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ASTHMA AND AIR POLLUTION IN SYDNEY

Visible air pollution in the Sydney metropolitan area on May 5, 6, 7 and 8, 1991 — caused by burning of firebreaks to the west and south-west of Sydney — was reported vividly in the press[1]. The news reports highlighted the fact that asthma is very common among our schoolchildren: 15 per cent of children report a doctor diagnosis of asthma and 20 per cent report a convincing history of asthma symptoms[2]. The reports implied that air pollution is a major cause of childhood asthma.

To investigate a possible link between this episode and asthma, we counted asthma attendances over a three-week period which straddled the episode — from April 28 to May 18. We chose to survey five metropolitan hospitals: Liverpool, Mt Druitt, Westmead, Prince of Wales and Sutherland, on the basis of their size, location and reasonable proximity to air pollution monitoring stations.

All attendances at these hospitals for either asthma or wheezing were identified from the triage register of each Accident and Emergency Department. Hourly ambient levels of ozone, oxides of nitrogen and particulates were obtained from the Sydney Air Pollution Monitoring Network operated by the State Pollution Control Commission. For this study, the only particulate data available was for smoke and dust particles with a mean aerodynamic diameter less than 2μm.

Between May 5 and 8, particulate levels were high at every monitoring station in the Sydney metropolitan area. The highest levels were at the Woolooware recording station (Fig 1). The peaks observed here between 6am and 8am also occurred at other stations, but were smaller. Levels of the other major pollutants were below average and well below existing air quality standards in NSW.

When we pooled the number of asthma attendances at all five hospitals there was an apparent excess in the number of cases attending on Monday, May 6 — the second day of the pollution episode. Peaks also occurred on each Sunday in this period, no matter what the pollution level (Fig 2).

An analysis of variance of data from individual hospitals was used to compare the mean number of attendances for asthma over the four days of high pollution with the 17 days of low pollution. We adjusted for the rise in attendances seen every Sunday. At Sutherland and Prince of Wales hospitals there were statistically significant rises in the mean number of attendances on high pollution days. At two of the other hospitals there was a small increase and at the third a small decrease in mean asthma attendances on the days

Continued on page 73 ►

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► Continued from page 72

of high pollution (Table 1). None of these variations was statistically significant.

The rise in attendances on one day, May 6, at Prince of Wales accounted almost entirely for the increase seen at that hospital and at all hospitals combined. The large increase on this day at Prince of Wales did not occur on the day of highest pollution in that area. On the day after this large increase, when pollution levels were even higher, asthma attendances at Prince of Wales Hospital were lower than average. There was no statistically significant correlation on a day-to-day basis between measurements of particulate pollution either on days of high pollution *($r = -0.35^a$, $p = 0.65$)* or on days of low pollution *($r = -0.32^a$, $p = 0.21$)*. Similarly, the levels of particulate pollution and asthma attendances the next day and the day after were not significantly correlated.

These data provide weak support for a link between the episode of particulate pollution in Sydney in May and asthma attendances at five metropolitan hospitals over the same period. Against such a link are the facts that a substantial rise in attendances was seen at only one hospital in Sydney and that the day-to-day correlation of attendances with particulate pollution levels was poor. Furthermore, asthma attendances normally rise in May and remain high through the winter months, probably as a result of a higher number of respiratory infections in the community. These seasonal factors may have been the cause of some of the increases observed.

There are limitations to the interpretation of these data. Three weeks is too short a time to assess fully the seasonal and weekly variation in asthma attendances. The influence of behavioural factors on these attendances, particularly the public response to media reports, is unknown.

[a] Pearsons correlation co-efficient

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EDITORIAL NOTE

This is one of the few occasions in which routinely collected air pollution monitoring data has been used to study possible links between air pollution and asthma in Sydney. The air pollution episode in May 1991 may have been prevented by better timing of controlled burning. Smoke can provoke broncho-constriction and an increase in asthma attendance would not be surprising after such an event. A number of other reports have shown an association between the prevalence of asthma and bronchitis and particulate pollution caused by industrial emissions[3,4] and wood smoke[5].

The adverse effects of projected urban development in the west and south-west of Sydney on air quality were highlighted in a recent report[6]. These projections and the findings of this study underline the need for better evaluation of associations between all major air pollutants and illness and mortality in our cities.

Acknowledgements: Margaret Williamson, Helen Moore, State Pollution Control Commission, staff of Casualty Departments at Mt Druitt, Liverpool, Prince of Wales, Westmead and Sutherland hospitals, Eastern Sydney Public Health Unit, South West Sydney Public Health Unit, Western Sydney Public Health Unit.

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FIGURE 1

MEASUREMENTS OF PARTICULATE AIR POLLUTION (SMOKE) MEASURED BY NEPHELOMETRY, WOOLOOWARE MONITORING STATION, MAY 5-8, 1991

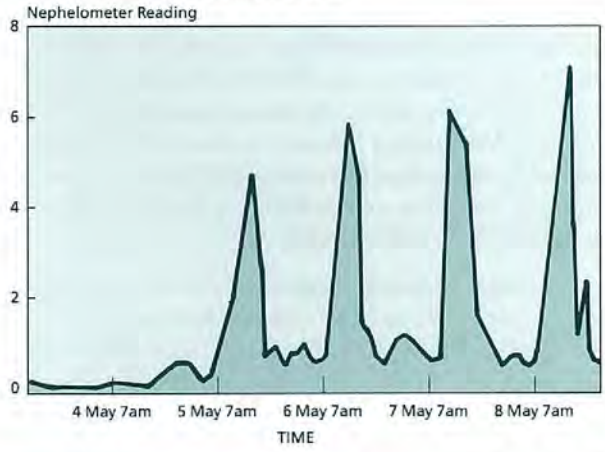


TABLE 1

MEAN NUMBER OF ATTENDANCES AT FIVE SYDNEY HOSPITALS ON DAYS OF HIGH AND LOW PARTICULATE POLLUTION, SYDNEY, MAY 1991

HOSPITAL	HIGH-POLLUTION DAYS†	LOW-POLLUTION DAYS†	p value‡
Prince of Wales	12.6	7.5	0.03*
Sutherland	6.3	4.2	0.02*
Westmead	5.3	5.7	0.81
Liverpool	5.4	4.4	0.37
Mt Druitt	4.7	4.3	0.35
All hospitals	**34.3**	**26.1**	0.001*

† Mean of all pollution or non-pollution days, excluding Sundays. Pollution days were defined as days of high visible air pollution. Nephelometry readings on these days were higher than at any other time in the three-week period
‡ Probability of observed number of cases compared to mean on non-polluted days, after adjustment for the increased number of attendances on Sundays
** $p < 0.05$*

TRAINING FOR A HEALTHIER FUTURE

The NSW Health Department established the NSW Public Health Officer Training Program in 1989 to develop public health professionals for the State's evolving public health network, which has been described in previous issues of the Bulletin[1,2,3]. The program aims to provide practical training with strong emphasis on epidemiology and strategic planning.

Professionals with at least three years' experience in health who have completed the course work for a Master of Public Health degree (or equivalent) are eligible to join the program, which comprises 16 Public Health Officers (PHOs): 11 medical practitioners and five with backgrounds in pharmacy, physiotherapy, anatomy, medical records administration and occupational health and safety. The program seeks participants who are committed to public health practice, have high academic standards and, most importantly, demonstrate enthusiasm.

