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# TACKLING CHILDHOOD LEAD POISONING

## THE NEWCASTLE LEAD STUDY

The major sources of environmental lead in Australia are lead processing facilities, paint in older houses and fuel additives. Epidemiologic research has identified adverse effects of lead on the intellectual development of young children at levels previously regarded as safe[1 2 3 4].

The number of children admitted to NSW hospitals has declined in the past 20 years. Between 1983 and 1989, eight NSW children were hospitalised for lead poisoning compared to 35 children admitted to the Royal Alexandra Hospital for Children between 1968 and 1978. Most of these admissions were a result of the ingestion of lead paint[5].

The lead and zinc smelter at Boolaroo on Lake Macquarie is one of three major lead processing plants in NSW. A smelter was first built at the site in 1897. It was demolished in 1922 but roasting of zinc ore and sulphuric acid production continued at the site until the modern plant was commissioned in 1961. Stack and fugitive emissions and dust from ore and slag within the smelter perimeter may continue to contaminate the local area. Houses in the suburbs of Boolaroo and Argenton range from within 270m to 2km from the plant (Fig 1).

In these suburbs it is likely that contaminated house dust and soil are the sources of lead exposure in young children. However, lead in old paint, from car exhausts and on the clothes of lead workers may also be important.

A 1973 survey of children in Boolaroo and Argenton revealed that 6 per cent of children had blood lead levels above 25 $\mu$g/dL[5], the current National Health and Medical Research Council level of concern. The survey was limited in that only 11 per cent of the participants were under four years of age — the group most at risk of exposure and most susceptible to the adverse effects of lead.

Concern by local residents about the adverse effects of lead contamination of the area prompted the Hunter Area Public Health Unit to initiate a new survey.

## METHODS

We first consulted a broad range of community groups including parents, teachers, carers, health professionals, local environmental groups and representatives from the lead smelter.

To define a study area of likely lead contamination we conducted a pilot study of soil lead levels in Boolaroo, Speers Point and Argenton. We then combined these data with results of a 1973 soil survey of the area and data on prevailing wind direction and topography. All children aged one-four years living in the study area were identified by house-to-house survey.

We collected venous blood samples from all participating children. From the houses of those children with blood lead levels $\geq 25$ $\mu$g/dL and from all houses in the two blocks closest to the smelter we collected samples of soil, ceiling and house dust and paint. We superimposed a grid on maps of the suburbs of Boolaroo, Speers Point and Argenton and sampled soil at each intersection point of this grid. To validate the results of blood lead, paint and soil testing, we sent samples to four participating analytical laboratories. This report presents our preliminary results.

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## RESULTS

A total of 149 children were eligible for inclusion in the study. Of these, 140 (94 per cent) agreed to be tested. The mean blood lead level was 15 μg/dL. Thirty children (21.4 per cent) had blood lead levels ≥ 20 μg/dL and of these 23 lived in Boolaroo within five streets of the smelter (Fig 1). Twelve (8.6 per cent) had levels ≥ 25 μg/dL.

For Boolaroo children blood lead levels tend to decrease with increasing distance of their house from the smelter (Fig 2). There is a similar, but less marked, gradient in Argenton children. Two of the three children living beyond the first five streets from the smelter with lead levels above 25 μg/dL lived in houses which may have a lead paint problem. The third child lived in Argenton.

Household assessments completed so far show levels of lead in ceiling dust ranging from 2759-30,764 parts per million (ppm) and in room dust from 23-35,870 ppm. Lead in soil ranged from 8-26,794 ppm. Australian guidelines for the assessment of contaminated sites recommend further investigation if soil levels are above 300 ppm[7].

## DISCUSSION

The high participation rate in this study reflects both the level of community concern and the successful interaction between the community and the Public Health Unit.

The mean blood lead level of 15 μg/dL in our study is high. It compares with a population mean of 8 μg/dL estimated in 1991 from a pooled blood sample from one-four year old children in the Newcastle area[8] and 21 μg/dL found in Port Pirie, South Australia[9]. A study at a number of Sydney schools in 1979 estimated that between 12 per cent and 25 per cent of children had blood lead levels greater than 25 μg/dL[10].

Reducing blood lead levels in these children will require a range of interventions. All households of children with blood lead levels ≥ 25 μg/dL have been assessed to identify sources of lead around the home and behaviour which could have resulted in increased lead absorption. The parents of all children with a blood lead level ≥ 15 μg/dL have been given education materials including a poster highlighting the main sources of lead exposure in young children and ways to control them. Other strategies may include removing heavily contaminated topsoil and contaminated ceiling dust, grassing uncovered and contaminated sites and erecting windbreaks around the smelter. The cost and effectiveness of each of these interventions will need to be assessed. The forthcoming soil testing results will help to delineate the extent of contamination.

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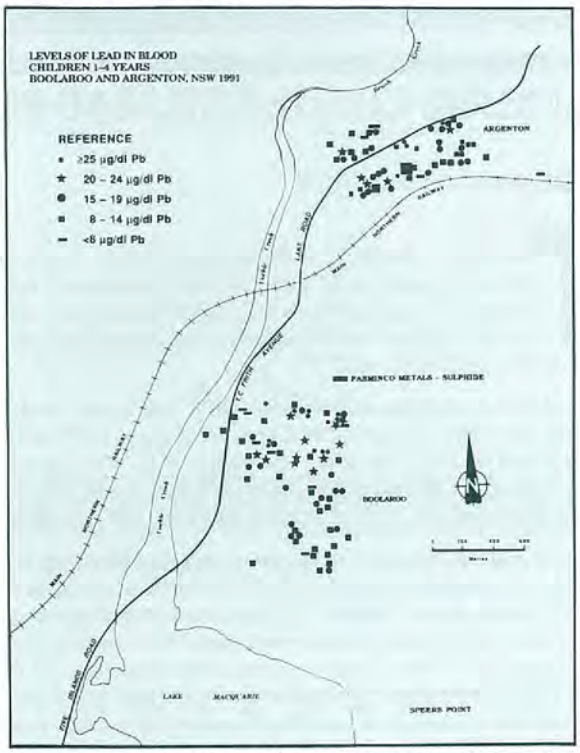
**FIGURE 1**

**LEVELS OF LEAD IN BLOOD CHILDREN 1-4 YEARS BOOLAROO, ARGENTON AND SPEERS POINT, NSW, 1991**

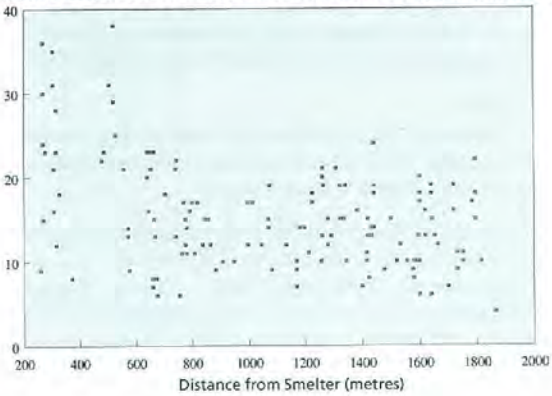



Map of Boolaroo, Argenton and Speers Point showing the place of residence and blood lead level.

