented without the allocation of additional resources to the Reference Laboratory or the PHU.

The advantage of having initially involved the senior staff was seen as the scheme progressed. Referring doctors recognised the involvement of the senior staff and responded accordingly. For example, in the few instances where they were unable to complete a field they generally included an unsolicited explanation of the cause of the omission. There has been a considerable reduction in the number of reminder letters required and in the past 12 months a telephone reminder has been needed only once.

TABLE 5

COMPLETENESS OF HIV DATA BEFORE AND AFTER INSTITUTION OF 'CALL BACK' SYSTEM

Information	Percentage of each field completed	
	1991 Request forms	1992-93 after call back
Unique patient identifier	33	96.9
Patient's home postcode	44	96.9
Sex	84	100
Date of birth	93	100
Exposure category	54	99
Clinical status	59	98.6
History of previous HIV test	64	100

It is still unclear why so many years elapsed before any real attempt was made to improve the quality of data collected on HIV infection. Perhaps it needed the force of the Public Health Act 1991 which made HIV infection a notifiable disease, although legislation has not been found necessary to achieve high quality data for other disease registries. However, two points became obvious to us soon after initiating the call-back system. The first was that there was no reluctance on the part of referring doctors to supply adequate information provided they were asked specifically for it and given a reasonable explanation for the request. Second, to be successful the call-back process needs to be a collaborative effort involving the testing laboratory and the PHU. This second observation is not surprising in view of the expertise and resources available in PHUs for collecting data on infectious diseases.

EDITORIAL COMMENT

The recently publicised case of patient-to-patient HIV transmission[1] has raised the question of whether this situation may have occurred again, before or since, and the ability of HIV/AIDS surveillance in NSW to detect such an event.

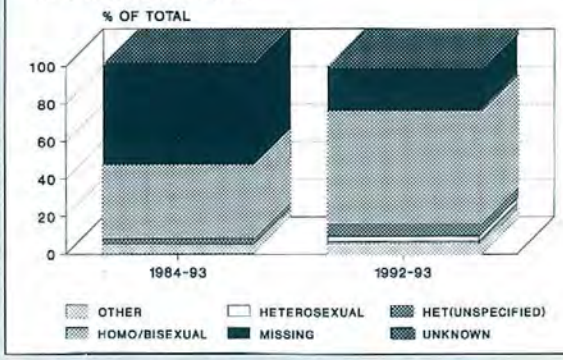
HIV testing that is readily available and free provides the potential for comprehensive surveillance. While there is an overriding need to ensure patient confidentiality, good quality data should include enough personal information to exclude the notification of the same individual for more than one positive test. This has been an insurmountable obstacle for most countries – Australia is one of only four known countries that routinely monitor and publish data on all HIV diagnoses. The others are New Zealand, the United Kingdom and Papua New Guinea. Considerable improvements have been made in NSW, although other laboratories have not been able to provide data as complete as that from Prince of Wales Hospital (POW). Figure 5 shows the impact of the introduction of the callback system by all laboratories in early 1992. The proportion of cases where information on risk exposures is missing has been reduced from 52 per cent overall to 22 per cent in the year 1992-93.

Apart from missing data, two categories of exposure are of concern in monitoring unusual means of transmission – unspecified heterosexual contact and the "unknown" category (figure 5). Reported unspecified heterosexual contact (where no more information on the source of infection is available or provided), may be a default category in some cases where another source of infection is not known or not revealed by the patient. The "unknown" category covers only a very small number of notifications which do not fall into existing categories, the majority being where common exposures are denied by the patient.

A national study of HIV surveillance data from January to October 1991 (before the call-back system began) collected more detail via a questionnaire on all notifications where

FIGURE 5

NSW HIV NOTIFICATIONS



exposure was not reported as either male homosexual contact or blood transfusion[2]. Of questionnaires returned, the two categories of main interest were:

- those where exposure was originally missing – 40 per cent were actually not new HIV diagnoses, 33 per cent were found to be new diagnoses actually due to male homosexual contact, and most of the remainder were found to be due to heterosexual contact; and
- those where exposure was originally reported as unspecified heterosexual contact – 26 per cent were old diagnoses and 16 per cent were new diagnoses due to male homosexual contact. Of those reported on the questionnaire as unspecified heterosexual contact, the doctor was satisfied with the patient's report in 64 per cent of cases.