The program aims to develop PHOs' ability to:

- utilise epidemiologic methods to determine the actual or potential burden of illness affecting a population and the efficacy and effectiveness of prevention and control measures
- identify health priorities for a community and recommend strategies to alleviate problems and promote health
- advocate the improvement of the health of the community to governments and others responsible for resource allocation
- communicate effectively with other health professionals and the public, through public presentations, written reports and scientific articles
- apply management principles to public health practice
- work successfully in multidisciplinary teams

For three years PHOs rotate through placements, ranging from 6 to 24 months. The placements offer opportunities to gain experience in different facets of public health practice and investigation, and have included:

- the Epidemiology and Health Services Evaluation Branch — communicable disease control, environmental health, chronic disease and injury prevention, reproductive health and health services evaluation
- the Department of Public Health, University of Sydney
- Public Health Units in Sydney, Newcastle and Wollongong
- the Centre for Health Economics and Evaluation
- the NSW Central Cancer Registry
- the Sydney Hospital AIDS and STD clinical services

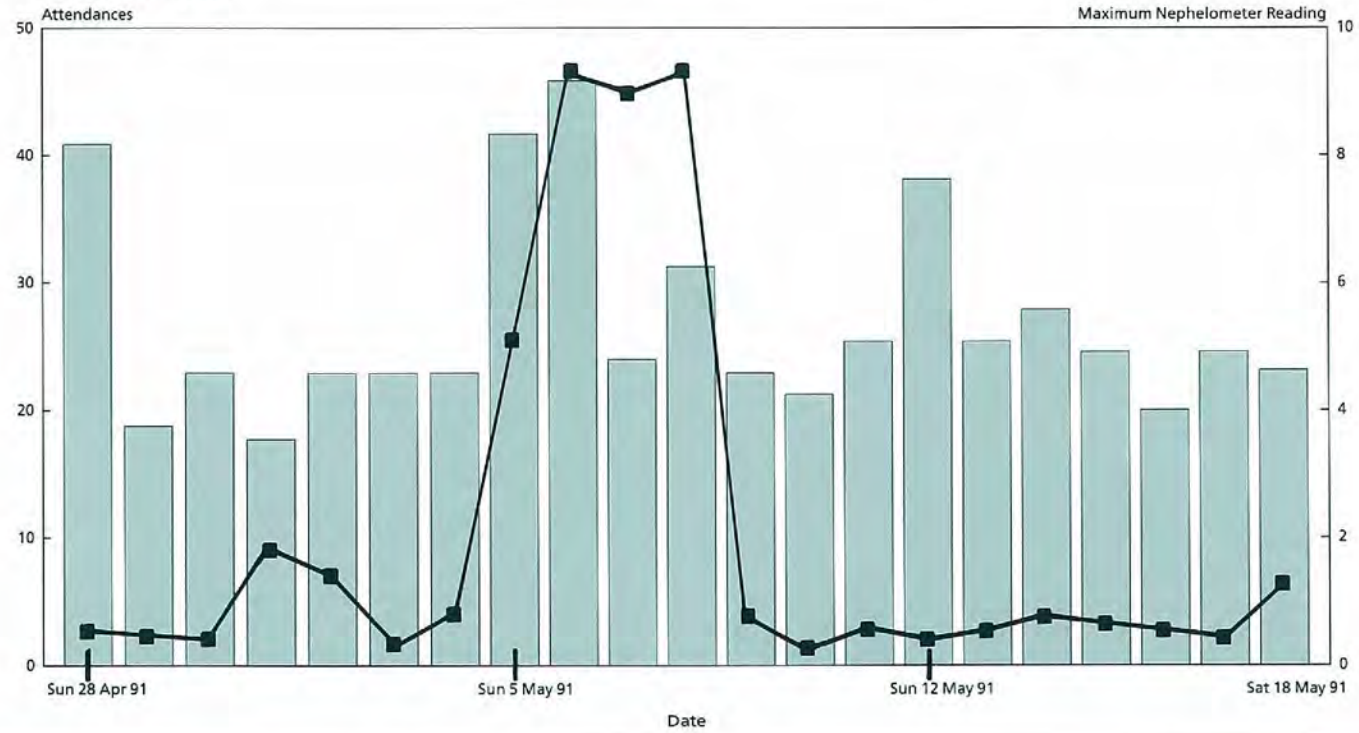
Continued on page 80 ►

Asthma and air pollution in Sydney

► Continued from page 73

FIGURE 2

ASTHMA ATTENDANCES AT FIVE SYDNEY METROPOLITAN HOSPITALS AND LEVELS OF PARTICULATE POLLUTION, APRIL 28-MAY 18, 1991



APPARENT CLUSTERS OF UNEXPLAINED INFANT DEATHS

Five unexplained infant deaths, attributed to Sudden Infant Death Syndrome, occurred in the Coffs Harbour local government area [LGA] between April and July 1990. Subsequently, 18 apparent SIDS deaths between January and October 1990 were noted in the Blacktown LGA. The deaths were investigated by the Epidemiology Branch and the North Coast Region and Western Sector Public Health Units.

Sudden Infant Death Syndrome (SIDS), is defined as the sudden death of an infant or young child which is unexpected by history and in which a thorough post-mortem examination fails to demonstrate an adequate cause of death. The cause of SIDS is unknown, but it appears to be associated with low birthweight, young maternal age, maternal smoking, and low socioeconomic status. The peak incidence is in the second and third months of life, with 80-90 per cent of the total incidence in the first six months of life[1]. SIDS is more common in winter and may occur in clusters[2]. It is more common in males[3]. Recent research has focused on possible relationships with ambient temperature[4,5] and with sleeping position, several studies having suggested SIDS is associated with sleeping in the prone position[6-9].

SIDS is a major cause of infant death in NSW. Excluding congenital anomalies and perinatal conditions, it accounts for about 70 per cent of infant deaths. From 1981 to 1988, the number of deaths attributed to SIDS ranged from 141 (in 1981) to 205 (in 1986). The lowest and highest levels also occurred in these years — the rates ranging from 1.7 to 2.4 per 1000 live births. In general, SIDS was more frequent in winter (June, July and August).

A major difficulty in the investigation of SIDS is the diagnosis. It is strictly a diagnosis of exclusion which often is not clearcut, especially where some pathology is detected at autopsy, and where it is difficult to assess the extent to which this pathology can explain the death. Even potentially lethal conditions may be difficult to detect at infant autopsies.

Although unexplained deaths resembling SIDS were recognised as long ago as 1834[10], Sudden Infant Death Syndrome was first proposed as a diagnostic term in 1969[11], and came into general use only in the late 1970s with the introduction of the 9th Revision of the International Classification of Diseases. Since that time SIDS has tended to displace some other diagnoses that may have been ascribed to unexplained infant deaths.

INVESTIGATION OF UNEXPLAINED INFANT DEATHS

Unexpected infant deaths in NSW are reported to the coroner. A police officer investigates the death scene and compiles a report which may include accounts of interviews with the parents and other witnesses. The coroner invariably orders an autopsy. In the Sydney metropolitan area autopsies are done at the Institute of Forensic Medicine (at the Coroner's Court in Glebe) or the Department of Forensic Pathology, Institute of Clinical Pathology and Medical Research (at Westmead Hospital). Elsewhere in the State the autopsies are done by Government Medical Officers. In most districts the GMO is a general practitioner with basic training in autopsy techniques, but in some areas forensic autopsies are done by local pathologists. The histopathology from country cases is referred to the Institute of Forensic Medicine for reporting and toxicology is done by the Division of Analytical Laboratories.

The Blacktown and Coffs Harbour investigations set out to answer the following questions: (i) Was the occurrence of SIDS in excess of expectation? (ii) What were the characteristics of the cases, and did they have any common features that might be amenable to prevention?

For both investigations 1981-87 NSW mortality data were used to establish the incidence of SIDS (both Statewide and in the Coffs Harbour and Blacktown LGAs) and to estimate the expected frequency. Efforts were made to ascertain all infant deaths attributed to SIDS during the index periods, and the observed numbers of deaths were compared with the expectations. Individual cases were reviewed in detail, including an inspection of coroners' records and extraction of data from medical records.

The Coffs Harbour investigations included interviews with the infants' parents and a review of environmental factors, particularly the use of agricultural chemicals on banana plantations and their levels in potable water. Respiratory disease admissions and infant attendances at the Coffs Harbour District Hospital's accident and emergency unit for apnoea, gastro-oesophageal reflux or "acute life-threatening events" (also known as "near-miss SIDS") also were reviewed.

FINDINGS — COFFS HARBOUR

A total of 10 deaths of Coffs Harbour LGA residents were attributed to SIDS from 1981 to 1987. No SIDS deaths were recorded in the years 1981-83, and four were recorded in 1986, paralleling the high Statewide level in that year. The annual incidence of SIDS in the Coffs Harbour LGA ranged from zero to 6.0 cases per 1000 live births. One SIDS death was recorded in 1988 and none in 1989.

Based on the 1987 NSW incidence of 2.0 per 1000 live births, 1.3 cases of SIDS are expected to occur in the Coffs Harbour LGA over a calendar year. Five deaths were attributed to SIDS between April and July 1990. Compared with the 1987 Statewide incidence, the probability of five or more cases occurring by chance in the Coffs Harbour LGA over a calendar year was less than 1 per cent.