Note: Two cases above 20 μg/dL were excluded from the figure as the children lived outside the study area.

**FIGURE 2**

**CHILDREN AGED 1 TO 4 FROM BOOLAROO AND ARGENTON BLOOD LEAD LEVELS BY DISTANCE FROM SMELTER, AUGUST 1991**




Source: Newcastle Lead Study, 1991

# IMPROVING AIDS SURVEILLANCE

An apparent shortfall in predicted new AIDS cases for 1990 recently prompted a review of records among HIV/AIDS-treating facilities in NSW. The review at the Albion Street Centre in Sydney found 24 previously unreported cases of AIDS. This article discusses methods and presents suggestions for improving on-going AIDS surveillance.

There are about 15,000 records of past and present clients at the clinic. A computer database is maintained but does not record the date a person starts living with AIDS or the date of onset of complications such as opportunistic infections or secondary cancers. CD4 lymphocyte (T4 cell) count is recorded. On the basis that most clients progressing to AIDS have a low CD4 count in the later stages of their illness, a search of the database was made for any client whose CD4 count had gone below 200/mm³ at any time in the past three years. This time limit was set assuming that fewer unreported living people with AIDS would be found among those with low CD4 counts before this period.

About 700 clients were identified by this criterion. Each client's history, examinations and test results were reviewed to determine whether the current case definition for AIDS, as provided by the Centers for Disease Control (CDC) in MMWR (1987)[1] was ever satisfied (with the exception of 40 absent records). About 150 people were thereby found to have progressed to AIDS. These cases were then carefully matched by name code and date of birth with entries on the NSW AIDS database at the Epidemiology Branch in the Health Department, to identify cases already notified.

After matching with the NSW AIDS database, 24 of the original 150 cases of AIDS were found never to have been reported by the Albion Street AIDS Centre or any other institution or doctor.

Table 1 presents the AIDS-defining illnesses and numbers for these previously unreported cases:

**TABLE 1**

**AIDS-DEFINING ILLNESS FOR CASES NOT PREVIOUSLY NOTIFIED.**

| AIDS-Defining illness | Number |
|---|---|
| Kaposi's sarcoma | 6 |
| HIV wasting disease | 4 |
| Pneumocystis Carinii Pneumonia (PCP) | 4* |
| Multi-dermatomal herpes zoster | 3 |
| Oesophageal candidiasis | 2 |
| Chronic herpes simplex | 1 |
| Disseminated Mycobacterium Avium Intracellulare | 1# |
| Lymphoma | 1 |
| Extra-pulmonary tuberculosis | 1 |

**two of these were previously diagnosed overseas, and one was diagnosed and treated in a private hospital in Sydney*
*#diagnosed at Prince Henry Hospital*

Most of the unreported cases were of recent onset. Fifteen occurred in 1990, five in 1989 and only four in 1988. There were eight different 'usual treating' doctors for the unreported cases. Differences between them in numbers of unreported AIDS cases were proportional to clinical workload.

Fourteen of the unreported cases had no record of attending a tertiary referral hospital since the onset of their AIDs-defining illness. However, ten had been admitted to hospital subsequently for treatment.

Our review would have missed people whose CD4 count has never been below 200/mm³ (especially those with Kaposi's sarcoma), or who progressed to AIDS more than three years ago and have remained undetected since. The number of the latter such cases is likely to be small, as the median time of survival after diagnosis of AIDS in Australia has been estimated as 10.4 months[2] (although longer for those on azidothymidine).

Nevertheless, we identified a substantial number of AIDS cases not previously notified. It may be useful to consider, from the clinician's view of case notification, what features of the AIDS-defining illnesses of these cases may be relevant to their being unreported.

Kaposi's sarcoma is a cancer which may occur in otherwise well patients and for which no specific treatment is usually given in its early stages. The clinician's minor response to a single lesion may contribute to its omission from reporting as an AIDS case. HIV wasting disease, defined as more than 10% weight loss accompanied by chronic diarrhoea or weakness and fever (without known cause) is a diagnosis which becomes evident slowly, often with no abrupt event. The point at which the condition becomes an AIDS-defining illness may be missed unless the case definition is kept constantly in mind.

Pneumocystis infection is the most common AIDS-defining illness. However, notification may not be uppermost in the mind of a clinician concerned with urgent treatment or referral of a patient with severe symptoms. The early symptoms of oesophageal candidiasis are often relatively mild and of gradual onset. Multi-dermatomal herpes zoster and chronic (more than one month's duration) herpes simplex are both similar in that the onset of the AIDS-defining condition may occur gradually over time. Lymphoma and extra-pulmonary tuberculosis are among the less common AIDS-defining illnesses.

A common feature of many of these unreported cases is a gradual onset with no acute change in the patient's health.

Opportunities for missed notification also exist in the movement of patients between initial and referral centres. Tertiary referral centres may not report a case, believing it to have been already reported by the primary treatment centre, particularly if the onset of the AIDS-defining illness occurred some time previously. The primary centre may, however, leave reporting to the tertiary centre. The possibility of this occurring will increase as centres such as Albion Street treat patients progressively later into the course of their disease.

It is possible that other medical practices providing primary health care to people with HIV, particularly if staffed by a changing population of doctors, have omitted reporting similar types of AIDS cases.

Identification of an AIDS case, however, is not always simple. The 1987 MMWR case definition for AIDS is

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# HOMELESSNESS: A HEALTH ISSUE

*The following article is an edited version of a background paper prepared by Professor Ian Webster for the Public Health Association of Australia.*

Homelessness is a public health issue which is important not only because of the effects on health, but also because it tests the social roots of community health.