Therefore, on further investigation, of those with either missing data on exposure or reported unspecified heterosexual contact, only 22 (25 per cent) remained in these categories after the questionnaire had been returned.

The HIV surveillance data from Prince of Wales Hospital for the period July 1, 1992 to September 30, 1993 are among the most complete available in NSW as laboratory callback was operating throughout this period. Of 188 notifications of new diagnoses, in 18 cases (9.6 per cent) the doctor was unable to ascertain the exposure category and 16 (8.5 per cent) reported unspecified heterosexual contact. Further, of the 2,673 NSW cases of AIDS notified to November 30, 1993, in 34 (1.3%) the exposure category was not established and 29 were not interviewed.

There is considerable room for improvement. The Chief Health Officer has written to Area Chief Executive Officers and District General Managers about this issue. In future rates of missing information for all notifications will be expected to be of the same standard as that from POW. Relevant PHUs will be encouraged to assist as Eastern Sydney Area PHU does for POW. The study mentioned above was intended as a pilot of an ongoing method of improved data collection. From January 1994 all notifications, except those reporting male homosexual contact, will be followed up by questionnaire, to verify the report, collect more information and detect cases requiring further investigation. In cases where the mode of transmission has not been established, a formal investigation will begin, subject to patient consent. The first stage of the investigation will be a standard patient interview similar to that developed by the US Centers for Disease Control for investigating such cases.

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1. Chant K, Lowe D, Rubin G et al. Patient-to-patient transmission of HIV in private surgical consulting rooms. *Lancet* 1993; 342:1548-9.
2. McDonald A. Personal communication, 1994.

NEWS AND COMMENT

NSW SENTINEL PRACTICE NETWORKS AWARDED ASSESSMENT POINTS

Illawarra Public Health Unit (PHU) has successfully applied to the Royal Australian College of General Practitioners (RACGP) on behalf of the NSW Sentinel GP Networks for consideration for Practice Assessment Quality Assurance point allocation. The RACGP has indicated it will award 15 practice assessment points per triennium.

GPs are required to be credited at least 20 practice assessment points every three years, as well as points for continuing medical education, as part of the Quality Assurance (QA) program of the RACGP. GPs fulfilling these requirements are entered on the Vocational Register, which qualifies them for higher medicare rebates.

Point allocation for participation in the NSW Sentinel Practice Networks depends on the following:

- each GP must participate in the project for the full triennium (1993-95);
- the RACGP requires a list from each PHU of the names, addresses and QA reference numbers of all participating GPs;
- each GP must submit a final report of the project to the RACGP in November 1995; and
- should individual GPs enter or leave the project during the triennium they will be considered for adjusted point allocation (e.g. five points a year).

SECOND NSW PUBLIC HEALTH NETWORK CONFERENCE

The second NSW Public Health Network Conference will be held at Westmead Hospital on March 29 and 30 this year. Registration forms and a draft program for the conference, titled Promoting Public Health – Achievements and Initiatives, are available from Public Health Units. More than 60 papers will be presented, and there will be plenary sessions, workshops and interactive sessions. Registration fees are $80 for attendance on both days, or $50 for one day. Registrations close on March 4.

ADDENDUM: NSW MIDWIVES DATA COLLECTION REPORT 1992

Table 20 (page 23) should include the following footnote: Vaginal tears for John Hunter Hospital include first, second and third degree tears.