The diagnosis of SIDS was substantiated in three of the five cases and SIDS was considered to be the most likely cause of death in one of the other two cases. In the remaining case there was autopsy evidence of pulmonary infection. The deaths occurred at widely dispersed sites and the histories of the individual cases did not suggest any common or preventable factors.

There was no toxicological evidence that any of the agricultural chemicals — which comprised fungicides, organophosphorous compounds, paraquat and an organic fertiliser — could cause SIDS, and it was

impossible to suggest a plausible biological mechanism to implicate these substances. While the Coffs Harbour Shire Council regularly sampled the reticulated water supply for testing by the Health Department's Pesticide Residue Laboratory in Sydney, the routine testing program included only one of the compounds used on the banana plantations, namely chlorpyrifos. Chlorpyrifos, an organophosphorous compound, had not been detected in the water, but the sampling was not synchronised with pesticide spraying.

The review of respiratory admissions and accident and emergency presentations to the Coffs Harbour District Hospital was largely non-contributory.

FINDINGS — BLACKTOWN

All unexpected infant deaths in the Blacktown area are referred to the Department of Forensic Pathology at Westmead. The Department's records for January to October 1990 were therefore assumed to include all the relevant deaths from the Blacktown area. Eighteen deaths were attributed to SIDS during this period, representing an incidence 1.57 times that of the remainder of the State from 1983 to 1987. Compared with the 1983-87 Statewide incidence, the probability of 18 or more cases occurring by chance in the Blacktown LGA over a calendar year was less than 1 per cent.

Data are incomplete from 1988 onwards. Provisional data from the Department of Forensic Pathology show that the 1988-90 incidence of deaths attributed to SIDS in the Blacktown LGA ranged from 4.6 to 5.8 deaths per 1000 live births. If the SIDS incidence in NSW remained constant at the 1988 rate of 2.2 per 1000 live births over this period, the incidence of SIDS in the Blacktown LGA would be at least double that of the State as a whole.

The 18 cases comprised 11 male and seven female infants of whom seventeen were less than six months old. Nine had had an upper respiratory tract infection in the week before death. Thirteen cases occurred in the cooler months (April to August). Half the mothers were aged 21 years or less, and 16 had had at least one previous pregnancy.

Again, there were no features to suggest a common and preventable cause. Seven of the infants had evidence of respiratory tract infection at autopsy, although two of these had no documented respiratory symptoms before death. A respiratory virus was isolated at autopsy from three of the seven. There were no remarkable features to account for the deaths or to link the cases.

LESSONS AND SUBSEQUENT ACTION

Two lessons emerged from the investigations. the first was the lack of any mechanism for obtaining timely surveillance data on the occurrence of unexpected infant deaths. While the apparent clusters in the Coffs Harbour and Blacktown LGAs were brought to the attention of the Epidemiology Branch and the local Public Health Units, contemporary information on the Statewide occurrence of such deaths was unavailable, and therefore the apparent clusters could be evaluated only in the context of previous years' data on SIDS. The second lesson was the variation in autopsy procedures, with the resultant likelihood of variation in the use of SIDS as a diagnostic label.

Two major initiatives have followed from the investigations. First, the (then) Minister for Health, Mr Peter Collins, announced that a Statewide surveillance program of unexpected infant deaths would be set up by the Health Department. The NSW Sudden Infant Death Syndrome Advisory Committee, chaired by the State Coroner, Mr Kevin Waller, has been re-convened with a primary objective of advising on and facilitating the establishment of the surveillance program. Central to the program is the provision of an expert autopsy service for all infant deaths reported to the coroner, irrespective of where they occur.

Second, in conjunction with other agencies, the North Coast Region Public Health Unit has undertaken a review of agricultural chemical use and monitoring in the Region, and the Coffs Harbour Shire Council's reticulated water-testing program has been synchronised with the pesticide spraying cycle.

There have been further developments nationally. In April 1991 a workshop jointly sponsored by the National SIDS Council, the Royal College of Pathologists of Australia and the Victorian Forensic Pathology Institute led to the design of a national SIDS autopsy protocol, with a first draft due to be presented in October. In May, Tasmanian data supporting Dutch, British and New Zealand findings on the relationship between SIDS and the prone sleeping position was published.

Mass-media programs in New Zealand, which has the highest known SIDS incidence in the world, have directed attention to sleeping position, breastfeeding and maternal non-smoking; contemporaneous with these programs the SIDS incidence has dropped appreciably, suggesting a possible preventive effect. Following from these developments, the Menzies Foundation convened a meeting of experts in July to decide on policy recommendations about infants' sleeping positions.

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DRAMATIC INCREASE IN HEPATITIS A

Sydney has been hit by an outbreak of hepatitis A. More than 400 cases have been reported to the NSW Health Department this year, compared with only 15 at the same time last year. The cases are concentrated in the inner eastern and central suburbs of Sydney and young homosexual men are the main group affected. The outbreak appears to be part of an international pattern: there are outbreaks among homosexual men in Melbourne[1], London, Dallas, Denver, New York, Rhode Island and Toronto[2].

BACKGROUND

The epidemiology of hepatitis A infection is highly variable and is constantly changing. Clinical hepatitis A infection is largely determined by two factors: age and the prevalence of antibodies to hepatitis A virus (anti-HAV) which indicate the level of immunity. Young children infected with hepatitis A have either no symptons at all or have a non-specific illness which is not recognisable as hepatitis. But in adults clinical symptons occur in the great majority of cases. The prevalence of anti-HAV also influences the clinical attack rate: the greater the prevalence of antibody in the community, the lower the incidence of clinical hepatitis A infection.

Gust and Feinstone have postulated three epidemiological patterns of hepatitis A infection which account for the observed differences in clinical attack rates around the world[3]:

PATTERN A occurs in conditions of overcrowding, lack of a clean water supply, inadequate sewerage and poor hygiene. In these circumstances, hepatitis A infection is usually acquired in early childhood and is usually subclinical. By the age of 10, most children are immune so clinical hepatitis A is uncommon.

PATTERN B is found where sanitation and general living conditions are improving as a result of social and/or economic reforms. In this situation the childhood infection is declining but so too is the level of immunity. The result of this combination is that clinical attack rates *increase* because there are more susceptible adolescents and adults. This paradoxical pattern occurs in countries like the USSR and China.

PATTERN C is seen in developed countries. Hepatitis A infection is very uncommon in young children and the majority of adolescents and young adults are not immune. Most cases occur as a result of travel to endemic areas or as part of an outbreak. The latter occur infrequently. Community-wide outbreaks of hepatitis A tend to occur in a 7- to 12-year epidemic cycle with low levels of infection in the inter-epidemic period [2,3,4].

In the 1970s and 1980s, hepatitis A became relatively uncommon. A seroprevalence survey in Sydney in the early 1970s[4] showed that prevalence of anti-HAV increased with age and that after the age of 40, more than 90 per cent of people were immune. If a seroprevalence survey were repeated today, levels of immunity would almost certainly have fallen. A recent survey of British blood donors[5] confirmed the falling immunity levels in developed countries: the overall prevalence of anti-HAV in blood donors under the age of 45 was less than 20 per cent.

The conclusion to be drawn is that the pool of people in Australia susceptible to hepatitis A is probably very large, as is the potential for hepatitis A epidemics.

DETAILS OF THE OUTBREAK

In February this year the Eastern Sydney Public Health Unit was notified of nine cases of hepatitis A in young men. Normally the unit would expect one or two notifications a month. The only apparent link between the people was that they were homosexual.

Over the next few months, notifications steadily increased to between 25 and 30 a month. Figure 3 shows the distribution of cases by month of onset of illness for NSW. The notification rate is sustained over a period of five months, and a pattern is typical of community-wide outbreaks[6]. By comparison, common source outbreaks are characterised by an explosive rise in case numbers over a one- or two-month period followed by an equally dramatic fall in case numbers.

Notification rates per 100,000 population for 1990 and 1991 by Area and Region are shown in Figure 4. Clearly, there is a dramatic increase in the number of notifications in 1991, but this is largely confined to the Eastern, Central and Southern Sydney Areas. Eastern Sydney has by far the highest attack rate of any Area or Region.