Father John Usher, of the Catholic welfare agency Centrecare, told a public meeting in 1988: "Adequate housing is the minimal physical condition to enable a household to create a home. This means much more than protection from the elements and overcrowding. It also implies security of tenure. A home is not a place where tenure is continually threatened and where external pressures are exercising undue control over the family's destiny."

As unemployment, housing costs and numbers on welfare increase, homelessness rises. Compared with the once-accepted yardstick that public housing tenants spend 20 per cent of their income on rent, sole parents renting privately can pay between 32 and 43 per cent of their income on rent. Privately renting couples on the dole spend between 35 and 40 per cent on rent[1,2].

Public concern about homelessness has been crystallised by the Burdekin Report on Youth homelessness[3] and by other reports[4,5,6,7,8].

Housing costs are a cause of poverty; and the lack of affordable housing is a major cause of homelessness.

In Australia, where home ownership is so highly valued, homeless people are marginalised, living outside the mainstream of society. Homelessness may deprive people of the basic necessities of life — personal space, privacy, food, possessions — and often leads to the point of irreversible social handicap. And with the widespread suspicion of welfare cheating and the desire that objective medical tests should be the criterion of invalidity, the environment is ripe for discrimination against the homeless disabled person needing income support through Social Security.

The groups at most risk for long-term homelessness are familiar to public health workers. They are Aborigines, migrant groups, single parent families, the long-term disabled and mentally ill people. These groups comprise a new underclass and are represented disproportionately among the homeless.

A homeless person was described by the Victorian Council to Homeless Persons as someone "without a conventional home and (who) lacks most of the economic and social supports that a home normally affords. S/he is often cut off from the support of relatives and friends, s/he has few independent resources and often has no immediate means and, in some cases, little future prospects of self-support. S/he is in danger of falling below the poverty line at least from time to time."[3]

The health issues in homelessness are:

- Homelessness and impaired health are strongly associated.
- Patterns of living are unhealthy.
- Access to health care is restricted.
- Risks of infectious diseases are high.

One process contributing to the impaired health of homeless people is that there are few ways for them to meet their immediate emotional needs other than in potentially damaging ways — through smoking, alcohol consumption and drug use. The incentives to be "healthy" are hardly evident to people who are oppressed by their marginal social status.

The 1990 annual general meeting of the Public Health Association of Australia (PHA) resolved:

1 To express its concern at the level of homelessness in Australia. It is a major social issue with serious implications for public health. PHA strongly recommends that both Federal and State governments and the wider community should give the highest priority to increasing the stock of affordable housing and the access of disadvantaged persons to accommodation of a reasonable standard.

2 Noting the lack of information about the extent and nature of homelessness and the needs of homeless people, PHA resolves, by all means possible, to promote collaborative research between epidemiologists, social scientists and other disciplines to develop methods for the study of the prevalence of homelessness.

3 Recognising the public concern about youth homelessness, PHA recommends that funding be provided to study the long-term outcomes of youth homelessness.

4 Noting the lack of data about homeless women and their needs, and the needs of homeless women and children, PHA recommends that high priority be given to these issues, both in terms of funding of targeted programs and research.

5 Recognising that the health of homeless persons is compromised by their social and physical environment and lifestyle patterns, PHA recommends that innovative approaches to health promotion be supported for this group, accompanied by thorough evaluation of their effectiveness.

6 Recognising that access to health care is frequently compromised for homeless people and their families, PHA recommends that existing primary health care services for homeless people be evaluated with a view to wider implementation. And that innovative approaches to health care, especially as it relates to homeless youth and women and the ageing homeless, be supported.

7 Noting the increasing problem of ageing and homelessness, PHA recommends that the Federal Government give consideration to directing the Home and Community Care Program to address this problem.

In this country the problem of homelessness is greatest in NSW, thus challenging the public health community in this State to become involved in practical and significant ways.

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# KEEPING THE ELDERLY COVERED

Influenza still has significant morbidity and mortality in people over 65 years. The risk factors for death from influenza are age and underlying disease, and it is estimated that two-thirds of the population 65 years and over can be expected to have one or more chronic medical conditions. During influenza epidemics it is estimated that 80 per cent to 90 per cent of excess deaths are attributable to influenza or its complications[1].

Despite recommendations that everyone over 65 years be immunised yearly against influenza,[2] it appears that coverage rates are less than optimal. Considering the proportion of elderly people in the community is increasing, a more energetic attempt to increase the coverage rate needs to be made.

Recent cases of tetanus highlight the need to increase the coverage rate of immunisation for this disease. Routine immunisation against tetanus began in 1954. There were, however, mass campaigns before that, most notably among servicemen in World War II. The four recent notifications for tetanus in NSW (1990-1991) have been in the unimmunised elderly, suggesting there is a pool of unimmunised people in this age group, especially those who did not serve in the armed forces.[3]

## METHODS

For a four-week period from May 20, 1991, a survey was conducted through the General Practitioner Sentinel Surveillance network of South Western Sydney Area Health Services (jointly administered by the Department of General Practice and the Public Health Unit). A total of 20 general practitioners from various suburbs of South Western Sydney participated in the survey.

The doctors determined (by patient recall or the doctors' records) whether or not all patients 65 years and over had ever been immunised against tetanus and/or been immunised **this year** for influenza. This was irrespective of their reason for presentation to the doctor. It was also recorded if the patient was suffering from influenza as determined by a clinical definition.[4]

## RESULTS

Of a total of 431 patients 65 years or over seen during the period, 173 (40 per cent) were male and 258 (60 per cent) were female. Forty-five (10.4 per cent) were suffering from clinical influenza. The oldest patient seen was 94 years. The table shows the numbers and proportions of those immunised against tetanus and influenza. Males were more likely to have received tetanus vaccine (65.3 per cent male, 46.9 per cent female — chi square 13.4, $p < 0.01$), but there was no significant difference for influenza vaccine (54.3 per cent male, 51.2 per cent female).

Of the 226 patients who had been vaccinated for influenza, only 5 (2.2 per cent) presented with clinical influenza. Of the 205 who were not vaccinated for influenza, 40 (19.6 per cent) presented with clinical influenza (chi square 32.8, $p < 0.01$).

Table 2 shows the age immunisation coverage by age for those 65 years and over. This showed relatively little variation by age.