Infectious diseases

► *Continued from page 9*

TABLE 6

INFECTIOUS DISEASE NOTIFICATIONS BY PUBLIC HEALTH UNIT, CUMULATIVE 1993

Condition	CSA	SSA	ESA	SWS	WSA	WEN	NSA	CCA	ILL	HUN	NCR	NER	OFR	CWR	SWR	SER	U/K	Total
Adverse event after immunisation	1	3	2	–	7	–	1	–	1	2	1	4	–	5	2	–	–	29
AIDS	81	14	126	18	19	14	40	4	5	6	29	1	2	5	8	–	–	372
Arboviral Infection	1	1	2	1	1	4	8	1	1	35	72	30	111	15	360	4	–	647
Brucellosis	1	1	–	–	–	–	1	–	–	–	1	–	–	–	–	–	–	4
Cholera	–	1	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1
Gonorrhoea	55	18	108	18	22	6	24	6	3	8	13	11	20	7	3	4	–	326
H. influenzae epiglottitis	1	7	1	3	–	2	4	1	2	3	2	2	1	–	2	3	–	34
H. influenzae meningitis	4	4	–	9	3	3	5	3	8	1	4	3	1	3	2	1	–	54
H. influenzae septicaemia	1	3	1	10	1	–	2	–	2	2	1	2	–	–	–	–	–	25
H. influenzae infection (NOS)	–	–	2	–	2	1	3	2	–	3	–	–	1	–	–	–	–	14
Hepatitis B – acute viral	7	5	21	2	9	1	–	1	–	–	33	5	3	–	2	3	–	92
Hepatitis B – unspecified	538	450	24	1,070	540	45	476	43	52	82	70	43	19	17	27	11	–	3,507
Hepatitis C – acute viral	1	–	–	–	4	–	–	2	1	3	2	6	3	1	–	3	–	26
Hepatitis C – unspecified	756	415	811	685	579	121	655	247	335	423	781	89	29	89	140	99	–	6,254
Hepatitis D – unspecified	2	1	3	1	1	–	–	–	–	1	1	1	–	–	–	–	–	11
Hepatitis E – unspecified	–	–	–	–	–	–	1	–	–	–	–	–	–	–	–	–	–	1
Hepatitis, acute viral (NOS)	–	–	2	–	–	–	–	–	–	1	–	1	–	2	–	–	–	6
HIV infection	72	12	201	15	13	8	38	8	3	13	12	1	1	–	5	2	108	512
Hydatid disease	–	–	2	–	–	–	–	–	–	–	–	–	–	1	–	–	–	3
Legionnaires' disease	11	1	–	14	14	1	4	2	3	5	1	–	1	2	1	1	–	61
Leprosy	–	1	–	1	1	–	–	–	–	–	–	–	–	–	–	–	–	3
Leptospirosis	–	–	–	–	–	–	–	–	–	4	5	3	1	–	3	–	–	16
Malaria	14	15	22	7	19	5	32	4	4	13	4	10	2	1	3	4	–	159
Measles	122	149	75	305	694	222	81	48	111	61	124	83	129	15	25	25	–	2,269
Meningococcal meningitis	3	6	4	16	12	3	7	6	8	5	7	3	6	2	1	7	–	96
Meningococcal septicaemia	4	8	3	4	2	4	5	–	2	3	2	3	1	–	–	1	–	42
Meningococcal infection (NOS)	–	–	1	–	–	–	1	2	1	1	–	–	4	1	–	–	–	11
Mumps	1	3	–	4	1	–	1	–	1	1	–	–	–	–	–	–	–	12
Mycobacterial – atypical	54	20	22	15	24	7	33	7	8	33	24	10	2	1	4	1	–	265
Mycobacterial tuberculosis	33	45	27	68	51	9	40	8	8	17	6	3	3	6	5	1	–	330
Mycobacterial infection (NOS)	20	1	2	2	5	–	23	3	9	2	3	1	1	–	2	–	–	74
Pertussis	51	156	133	212	127	65	171	18	48	55	144	31	66	55	5	9	–	1,346
Q fever	–	1	1	2	6	2	2	1	2	26	75	125	91	12	4	16	–	366
Rubella	7	15	15	86	101	46	132	12	11	98	51	80	1	5	12	11	–	683
Syphilis	97	42	99	170	48	7	34	7	6	8	55	40	77	6	9	5	–	710
Tetanus	–	1	–	–	–	–	–	–	–	–	2	–	1	–	–	1	–	5