The age and sex distribution of cases (shown in Figure 5) is unusual in that the male-to-female ratio is almost three to one. This can be explained by the fact that more than 60 per cent of cases from the Eastern Sydney Area are male homosexuals (see Figure 6).

Figure 7 compares the cases distribution in male homosexuals with the remaining cases in the Eastern Sydney Area. The "first wave" of the epidemic in homosexual men clearly precedes the "second epidemic wave" through the heterosexual community. About 27 per cent of the homosexual group are known to be HIV antibody positive.

In terms of other risk factors for hepatitis A infection, 21.1 per cent of cases gave a history of recent contact with a confirmed or suspected hepatitis A case and only 8.2 per cent had recently travelled overseas. Most of the cases were in young single adults. In Eastern Sydney, only a very few cases had or have contact with children.

Hepatitis A notifications are continuing to come in and the epidemic is showing no sign of abating.

DISCUSSION

The hepatitis A outbreak, although community based, is largely confined to young male homosexuals in the Eastern Sydney Area. Why this is occurring is not clear. Certainly, hepatitis A infection has long been recognised as being relatively common in male homosexuals[7]. Corey and Holmes[8] studied the incidence and prevalence of homosexual and heterosexual men with hepatitis A attending an STD clinic. They found the prevalence of anti-HAV almost three times greater in the homosexual group and that the incidence of hepatitis A in susceptible homosexuals was 22 per cent while none of the susceptible heterosexual men seroconverted during the study. They also found a correlation between frequent oral-anal sexual contact and incidence of hepatitis A infection. In this AIDS era, oral-anal contact may be a common sexual practice because it is regarded as relatively safe.

Another unresolved issue is how concurrent HIV infection affects the course of hepatitis A infection. Both the clinical course and the infectious period may be prolonged, a finding which would influence the course of the epidemic.

Community-wide outbreaks of hepatitis A are notoriously difficult to control[6,9,10]. By the time cases are detected and diagnosed, contacts have usually already been exposed for more than a week and often more than two weeks. Post-exposure prophylaxis with normal immunoglobulin is effective only if given within 14 days of exposure. For this reason the focus of activities during this epidemic has been to communicate the risk to medical practitioners and the general community and to encourage early diagnosis and intervention. Warning letters about the prevention and control of hepatitis A have been sent out to general practitioners and hospital accident and emergency centres in the affected areas, the Health Department issued a hepatitis A warning in both the gay and general press and educational posters have been distributed.

Department staff have also met with staff of the AIDS Bureau and the Aids Council of NSW to discuss strategies for delivering appropriate hepatitis A health messages to the target communities.

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Dramatic increase in Hepatitis A

► Continued from page 77

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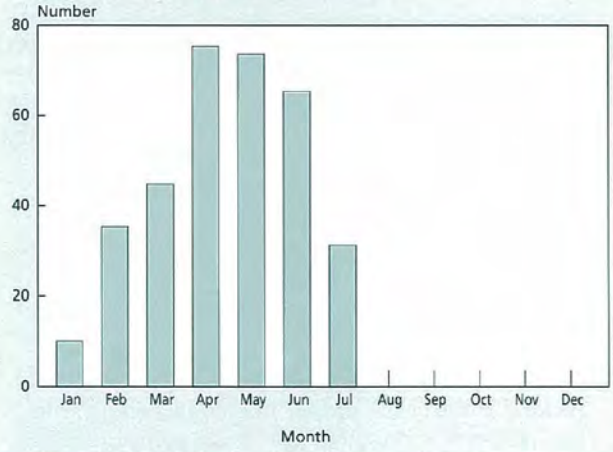
FIGURE 3

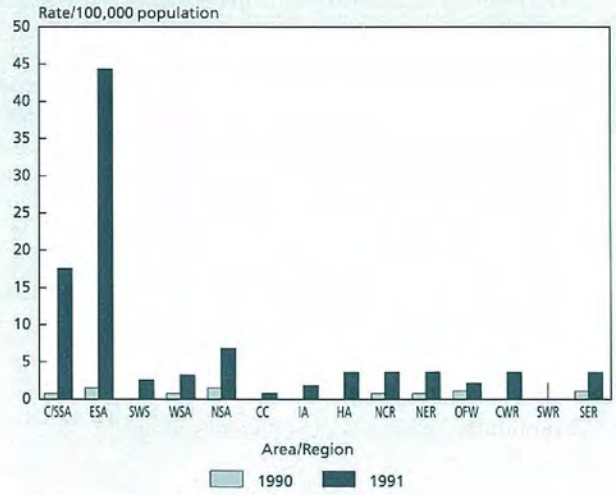
Hepatitis A notifications by month of onset, NSW 1991



Source: PHUs (as at 22/7/91)

FIGURE 4

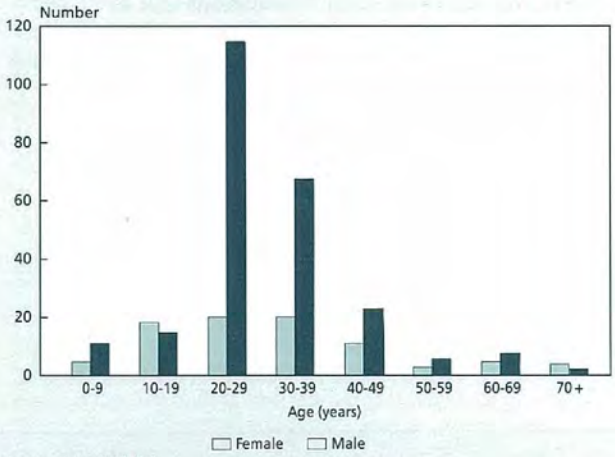
Hepatitis A notification rate Area/Region, NSW January to July 1990/1991



No. cases: 1990 — 15; 1991 — 342

FIGURE 5

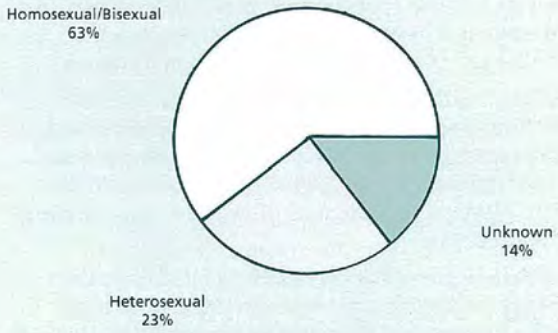
Hepatitis A cases age/sex dist, NSW January to July 1991



Source: PHUs (22/7/91)

FIGURE 6

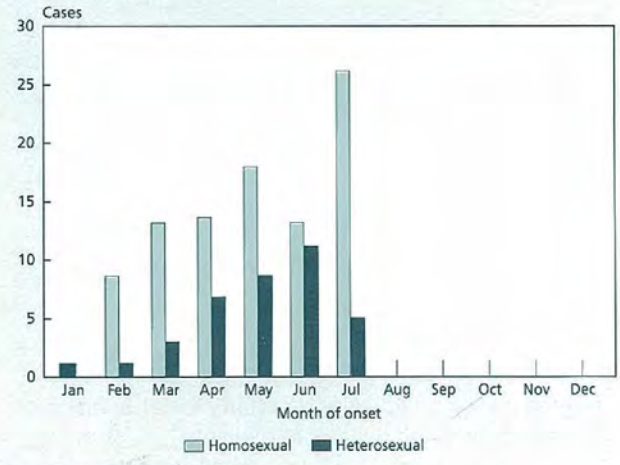
Hepatitis A cases by sexual preference, Eastern Sydney



Source: Eastern Sydney PHU (1/8/91)

FIGURE 7

Hepatitis A cases Epicurve by sexual preference



Source: Eastern Sydney PHU (1/8/91)

INVESTIGATION OF A TYPHOID NOTIFICATION

A case of typhoid fever was notified to the Public Health Unit of the Northern Sydney Area Health Service on March 18, 1991. The patient was an 11-month-old boy in the children's ward of Hornsby Ku-ring-gai Hospital. He had been in India from October 1990 and had not had any illness while there. His mother was first ill on February 13, 1991 and was admitted to hospital in India. She was diagnosed as having typhoid fever and treated with intravenous antibiotics. Mother and child returned to Australia on March 4.