## DISCUSSION

It is stressed that this was a one-month only preliminary survey to estimate coverage rates. There may be bias due to problems associated with patient recall and record documentation. Nevertheless it showed less than optimal influenza and tetanus coverage rates in the elderly. The number of cases of influenza and tetanus in the elderly warrant a more rigorous approach to improving the coverage rates for immunisation against these diseases. The local general practitioner is in a good position to improve the situation by considering the immune status of people 65 years and over on presentation to the surgery no matter the initial reason for consultation. Consideration might also be given to including promotion of tetanus vaccination in association with the next influenza vaccination campaign.

**TABLE 2**

**PROPORTION OF PATIENTS 65 YEARS AND OVER EVER IMMUNISED AGAINST TETANUS OR WHO HAD RECEIVED INFLUENZA VACCINE THIS YEAR.**

| | Tetanus Vaccination No. (%) | Influenza Vaccination No. (%) | Total Patients |
|---|---|---|---|
| Male | 113 (65.3) | 94 (54.3) | 173 |
| Female | 121 (46.9) | 132 (51.2) | 258 |
| Total | 234 (54.3) | 226 (52.4) | 431 |

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# THE CONTINUING CONCERN WITH TB

## TUBERCULOSIS (ICD-9 010-018)

Latest data on tuberculosis (*Mycobacterium tuberculosis*, "TB") notifications for 1989 and 1990 show that TB continues to be a problem in NSW, and may be increasing. The rate of new cases of tuberculosis in NSW in 1989 was 4.89/100,000, and 5.80/100,000 in 1990. The number of total mycobacteria notifications (including atypical mycobacteria) in 1990 was the highest for at least eight years (see Figure 3). The number of annual new TB cases over this period has not been determined; mainframe records containing these data will be analysed at a later stage.

There were 17 deaths from tuberculosis in 1989 (annual rate of 0.30/100,000 population) and 20 deaths in 1990 (annual rate of 0.35/100,000).

There were 14 reactivations of M tuberculosis in 1989 (4.5 per cent of notifications) and six reactivations in 1990 (1.7 per cent of notifications). In addition, there was one case reactivated atypical mycobacteria in each of the two years that we report.

There were 59 cases denotifiec in both 1989 and 1990. This reflects the situation that tuberculosis notifactions are made provisionally, with microbiological confirmation coming up to two months later. Total notifications reflect the workload of the tuberculosis service with regard to contact tracing.

Case definitions used in this report are:

**New TB:**

- A patient who has a diagnosis of TB with culture confirmation of Mycobacterium tuberculosis, or
- A patient diagnosed as having active TB on clinical and/or radiological grounds, in the absence of bacteriological evidence, and who is receiving a course of anti-tuberculous chemotherapy.

**TB reactivation:**

- A patient diagnosed again as having active TB who has previously been notified in NSW and had received a recognised course of anti-tuberculous chemotherapy. (Patients with a history of TB and/or treatment prior to coming to NSW are classified as new cases.)

**"Atypical" mycobacterial infection:**

- A patient with a clinically apparent infection caused by bacteriologically confirmed "atypical" mycobacteria, or
- A patient from whom bacteriologically confirmed "atypical" mycobacteria are recovered from sites which are normally sterile, or
- A patient from whom the same "atypical" mycobacterium has been isolated in moderate amounts from the same site.

The figures relate to new cases unless otherwise stated. Table 3 presents more details on the 1989 and 1990 TB data. The table shows that new TB cases made up fewer than 60 per cent of all mycobacteria notifications during these years. More than three-quarters of new TB cases were pulmonary in site. Total new TB cases rose 20 per cent from 1989 to 1990, although this may be within normal year-to-year variability.

**TABLE 3**

**MYCOBACTERIA NOTIFICATIONS, NSW, 1989 AND 1990**

| | 1989 | | 1990 | |
|---|---|---|---|---|
| | No. | % | No. | % |
| Total mycobacteria notifications | 515 | 100 | 584 | 100 |
| Cases denotified | 59 | 11.5 | 59 | 10.1 |
| New TB cases — total | 310 | 60.2 | 346 | 59.2 |
| New TB cases — pulmonary | 241 | (77.7) | 263 | (76.0) |
| New TB cases — extrapulmonary | 69 | (22.3) | 83 | (23.9) |
| Atypical mycobacteria | 14.6 | 28.3 | 179 | 30.7 |

Figure 4 shows new reported cases of tuberculosis for 1990 by age and sex. There is a male preponderance of cases (199 of 338 — 59 per cent). The highest incidence occurred in the 20-39 years and 60 years and over age groups. The 1989 data also demonstrate a male preponderance and peak incidences in these age groups. This is consistent with previous age distribution patterns for tuberculosis in NSW.

Rates for new cases by Area/Region in 1990 are shown in Figure 5. Note that the data for Wentworth and Western Sydney Areas have been combined to produce a single rate. The Areas predominate, particularly those of the Sydney metropolitan region, with the highest rate in the Central Sydney Area. This distribution may be explained by greater population densities and high numbers of overseas-born residents in these areas. It is of note that Central Sydney has an incidence of TB more than twice that of the overall NSW rate, and that all Sydney metropolitan Areas except Northern Sydney have incidence rates above the State average. However, these rates have not been adjusted for age or sex.

Table 4 provides a breakdown by region of birth/ Aboriginality. Of note is the very low rate among Australian Aborigines, and a high rate among Asian-born immigrants, reflecting the endemicity of TB in that region.

**TABLE 4**

**NEW TB CASES BY BIRTHPLACE AND ABORIGINALITY, NSW, 1989 AND 1990**

| | 1989 | | 1990 | |
|---|---|---|---|---|
| | No. | % | No. | % |
| Australia (caucasian) | 83 | 26.8 | 90 | 26.0 |
| Australia (Aboriginal) | 4 | 1.3 | 3 | 0.9 |
| Asia | 135 | 43.5 | 158 | 45.7 |
| Europe | 34 | 11.0 | 38 | 11.0 |
| Oceania | 11 | 3.5 | 16 | 4.6 |
| Others | 43 | 13.9 | 41 | 11.8 |

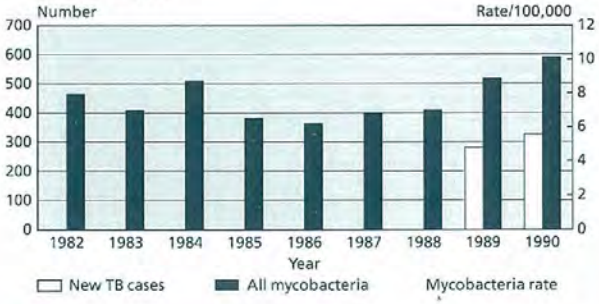
In 1990 there was no clear temporal or seasonal occurrence of new TB cases (Figure 6). Reports may be low in December due to low patient attendance or reporting activity in that month.