On March 7 the boy became febrile and saw his GP, who prescribed Amoxicillin and Paracetamol. By March 9 he was still febrile and was prescribed Trimethoprim/ Sulphamethoxazole in place of Amoxicillin. On March 10 he had diarrhoea three times after his second dose of Trimethoprim/Sulphamethoxizole, and still had a high fever. The GP advised referral to Accident and Emergency at the Hornsby Ku-ring-gai Hospital. He was not febrile while there and was sent home on Amoxicillin after a diagnosis of Otitis Media. On March 12 he was still febrile and the GP arranged for his admission to hospital.

He was intermittently febrile and drowsy after admission, but at other times was cheerful and playing normally. His bowels were normal, he was eating and drinking normally and he had no cough. There were no spots on the trunk and there was no bradycardia. There was no splenomegaly and no lymphadenopathy.

The baby's white cell count was 14.8, AST was 70 (0-41) & ALT 81 (0-65). Stool and blood cultures were taken. Giardia was seen in his stool on March 13 and he was

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IDENTIFICATION
Typhoid fever is a systemic bacterial disease characterised by insidious onset of sustained fever, headache, malaise, anorexia, a relative bradycardia, splenomegaly, rose spots on the trunk, non-productive cough, constipation more commonly than diarrhoea (in adults) and involvement of the lymphoid tissues.

The usual fatality rate of 10 per cent can be reduced to less than 1 per cent with prompt antibiotic therapy. Relapses occur in 5-10 per cent of untreated cases and may be more common (15-20 per cent) after antibiotic therapy. Mild and unapparent illnesses occur, especially in endemic areas.

The aetiological organisms can be isolated from the blood early in the disease and from urine and faeces after the first week. A four-fold rise in agglutination titre in paired sera appears during the second week in fewer than 70 per cent of cases of typhoid fever. When it occurs it supports the diagnosis, provided vaccine had not been given recently. Because of its limited sensitivity, serology is of little diagnostic value.

INFECTIOUS AGENT
Salmonella typhi, the typhoid bacillus.
Presently 106 types can be distinguished by phage typing, which is of value in epidemiological studies.

OCCURRENCE
Worldwide. Strains resistant to recommended antibiotics have appeared in several areas of the world. Multi-resistant strains have been reported from Asia, the Middle East and Latin America.

RESERVOIR
Man. Family contacts may be transient carriers. The chronic carrier state is most common among persons infected during middle age, especially females; carriers frequently have gall bladder pathology.

MODE OF TRANSMISSION
By food and water contaminated by faeces and urine of patients and carriers. Important vehicles in some parts of the world include shellfish taken from sewage-contaminated beds, raw fruit, vegetables contaminated by nightsoil, contaminated milk and milk products (usually by hands of carriers) and missed cases.

INCUBATION PERIOD
The incubation period depends on the size of the infecting dose; usual range is one to three weeks.

PERIOD OF COMMUNICABILITY
As long as the bacilli appear in excreta, usually from the first week throughout the convalescence; variable thereafter. About 10 per cent of untreated typhoid fever patients will discharge bacilli for three months after onset of symptoms, and 2-5 per cent become permanent carriers.

SUSCEPTIBILITY
Susceptibility is general and is increased in individuals with gastric achlorhydria. Relative specific immunity follows recovery from clinical disease, unapparent infection and active immunisation, but is inadequate to protect against ingestion of large numbers of organisms.

PREVENTATIVE MEASURES
1. Educate the public about hand-washing.
2. Dispose of human faeces in a sanitary manner.
3. Protect, purify and chlorinate public water supplies.
4. Use scrupulous cleanliness in food preparation and handling
5. Pasteurise or boil all milk.
6. Exclude carriers from handling food and from providing patient care.
7. Immunise before entering endemic areas.

started on Metronidazole. By March 15 he was still febrile, and so remained in hospital.

On March 16 the blood culture grew salmonella typhi which was resistant to Cotrimoxazole, Ampicillin, Choramphenicol and Tetracycline, and sensitive to Cefotaxime. Typhoid serology was Poly O (weakly positive) and Vi (strongly positive). The laboratory Microbiologist was consulted and intravenous Cefotaxime was commenced.

By March 18 the boy was afebrile and has remained well since. The Public Health Unit was notified that day. On March 22 the Cefotaxime was ceased and the baby was discharged on oral Amoxicillin/Clavulanic Acid for one week.

PUBLIC HEALTH INTERVENTION

On the day it was notified, the Northern Sydney Area Health Service PHU discussed the case with the Microbiologist, the Health Department Infectious Diseases Epidemiologist, the General Practitioner and the Hospital Infection Control Nurse Consultant. PHU staff took a full history from the patient's mother. After consultation with the GP, infection control measures — such as strict hand-washing procedures and the mother not preparing any food or drink as she had recently had typhoid fever — were instituted at their home.

The only contacts in Australia were the grandmother, grandfather, uncle and mother. They were not involved with public food preparation or handling. Stool culture included all family members. Three stool samples on three different days were taken for culture looking for typhoid. All the stool samples were negative.

In follow-up all the family, including the patient, remained well. There have been no further cases of typhoid fever in the Northern Sydney Area.

DISCUSSION

This is a case of typhoid fever in an unimmunised 11-month-old child, acquired in India and diagnosed and treated in Australia soon after his return. As he had acquired the disease overseas, the Public Health investigation was limited to the immediate family.

History provided by the mother indicated that home conditions in India were good, and included running water, sewerage and refrigeration. Hypothetical sources of the typhoid could have included:

- water drunk on the train to New Delhi on the journey home
- water or food from visits near the home near Bombay
- transfer of the disease from the mother

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Benenson A et al. Fifteenth Edition 1990, Control of Communicable Diseases in Man, American Public Health Association, Washington.

Training for a healthier future

► *Continued from page 74*

Placements have also been offered at the National Health and Medical Research Council's Clinical Trials Centre, the Centre for Clinical Epidemiology and Biostatistics at the University of Newcastle, the Communicable Disease Section of the Commonwealth Department of Community Services and Health, the National Centre in HIV Epidemiology and Clinical Research and the Australian Institute of Health.

PHOs undertake, under supervision, day-to-day duties of the unit to which they are attached. At the beginning of each assignment PHOs and supervisors outline a plan of projects to be completed during the attachment.

Monthly seminars in the areas of reproductive and child health, infectious disease, chronic disease and injury prevention, environmental health and health services evaluation are held at the Public Health Division in North Sydney. PHOs are encouraged to participate in short courses on epidemiological methods, health economics, management and computer skills.

The program provides the practical experience required to join the Australian Faculty of Public Health Medicine. For non-medical PHOs it will aim to provide training for accreditation by relevant professional bodies as these develop.

Overseas training opportunities are being established with the US Centers for Disease Control (Atlanta) and the British Communicable Disease Surveillance Centre (Colindale). One PHO now participates in the US Centers for Disease Control's Epidemic Intelligence Service.

Several key experiences have been identified for PHOs to complete to meet the objectives of the program. They are:

- investigation of at least two disease clusters/outbreaks
- substantial involvement in the establishment or evaluation of a surveillance system
- evaluation of the burden of one or more illnesses or injury types in a specific community
- substantial involvement in planning, implementation or evaluation of a public health program
- research and report on a public health problem, such as assessment of an environmental hazard
- publication of at least one scientific article in a refereed journal and at least three articles in the Public Health Bulletin
- presentation of a paper/poster at a scientific meeting and at least one paper/poster at the annual Public Health Officer Program Scientific Conference

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2. Rubin G, Frommer M, Morey S, Leeder S. On the right track. *NSW Public Health Bulletin*, 1990; 2: 1-2.
3. Ward M, King L, Rubin G, Stewart G. Setting a new agenda. *NSW Public Health Bulletin*, 1990; 2: 5-6.

INFECTIOUS DISEASES

The process of implementation of the Public Health Act (1991) continues. The list of medical practitioner notifiable conditions has been amended further with the addition of "Adverse Immunisation Events". This has been decided on to improve our understanding of the benefits and risks of immunisation.

The Response Protocols have been largely rewritten by staff of the Infectious Diseases Section and a further round of consultation with experts in infectious diseases. Now called the *Infectious Diseases Manual*, the document has been submitted to Parliamentary Council for incorporation into the Regulation of the Public Health Act.