Overall, the data show that TB continues to be a problem in NSW, and may be increasing. Considerable variation exists in TB rates across Areas/Regions of the State, with the highest rates occurring in the Central, Eastern and Southern Sydney Areas.

Prevention of tuberculosis will continue to depend on early detection (including investigation of contacts and, where appropriate, screening of high-risk groups), effective treatment and adequate follow-up of both treated and suspected cases.

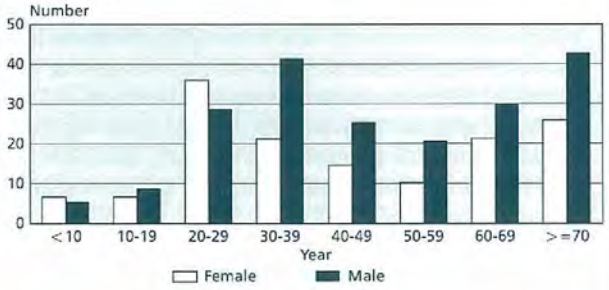
## FIGURE 3

MYCOBACTERIA NOTIFICATIONS
NSW, 1982-1990



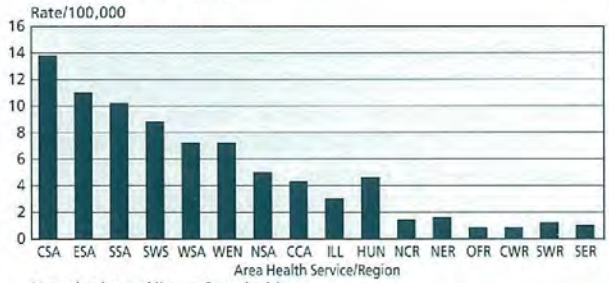

## FIGURE 4

TUBERCULOSIS
NEW CASES NOTIFIED BY AGE AND SEX
NSW, 1990




## FIGURE 5

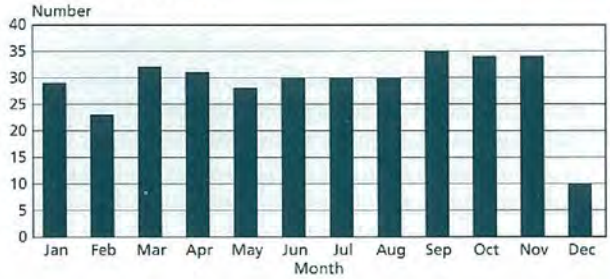
TUBERCULOSIS
NEW CASES NOTIFIED
BY AREA/REGION, NSW, 1990




Note: horizontal line = State incidence

## FIGURE 6

TUBERCULOSIS
NEW CASES NOTIFIED
BY MONTH, NSW, 1990




## Improving AIDS surveillance

► Continued from page 100

complex and at times vague and ambiguous. It also appears to be deficient; for example, HIV-related thrombocytopaenia, which may cause gastrointestinal haemorrhage and death, is not included in the definition as a specific entity. The case definition takes no account of immune system status, so a patient with a CD4 count of zero may never constitute a case. Additionally, prophylactic or other treatments may prevent or shorten or lessen the severity of attacks of illness so the threshold of AIDS diagnosis (for example, for PCP, 'chronic HSV' or oesophageal candidiasis) is never reached.

Funding formulas that rely on numbers of AIDS cases diagnosed, using the current definition, may therefore underestimate the resources required for dealing with HIV-related disease.

In conclusion, possible improvements to on-going AIDS surveillance noted during our review include the following:

1. Clinical services treating substantial numbers of HIV-positive people should maintain a computer database of clients, containing fields at least for seropositivity, CDC stage, significant diagnoses and whether notified, if a case.

2. A record should be kept in patients' files indicating whether they have been notified if they fulfil AIDS-defining criteria.

3. It would be valuable if one senior, motivated clinician were made responsible for correct and timely notification in all mutliple-doctor centres. They could provide copies of the most recent CDC/MMWR case definition for AIDS, as well as material on the importance of notification, to all doctors in contact with clients/patients in their centre. Regular reminders may also be necessary.

4. There should be feedback to doctors after notification — a receipt for the notification has been suggested and may be instituted. The circulation of a regularly updated (confidential) list of cases notified by a centre to its practising doctors may be sufficient.

5. Depending on the size and type of institution, a regular review could be undertaken of records of HIV positive patients to look for missed notifications.

6. Clear guidelines on who is responsible for notification also need to be provided to all centres. Perhaps both referring and tertiary treatment centres should be asked to notify, with duplication detected and removed by the Epidemiology Branch.

Many of these facilities or procedures already exist in some centres, or are being implemented.

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# DOES INTRADERMAL HEPATITIS B VACCINE WORK?

The hepatitis B vaccine used in NSW is of the recombinant or genetically engineered type. The genetic material within the DNA of the hepatitis B virus was initially cloned into the DNA of *Escherichia coli*, enabling the isolation of the S-gene and its incorporation into an expression plasmid containing the components necessary for replication and maintenance in yeast cells. Further manipulation enabled the transformed yeast cells to produce hepatitis B surface antigen (HBsAg)[1].

Hepatitis B vaccine is recommended for use in the following groups[2,3]:

1 Households, institutions, schools
   - seronegative household contacts of a carrier
   - seronegative mentally retarded persons in care
   - seronegative children in kindergartens and primary school where there is a significant ethnic carrier population.
2 High-risk individuals
   - sexual partners and children aged less than five years of chronic carriers and acute hepatitis B patients
   - intravenous drug users
   - homosexuals with multiple partners
   - prostitutes
3 Health care workers, including health care profession students, mortuary attendants and some laboratory personnel
4 Babies born to carrier mothers and babies born into ethnic groups with high carrier rates
5 People receiving multiple transfusions/haemodialysis
6 Prisoners

Hospital personnel in contact with blood and body fluids are at significant risk of contracting hepatitis B. In the United States, Dienstag et al found a prevalence of markers of prior hepatitis B virus (HBV) infection in 16 per cent of health care workers[4]. In Australia the highest prevalence of HBV markers among health care workers was 16 per cent among the dental professions, and the highest prevalence among patients was 80 per cent in a group attending Sydney's Sexually Transmitted Diseases Clinic[5,6].