Public Health Units have undergone further training with the Infectious Diseases Database System. Eleven of the 14 (79 per cent) Public Health Units contributed data to the database in July — a marked improvement on the June data, which came from only three (21 per cent) Units.

The hepatitis A outbreak is continuing in NSW. By July 22, 342 hepatitis A notifications were received by PHUs. (On August 5, 312 notifications were recorded on the database. Hepatitis A notification rates range from 40.5/100,000 population in the Eastern Sydney Area, 17.4/100,000 population in the Central and Southern Sydney Area, 8.54/100,000 population in the Northern Sydney Area and fewer than 5/100,000 in other Areas and Regions. Of 313 cases with recorded age and sex data, 80.1 per cent were male (male:female ratio 2.9:1) and 36.7 per cent were males in the 20- to 29-year age group.

The case of cholera reported in the July *Public Health Bulletin* was due to *vibrio cholerae* non type 01 and has been denotified. We now report the second case of *vibrio cholerae* non type 01 for 1991. A 41-year-old woman returned from Laos with a diarrhoeal and wasting illness. Stool cultures were positive for *vibrio cholerae* non type 01. The woman was successfully treated with tetracyclines. Tracing the patient was only possible due to cooperation between the local medical practitioner, local council staff, the New England Public Health Unit and the office of the Chief Health Officer. Environmental samples were negative for vibrio.

The Communicable Diseases Intelligence has notified Epidemiology Branch of an increase in isolation of Echovirus 17, primarily from cerebrospinal fluid. This enterovirus causes aseptic meningitis and encephalitis. The previous outbreak of this virus was in 1984-1985. Although there is no public health response in relation to this organism, clinicians are advised to expect an increase in meningitis. Care must be taken to exclude a septic cause for meningeal symptoms (see below).

A cluster of seven cases of meningococcal meningitis have been reported to the South Western Sydney Public Health Unit since July 17, 1991. Five of these were in Campbelltown. No identifiable contact could be found between any of the cases, although two cases attend the same school. All five Campbelltown cases were group C. The remaining two were untypeable.

Meningococcal vaccination will be offered to all children of the school, their siblings aged two to five years and children attending an adjoining preschool.

TABLE 2

INFECTIOUS DISEASE NOTIFICATIONS, NSW
Notifications to the end of July, 1991

CONDITION	Number of Cases Notified			
	Period		Cumulative	
	July 1990	July 1991	July 1990	July 1991
AIDS	17	N/A	*180	*124
Arboviral infection (NOS)	1	5	2	383
Brucellosis	–	–	5	3
Cholera	–	–	–	–
Diphtheria	–	–	–	–
Foodborne illness (NOS)	279	52	1493	1852
Gastroenteritis (instit.)	N/A	4	N/A	28
Gonorrhoea	42	12	243	211
H influenzae infection (NOS)	–	10	10	81
H influenzae B – meningitis	–	–	10	–
H influenzae B – septicaemia	–	–	2	1
Hepatitis (NOS)	–	51	2	364
Hepatitis A	3	9	17	312
Hepatitis B – acute	–	–	6	3
Hepatitis B – carrier	–	1	–	8
Hepatitis B – unspecified	39	17	235	392
Hepatitis C	2	1	17	71
HIV infection	N/A	N/A	*273	*408
Hydatid disease	–	–	2	–
Legionnaires' disease	–	1	18	17
Leprosy	–	–	–	–
Leptospirosis	4	1	26	24
Listeriosis	N/A	1	N/A	1
Malaria	18	2	95	23
Measles	12	5	51	106
Meningococcal infection (NOS)	6	2	22	25
Meningococcal meningitis	–	1	17	9
Meningococcal septicaemia	–	–	5	5
Mumps	N/A	–	N/A	5
Mycobacterial infection (NOS)	54	5	313	101
Mycobacterial tuberculosis	–	–	–	9
Mycobacterial – atypical	–	1	14	12
Pertussis	3	–	103	31
Plague	–	–	–	–
Poliomyelitis	–	–	–	–
Q fever	22	6	77	132
Ross River fever	11	–	230	115
Rubella	N/A	5	N/A	24
Salmonella infection (NOS)	95	19	913	605
Syphilis	37	21	204	233
Tetanus	–	–	–	2
Typhoid & paratyphoid	3	–	21	15
Typhus	–	–	–	–
Viral haemorrhagic fevers	–	–	–	–
Yellow fever	–	–	–	–

* Data January-June only

Continued on page 82 ►

► *Continued from page 81*

Confirmed new HIV positive test results can now be reported for all three NSW HIV Reference Laboratories (Prince of Wales, St Vincent's and Westmead hospitals) and Royal Prince Alfred Hospital. Duplicate tests on individuals have been excluded to the best ability of each laboratory. Further manual matching and record linkage within St Vincent's laboratory may result in minor adjustments of future cumulative report rates. Inter-laboratory repeats cannot be excluded, but people self-reporting a previous positive result (anywhere) are excluded. While some duplicate tests will be included, the number of reported cases is still fewer than the total number of people with HIV in NSW, as an unknown number remain untested and have asymptomatic infection.

In this issue of the Public Health Bulletin both age and exposure category breakdowns by sex are presented for cumulative HIV data to the end of June 1991. It is important to note the large number of tests of unknown gender, age or exposure category, due to limitations in data collection, especially during the early years of the HIV epidemic. Data collection has been improved and call-back mechanisms exist in each laboratory to obtain missing data from the test-requester. However time and resource constraints still limit the amount of data that can be obtained by call-back and variables such as postcode or residence and exposure category remain unknown for a substantial number of cases. It is thus important to view the figures conservatively as the measurement error may distort the relative proportions of cases in any one group.

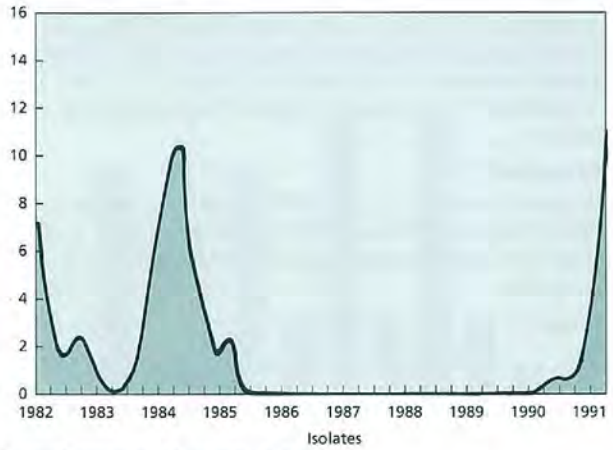
Several meetings with representatives from the Reference Laboratories, Health Department and National Centre in HIV Epidemiology & Clinical Research have been held to discuss issues including call-back mechanisms, standardising of exposure categories and piloting of a proposed national study of heterosexually acquired HIV transmission. The study will try to determine the validity of various reported exposures.

Of the 22 infants under one year of age with HIV infection, eight were reported as having been exposed via maternal infection with HIV; one transfusion was reported and 13 had an unknown exposure. The latter are likely to be further vertical exposure cases or infants with haemophilia who received infected blood components before routine screening of all blood products in May 1985. The majority of cases remain as young, homosexual males, with relatively few being reported as exclusively heterosexual transmission (n=166) or exposure via sharing of drug injecting equipment (n=196). The number of cases tails off with increasing age.