The vaccination schedule recommended for adults is a course of three injections of 1.0mL, with the second after a one-month interval and the third after a six-month interval following the initial injection. The prescribing information for the vaccine states it should be injected intramuscularly[1].

The reason for instituting the *intradermal* instead of the intramuscular route of vaccination was one of economy. It was believed the intradermal route was as efficacious as the intramuscular route. The vaccination program was not instituted for the purpose of a scientific survey and no specific consent was obtained. The cost difference between the course of vaccination and post-screening per health care worker was $46.30 (73 per cent) less at 1988 prices, because a smaller intradermal dose was able to be used. Details are given in Table 5.

Some studies of health care workers or trainee health care workers found comparable seroconversion rates between the intramuscular and intradermal regimes[8,9].

**TABLE 5**

**COST OF HEPATITIS B VACCINATION 1988***

| Item | Intramuscular Route 1.0 mL Dose | Intradermal Route 0.1 mL Dose |
|---|---|---|
| Vaccine — 3 doses | $54.00 | $ 7.70 |
| Post-screening | $ 9.20 | $ 9.20 |
| Total cost per Health Care worker | $63.20 | $16.90 |

*The hospital contract cost of hepatitis B vaccine per adult dose in March, 1991 is $7.65.

Wilkins and Cossart found seroconversion to protective levels of antibodies to hepatitis B surface antigen (anti-HBs) in 89 per cent after four intradermal doses, but they used the first generation serum-derived vaccine which would have contained more antigenic material because of impurities, their cohort was young, and according to the methods description there was no follow-up after seroconversion[9]. Other studies found the intradermal route produced significantly lower levels of anti-HBs at all points measured up to 18 months; and 19 to 21 months[10,11].

Because of continuing problems with waning immunity at two hospitals following the intradermal course, the Central Western Health Region administration at Bathurst was asked this year to review the situation and to advise on remedial measures. We believe this is the first retrospective survey conducted in Australia of the intradermal administration of the second generation or recombinant hepatitis B vaccine where vaccinees were followed up for persistence of antibody levels. The vaccine used was Engerix-B, from Smith, Kline and French Laboratories Australia Limited, a product manufactured and imported from the parent company Smith Kline-RIT, Belgium.

## METHODS

From April 1988, 161 health care workers at Parkes and Peak Hill hospitals, 400km west of Sydney, were given a course of 0.1mL (2$\mu$g) of vaccine by the intradermal route into the deltoid area. Timing of the schedule was the same as for intramuscular use (0, 1 and 6 months). Each single 1.0mL vial of vaccine for intramuscular use yielded seven doses of 0.1mL for intradermal use. The vaccine batches known to have been used were ENG165B4 until about November 1989, then ENG181A4, and later in 1990 ENG186B4, ENG639A4 and ENG628A4A. No screening for prior HBV infection was undertaken because of cost considerations.

Post-vaccination assays employed the Amersham ELISA test initially. Later in 1990 the Abbott Laboratories' Radioimmunossay AUSAB was also used. Immunity was determined at $\geq$ 10 IU/L anti-HBs (or its equivalent by AUSAB).

The 161 subjects consisted of 36 males and 125 females. Among the 83 subjects whose age was recorded, the range was 24 to 63 with a mean age of 42 years.

**TABLE 6**

**TYPICAL HEPATITIS B VACCINATION HISTORY OF A PARKES HOSPITAL HEALTH CARE WORKER WITHOUT A PROTECTIVE ANTIBODY LEVEL FOLLOWING THE INTRADERMAL COURSE**

| Date | Batch | Route | Test |
|---|---|---|---|
| 31.5.88 | ENG165 B4 | ID | |
| 28.6.88 | ENG165 B4 | ID | |
| 14.12.89 | ENG181 A4 | ID | |
| 21.2.89 | | | anti-HBs negative |
| 22.3.89 | ENG186 B4 | IM | |
| 8.5.89 | | | anti-HBs positive |
| 16.7.90 | | | anti-HBs negative |
| 23.7.90 | ENG628 A4 | IM | |
| 21.8.90 | ENG628 A4a | IM | |
| March 1991 | | | Test recommended — not yet undertaken |

Initially those who did not demonstrate anti-HBs at ≥ 10 IU/L some two to three months after the intradermal course were offered a one-dose booster of 1.0mL of vaccine by the intramuscular route, and were retested about two months later. During the program it became apparent that the single boosters did not yield lasting protective antibody levels in many subjects. Some then received the full course of intramuscular injections following the intradermal course, without any intervening antibody assay, although such assays were available and were recommended in the event of sharps/needlesticks injuries.

## RESULTS

i) **Initial evaluation two-three months post-vaccination.**
Of the 161 subjects, 103 (64 per cent) had anti-HBs at the protective level of ≥ 10 IU/L, and 58 (36 per cent) did not have anti-HBs to this level.

ii) **Monitoring 18 months after the intradermal course.**
Of the 103 who had anti-HBs after the course, 43 presented for retesting within 18 months. Of these 26 (60 per cent) had protective levels of anti-HBs, while in 17 (40 per cent) the immunity had waned to < 10 IU/L.

iii) **Monitoring after the intradermal course and one intramuscular booster.**
Of the 58 who failed to demonstrate anti-HBs ≥ 10 IU/L after the intradermal course, 29 (50 per cent) achieved protective levels after one intramuscular booster. Thirteen of these were tested again within 18 months. Only five (38 per cent) remained at protective levels and in eight (62 per cent) immunity had waned to < 10 IU/L. The antibody status of the other 16 is unknown. Detailed records were not available for the calculation of geometric mean titres.

iv) **Typical profile**
The recommendation was subsequently made that the intradermal course be followed by a three-dose intramuscular course. An example of a typical profile of procedures of one of the 58 health care workers, who did not have protective antibody levels after the intradermal course, is given in Table 6.

## DISCUSSION

The present investigation was initiated when the Parkes and Peak Hill hospitals requested assistance with their hepatitis B immunisation program because immunity had waned in many vaccinees, following either the intradermal course or the subsequent intramuscular booster. The investigation was not designed to compare the intramuscular with the intradermal route, and some data that would have been required for such a comparison were therefore not collected.

In 1981 hepatitis B immunisation by the intramuscular route using serum-derived hepatitis B vaccine was shown to yield seroconversion in 96 per cent following a course of three injections (0,1, 6 months)[7]. The Parkes and Peak Hill hospitals' program of intradermal vaccination yielded protective levels of anti-HBs in only 64 per cent. We were disappointed with this response since it was lower than those reported in the initial trials by the intramuscular route. Of concern is that among the 64 per cent of subjects who had protective antibody levels after the course, they waned in 40 per cent within 18 months.

This is in contrast to antibody levels following the use of first generation vaccine by the intramuscular schedule, which are generally considered to remain at protective levels for more than five years, though two recent studies have shown they also wane within five years in 19 per cent and 35 per cent of adult subjects[12,13].

Furthermore, a single intramuscular booster following the intradermal course resulted in protective antibody levels in only half the subjects, and immunity again waned among 62 per cent within 18 months. It is probable that the immunological memory or anamnestic response would have been protective for some who subsequently contracted a HBV infection.

Based on these data, the use of the intradermal route with Engerix-B cannot be recommended. The only two recombinant hepatitis B vaccines licensed for use in Australia are Engerix-B and H-B-VAX II (Merck Sharp & Dohme). Both are recommended for intramuscular use[1,14].

As the yeast-derivation, immunogenicity and purified HBsAg content of H-B-VAX II is similar to Engerix-B, our results indicate that this vaccine would also need careful evaluation before intradermal use is adopted.

*Continued on page 107* ►

## Does intradermal vaccine work?

► Continued from page 106

From the data of the Red Cross Blood Transfusion Service and health care worker surveys, it is considered likely that between 5 and 10 per cent of the 161 subjects would have had prior markers of HBV infection if the pre-vaccination tests had been employed[5,6]. Those from Peak Hill may have had a higher percentage of markers because of the high proportion of Aborigines in that town. For those who had previously acquired anti-HBs, the vaccine course would have boosted immunity, while it would have had no effect in the expected 0.5 per cent of HBsAg carriers, irrespective of the route of administration. Those with evidence of prior infection need not have been vaccinated.

There is no history of adverse factors, such as freezing the vaccine, a break in the cold chain or injection into adipose tissue, but the subject population had a mean age of 42 and it is known that in those over 40 years the seroconversion rate is lower following the intramuscular route of vaccine administration[15]. However, the intramuscular route response by age may not be equated to an intradermal response without the evidence of a clinical trial. Such an age bias is common among hospital employees in NSW country regions. Anti-HBs levels following intramuscular hepatitis B immunisation may not reach a peak until three months after the course[16]. The determination of assays in this program two to three months post-vaccination are considered not to have significantly affected the results obtained. The value of the post-vaccination assay for anti-HBs level is re-emphasised[17].

In the Central Western Region those who had received the vaccine intradermally were recommended to have a course of three immunisations by the intramuscular route.

There is some evidence that health care workers elsewhere may be in a similar position because other institutions adopted hepatitis B vaccination by the intradermal route for their health care workers. It is recommended that an assessment of the situation be instituted by all Public Health Units in NSW and that other States be made aware of the finding.

The hospitals' responsibilities under the Occupational Health & Safety Act must be taken into consideration. These include the absolute obligation of the employer to ensure the employees are not subjected to a known health hazard. Given the disappointing results of the intradermal program the hospitals had a moral and legal obligation to pursue the workers' interests and initiate a remedial program, aimed at achieving successful immunity to hepatitis B. The initial economy considerations, which were the reasoning behind the intradermal program, would likely be outweighed by the compensation costs of a single successful court case, should a work-related infection take place where the health care worker subsequently becomes a carrier of HBsAg. The economic consideration is also not relevant now as the vaccine price has been greatly reduced since 1988, and in March 1991 was $7.65 per adult dose (hospital contract price).

Many procedures were required for each health care worker as can be seen from Table 5. Additional post-vaccination screening costs have been incurred by hospitals. There is now a degree of ill-feeling toward the intradermal program and some personnel may no longer be presenting for further vaccinations or antibody assays.

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# INFECTIOUS DISEASES

## NOTIFICATIONS

Cumulative notifications of infectious diseases for 1991 are presented in Table 7.

As a result of a review of 1991 data, previously published cumulative data have been amended. Duplicate notifications have been deleted, and some notifications have been reclassified, for example, from acute viral hepatitis to hepatitis A or B. Reporting from Public Health Units has improved, and data from backlogs of notifications have been incorporated. The data quality review is continuing and may result in further changes to cumulative figures.

This month 13 of the 14 PHUs have provided data to Epidemiology Branch, a great improvement over previous rates. However, there is delay in reporting infections to the PHUs by doctors and laboratories, so September notifications presented in tables 8 and 9 will be considerably lower than the eventual totals.

Of note this month is the continuing high rates of syphilis in the Orana/Far West region (111 cases per 100,000 population per year, compared with the State average of 10 per 100,000). Hepatitis A cases also continue to occur in the inner Areas of Sydney, predominantly among males in the 20- to 39-year age group.

## HEALTH INFORMATION FOR INTERNATIONAL TRAVEL

The third edition of the Commonwealth Department of Health, Housing and Community Service booklet *Health Information for International Travel* is expected to be published late this month. This booklet is intended as a comprehensive reference for health care providers and associated professionals who may be called on to advise overseas travellers on health matters. The new edition has been considerably revised and expanded (now about 100 pages) and includes a detailed index. In addition, three information sheets directed at intending overseas travellers, covering immunisation, malaria, and food and water-borne disease, have been included. The booklet will be available from the Australian Government Publishing Service at a cost of $9.95.

## RUBELLA IN THE ACT

More than 80 cases of rubella (german measles) were identified early in September at one primary school in Canberra. Investigations by the ACT Board of Health revealed cases at other primary schools and some child care centres. Occasional outbreaks of rubella are not unusual. Rubella is generally a mild disease, but presents a danger during pregnancy, potentially causing birth defects.

Prevention of rubella, and of the congenital rubella syndrome, relies on:

- immunisation of all children at the age of 12 months
- immunisation of schoolgirls in Year 6 or 7
- determining rubella antibody titres of all pregnant women
- postnatal immunisation of women found to have low antibody titres during pregnancy.