Continued on page 84 ►

TABLE 3

ECHOVIRUS 17 — NSW
JANUARY 1982-JUNE 1991



Source: Communicable Diseases Intelligence

TABLE 4

NSW HIV +VE TESTS
EXCLUDING PREVIOUS +VES
DATA = NSWHIV.SSD,
ALL TO JUNE 30, 1991

GENDER	Frequency	Cumulative Frequency
F	394	394
M	7728	8122
T	2	8124
U	1960	10084

TABLE OF AGEGRP BY GENDER

AGEGRP (Yrs) Frequency	GENDER F	M	U*	Total
<1	3	18	1	22
1-4	2	1	1	4
5-14	3	31	1	35
15-24	74	1023	34	1131
25-34	112	2543	99	2754
35-44	45	1744	58	1847
45-54	15	525	14	554
55-64	15	131	2	148
65 & over	8	35	0	43
Unknown	117	1677	1752	3546
Total	394	7728	1962	10084

*includes two transsexual cases

TABLE OF RISK BY GENDER

RISK Frequency	GENDER F	M	U*	Total
Homosexual/bisexual	15	3714	128	3857
Heterosexual	52	110	4	166
Drug injector	40	141	15	196
Transfusion	35	45	1	81
Haemophilia	0	60	0	60
Homosexual/bisexual + IDU	2	70	4	76
Heterosexual + IDU	16	14	0	30
Homosexual + transfusion	0	2	0	2
Transfusion + IDU	1	1	0	2
Vertical	6	7	4	17
Specified N.E.C.	9	33	17	59
Unknown	218	3531	1789	5538
Total	394	7728	1962	10084

*includes two transsexual cases

TABLE 5

INFECTIOUS DISEASE NOTIFICATIONS, BY HEALTH AREA & REGION
For July, 1991

CONDITION	CSA	SSA	ESA	WSA	WEN	NSA	CCA	ILL	NER	OFR	CWR	SWR	SER	OTH	TOTAL
Arboviral infection (NOS)	–	–	–	–	–	1	–	–	–	2	–	2	–	–	5
Foodborne illness (NOS)	2	10	–	17	5	–	–	–	10	8	–	–	–	–	52
Gastroenteritis (instit.)	–	–	–	–	–	–	–	–	2	2	–	–	–	–	4
Gonorrhoea	–	–	–	4	–	–	–	–	1	6	–	1	–	–	12
Haemophilus influenzae	–	2	–	–	–	–	–	–	–	–	–	–	–	–	2
Haemophilus influenzae infection (NOS)	–	–	–	1	–	3	–	–	–	2	2	–	–	–	8
Hepatitis (Acute Viral NOS)	–	–	–	17	2	30	–	–	–	2	–	–	–	–	51
Hepatitis A	4	–	–	3	–	–	–	–	–	1	–	1	–	–	9
Hepatitis B — Carrier	1	–	–	–	–	–	–	–	–	–	–	–	–	–	1
Hepatitis B — Unspecified	1	1	–	4	1	–	–	–	4	2	–	–	4	–	17
Hepatitis C	1	–	–	–	–	–	–	–	–	–	–	–	–	–	1
Legionnaires' disease	–	–	–	–	–	1	–	–	–	–	–	–	–	–	1
Leptospirosis	–	–	–	–	–	–	–	–	–	1	–	–	–	–	1
Listeria	–	1	–	–	–	–	–	–	–	–	–	–	–	–	1
Malaria	–	1	–	1	–	–	–	–	–	–	–	–	–	–	2
Measles	–	1	–	–	1	1	–	–	–	1	–	–	1	–	5
Meningococcal inf. (NOS)	–	–	–	–	–	–	–	1	–	–	–	1	–	–	2
Meningococcal meningitis	–	1	–	–	–	–	–	–	–	–	–	–	–	–	1
Mycobacterial atypical	–	1	–	–	–	–	–	–	–	–	–	–	–	–	1
Mycobacterial infection (NOS)	–	–	–	–	1	1	1	–	1	–	–	–	1	–	5
Q Fever	–	–	–	–	–	–	–	–	4	2	–	–	–	–	6
Rubella	–	–	–	2	–	2	–	–	–	–	–	1	–	–	5
Salmonella infection (NOS)	1	2	–	1	4	8	–	–	1	–	–	1	1	–	19
Syphilis	2	1	1	2	1	–	–	–	–	12	–	1	–	1	21

TABLE 6

INFECTIOUS DISEASE NOTIFICATIONS, BY HEALTH AREA & REGION
For period January 1 to July 31, 1991

CONDITION	CSA	SSA	ESA	SWS	WSA	WEN	NSA	CCA	ILL	HUN	NCR	NER	OFR	CWR	SWR	SER	OTH	U/K	TOTAL
AIDS*	19	3	50	3	12	5	13	5	3	4	3	–	–	–	–	–	–	4	124
Arboviral infection (NOS)	–	–	14	–	–	–	1	–	–	6	–	137	189	4	30	2	–	–	383
Brucellosis	–	–	3	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	3
Foodborne illness (NOS)	63	80	846	40	143	104	43	8	13	53	185	88	86	24	57	2	17	–	1852
Gastroenteritis (instit.)	–	–	–	4	9	6	–	–	–	–	–	7	2	–	–	–	–	–	28
Gonorrhoea	4	3	116	18	12	–	1	–	5	3	6	2	36	–	5	–	–	–	211
Haemophilus influenzae type B	1	3	–	–	3	1	2	–	2	–	–	1	–	–	2	–	–	–	15
Haemophilus influenzae infection (NOS)	–	–	15	–	9	10	5	2	4	4	–	–	5	2	7	3	–	–	66
Hepatitis influenzae septicaemia	–	1	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1
Hepatitis (Acute Viral NOS)	2	–	–	19	192	11	69	1	8	20	–	1	26	–	7	8	–	–	364
Hepatitis A	20	9	244	4	7	–	7	–	1	4	3	4	4	–	1	3	1	–	312
Hepatitis B — Acute	1	–	–	–	–	–	–	–	–	–	–	–	–	–	–	2	–	–	3
Hepatitis B — Carrier	5	1	1	–	1	–	–	–	–	–	–	–	–	–	–	–	–	–	8
Hepatitis B — Unspecified	66	26	147	13	10	4	13	–	2	9	17	27	44	–	–	12	2	–	392
Hepatitis C	39	10	–	1	3	–	–	–	–	–	8	8	–	–	1	–	1	–	71
HIV infection*	32	6	95	11	20	5	21	2	2	12	11	–	2	35	–	1	3	184	408
Legionnaires' disease	–	–	–	4	5	2	2	–	–	2	–	–	–	–	1	–	1	–	17
Leptospirosis	–	–	–	–	–	–	1	–	–	8	1	3	4	–	4	–	3	–	24
Listeria	–	1	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1
Malaria	1	1	4	–	4	1	3	–	1	1	1	–	–	–	4	2	–	–	23
Measles	1	1	10	6	16	3	9	2	3	27	13	2	3	–	1	9	–	–	106
Meningococcal inf. (NOS)	–	–	1	2	2	1	1	3	5	1	1	4	2	–	1	1	–	–	25
Meningococcal meningitis	1	3	–	–	–	–	1	–	–	2	1	–	–	–	–	1	–	–	9
Meningococcal septicaemia	–	–	–	–	–	–	–	–	–	–	4	–	–	–	–	1	–	–	5
Mumps	–	–	3	–	1	–	–	–	–	–	–	–	–	–	1	–	–	–	5
Mycobacterial atypical	4	5	–	–	1	–	2	–	–	–	–	–	–	–	–	–	–	–	12
Mycobacterial infection (NOS)	–	–	18	7	29	8	7	1	13	1	3	6	2	3	2	1	–	–	101
Mycobacterial tuberculosis	2	6	–	–	–	–	1	–	–	–	–	–	–	–	–	–	–	–	9
Pertussis	–	–	10	3	2	1	1	–	–	1	3	–	7	–	3	–	–	–	31
Q Fever	–	–	–	1	–	–	–	–	–	3	7	29	86	3	2	1	–	–	132
Ross River fever	–	–	–	–	–	–	–	–	–	1	11	71	21	–	4	–	7	–	115
Rubella	–	1	15	–	2	1	2	–	1	1	–	–	–	–	1	–	–	–	24
Salmonella infection (NOS)	40	49	7	55	111	58	43	–	36	14	41	40	59	14	11	9	18	–	605
Syphilis	16	5	13	23	19	4	1	–	3	13	30	12	78	3	9	1	3	–	233
Tetanus	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	2	–	–	2
Typhoid & paratyphoid	2	–	4	–	–	–	3	–	1	2	–	2	–	–	–	–	1	–	15

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 & 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.

► *Continued from page 82*

LEGIONNAIRES' DISEASE (ICD-9 482.8)

Twenty-three cases of Legionnaires' Disease were notified to Epidemiology and Health Services Evaluation Branch in 1990/91 for a rate of 0.41 cases per 100,000 population. Cases were reported in seven of the 16 Health Areas/ Regions, with the highest rate being reported from Western Sydney (1.5 cases per 100,000).

No definite seasonal trend can be discerned from the small number of notifications received, although a trough is noted from April to June.

Reported Legionnaires' Disease occurred in persons 30 and older, particularly males. No analysis by risk factor, or of specific isolate, has been possible with the current data, although both issues will be addressed by Public Health Units using a new database (Infectious Diseases Database system).

With the introduction of new notification system under the Public Act 1991, Legionnaires' Disease will be notifiable by all laboratories and by Hospital Chief Executive Officers (or their delegates).

FIGURE 8

LEGIONNAIRES' DISEASE
NSW — 1990/91, BY AGE

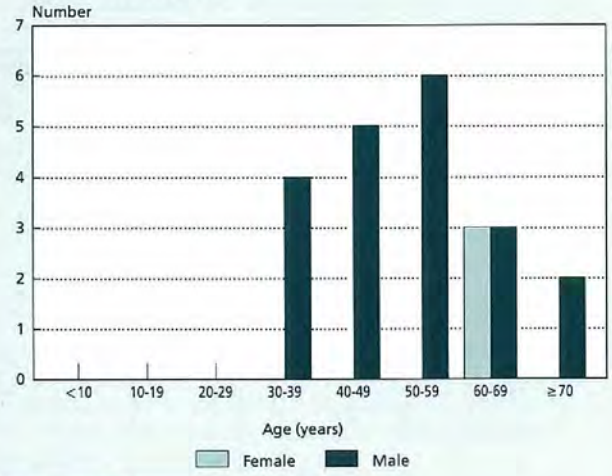


FIGURE 9

LEGIONNAIRES' DISEASE
NSW — 1990/91, BY AREA/REGION

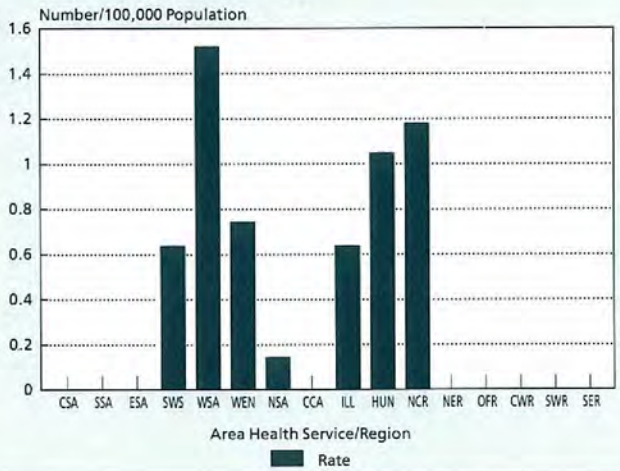


FIGURE 10

LEGIONNAIRES' DISEASE
NSW — 1990/91

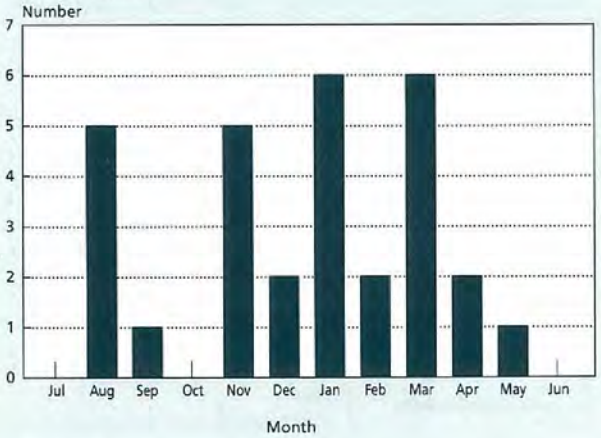
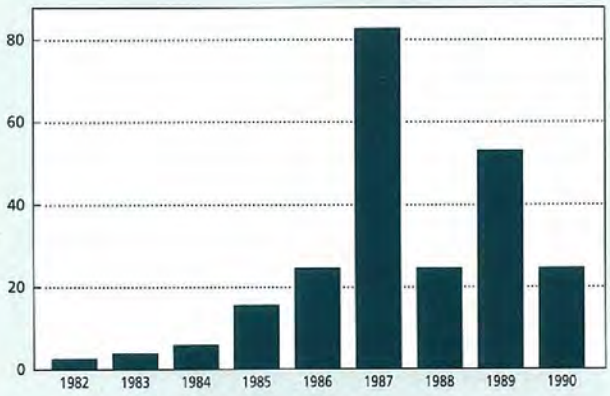


FIGURE 11

LEGIONNAIRES' DISEASE
NSW — 1982-1990



Source: NSW Infectious Diseases Database

NEWS AND COMMENT

PUBLIC HEALTH UNIT MEETING

A meeting of Public Health Unit Directors was held on July 11 at the NSW Health Department, after which a general meeting for all the Public Health Units was held at the Royal North Shore Hospital. These are the key points:

PLANNING

Proposed structure for the NSW Health Enhancement Program Annual Report 90/91 was circulated. It was suggested that "key staffing activities" be added as a category.

It was agreed that each PHU will:

- provide copies of annual reports/planning reports to other PHUs
- provide draft outline of the Unit's report according to the proposed structure to Epidemiology & Health Services Evaluation Branch as a wordperfect document on floppy disc by the end of July
- report on a quarterly basis as a minimum requirement

HEALTH OUTCOMES DOCUMENT — PROGRESS

George Rubin reported on the Year 2001 Health Outcome Goals for NSW document. The existence of health outcome indicators in CEO performance agreements should lead to increased monitoring of health outcomes by CEOs and shift the focus from health service indicators.

Key discussion points:

- The document encourages uniform definitions with decentralised monitoring
- Issues of the accessibility, collection and adequacy of data for measuring outcome targets
- Source of funds for programs
- Role of private hospitals/practitioners in data collection and program implementation.
- Need to set goal priorities in individual Areas/Regions
- Profile and place of environmental health in document

Current status:

- Health outcomes will be incorporated into the Department's corporate plan for 91/92 with key intermediate indicators
- Aim to finalise the document by May 1992 after consultation throughout the Department
- Incorporate outcomes into CEOs' performance agreements — possibly by the 92/93 financial year.

OUTCOME

A decision on the nature and timetable for the consultative process within the Department for the health outcomes document would be made at the Senior Executive Council Meeting (CEOs) in August. (Following distribution of the Year 2001 Health Outcome Goals for the NSW document, George Rubin argued the case for incorporating health outcomes in State, Area and Regional planning and strategy development at the August 7 meeting of the council. Area Chief Executive Officers and Regional Directors gave unanimous support to move to continue the consultative process to develop the document and head towards steady implementation. It was suggested that local and State level consultation take place over the next eight months to determine health outcome areas, targets and intermediate objectives and that health outcomes increasingly be incorporated in senior executive performance agreements.)

STRATEGIC DIRECTIONS

The Epidemiology & Health Services Evaluation Branch's Strategic Plan, 91-95 was tabled for discussion at the next meeting based on the following:

- Relationship of PHU health outcome strategies to other public health agencies — especially community health and health promotion units/agencies
- Inter-sectoral cooperation — strategies to integrate public health activities at the local level.
- Integration of public health strategies within Areas and Head Office of the Department.
- Outline of strategic direction of each PHU for 91-95 period

A draft implementation plan for the 91-95 Strategic Plan will be developed by a working party whose members are to be determined.

BUDGET

It was agreed that:

- funding for PHUs be maintained in real terms for 91-92 financial year
- 1/6th total PHO time will be spent in regional PHUs each year
- PHOs should service Public Health Network — outside funding should be sought for other public health agencies with some exceptions (eg CDC)
- Decision on funding options for uncommitted Line 3 funds be made at next PHU Directors meeting. Options to be considered include training, research, PHU funding, infrastructure, program development/implementation, coordination, E&HSE Branch funding, data systems (including sentinel practices) and new PHOs

Decisions will be made on the Public Health Officer Training Program at the next meeting. They will include a mandatory placement program, composition of the selection committee for PHOs, the number of new PHOs for 91-92 and the medical/non-medical mix of new PHOs.

The next PHU Directors meeting will be held on Friday, August 23 from 10.30am to 5.30pm at Central Office in the level 7 conference room. Agenda items will include: performance agreements for PHUs for 91-92 activities contained in the strategic plan, Epidemiology Branch Strategic Plan 91-95, and decision-making process at Directors meetings.