The rubella vaccine is contra-indicated in pregnancy. Under the NSW Public Health Act, 1991, rubella will be notifiable by laboratories.

## MEASLES

Reports of measles cases continue, particularly from the Hunter and Central Sydney Areas. Prompt action by the Public Health Units to immunise susceptible school contacts prevented larger outbreaks. The Epidemiology Branch has been informed of the death of a 14-year-old NSW boy from subacute sclerosing panencephalitis (SSPE), a late sequel of measles infection.

## ERRATA: SEPTEMBER 1991 PUBLIC HEALTH BULLETIN

Tables 8 and 9 of the September 1991 Public Health Bulletin, on new 1991 diagnoses of HIV infection in NSW cumulative to August 30, 1991, contain several errors. The heading for Table 10 was also incorrect, and should have read "Infectious Diseases Notifications by Area and Region from January 1 to August 31, 1991". Corrected versions of these tables are provided on a re-issue of pages 94 and 95 for substitution in the September 1991 Bulletin. The corrected Tables 8 and 9 refer to the period January 1-August 31, 1991.

## TABLE 7

**INFECTIOUS DISEASE NOTIFICATIONS, NSW**
**Notifications to the end of September, 1991**

| CONDITION | Number of Cases Notified | | | |
|---|---|---|---|---|
| | Period | | Cumulative | |
| | September 1990 | September 1991 | September 1990 | September 1991 |
| AIDS | *32 | *14 | *255 | *184 |
| Arboviral infection (NOS) | 1 | 1 | 3 | 384 |
| Brucellosis | – | – | 5 | 2 |
| Cholera | – | – | – | – |
| Diphtheria | – | – | – | – |
| Foodborne illness (NOS) | 242 | 64 | 1949 | 2281 |
| Gastroenteritis (instit.) | N/A | – | N/A | 37 |
| Gonorrhoea | 16 | 2 | 285 | 264 |
| H influenzae B – epiglottitis | N/A | 1 | N/A | 7 |
| H influenzae B – meningitis | – | 5 | 10 | 30 |
| H influenzae B – septicaemia | – | – | 2 | 6 |
| H influenzae infection (NOS) | – | 3 | 10 | 99 |
| Hepatitis A | 2 | 28 | 22 | 612 |
| Hepatitis B – acute | – | – | 6 | 17 |
| Hepatitis B – carrier | – | – | – | 22 |
| Hepatitis B – unspecified | 38 | 20 | 318 | 743 |
| Hepatitis C | 1 | 8 | 20 | 187 |
| Hepatitis, acute viral (NOS) | – | 12 | 2 | 295 |
| HIV infection | 46 | 20 | 573 | 611 |
| Hydatid disease | – | – | 2 | 2 |
| Legionnaires' disease | 1 | – | 23 | 22 |
| Leprosy | – | – | – | – |
| Leptospirosis | 5 | – | 32 | 25 |
| Listeriosis | N/A | – | N/A | 4 |
| Malaria | 19 | – | 131 | 100 |
| Measles | 81 | 6 | 176 | 254 |
| Meningococcal meningitis | – | 2 | 17 | 30 |
| Meningococcal septicaemia | – | – | 5 | 10 |
| Meningococcal infection (NOS) | 12 | 2 | 51 | 32 |
| Mumps | N/A | – | N/A | 3 |
| Mycobacterial tuberculosis | – | 2 | – | 136 |
| Mycobacterial – atypical | – | – | 14 | 43 |
| Mycobacterial infection (NOS) | 51 | 1 | 406 | 121 |
| Pertussis | 10 | – | 126 | 34 |
| Plague | – | – | – | – |
| Poliomyelitis | – | – | – | – |
| Q fever | 9 | – | 99 | 158 |
| Ross River fever | 1 | – | 247 | 128 |
| Rubella | N/A | 1 | N/A | 25 |
| Salmonella infection (NOS) | 91 | 10 | 1117 | 911 |
| Syphilis | 24 | 26 | 271 | 431 |
| Tetanus | – | – | – | 2 |
| Typhoid & paratyphoid | 5 | 1 | 29 | 35 |
| Typhus | – | – | – | – |
| Viral haemorrhagic fevers | – | – | – | – |
| Yellow fever | – | – | – | – |

* Data January-August only
(NOS) Not otherwise specified

## *Infectious diseases*

► *Continued from page 108*

### TABLE 8

**INFECTIOUS DISEASE NOTIFICATIONS, BY HEALTH AREA & REGION**
**For September, 1991**

| CONDITION | CSA | SSA | ESA | SWS | WSA | WEN | NSA | CCA | ILL | HUN | NCR | NER | OFR | SWR | SER | U/K | TOTAL |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Arboviral infection (NOS) | – | – | – | – | – | – | – | – | – | – | – | – | – | – | 1 | – | 1 |
| Foodborne illness (NOS) | 2 | 11 | 2 | 5 | 7 | 8 | – | – | – | – | 6 | 1 | 8 | 14 | – | – | 64 |
| Gonorrhoea | 1 | – | – | – | – | – | – | – | – | – | – | – | – | 1 | – | – | 2 |
| H. influenzae B | – | 3 | – | – | – | – | – | – | – | – | – | – | – | – | – | – | 3 |
| H. influenzae epiglottitis | – | – | – | – | – | – | 1 | – | – | – | – | – | – | – | – | – | 1 |
| H. influenzae meningitis | – | 2 | – | 1 | 2 | – | – | – | – | – | – | – | – | – | – | – | 5 |
| Hepatitis A | 10 | 4 | – | 1 | 2 | 1 | 8 | – | 1 | – | – | – | – | 1 | – | – | 28 |
| Hepatitis B — Unspecified | – | 4 | – | 4 | 7 | – | 3 | – | – | – | – | – | 2 | – | – | – | 20 |
| Hepatitis C | – | – | – | 1 | 2 | – | 5 | – | – | – | – | – | – | – | – | – | 8 |
| Hepatitis, acute viral (NOS) | – | – | – | – | – | 1 | – | – | – | 3 | – | – | – | – | 8 | – | 12 |
| HIV infection | 1 | – | 4 | – | – | – | – | – | – | – | – | – | – | – | – | 15 | 20 |
| Measles | 4 | – | – | – | – | – | – | 1 | 1 | – | – | – | – | – | – | – | 6 |
| Meningococcal meningitis | – | – | – | – | – | – | – | – | 2 | – | – | – | – | – | – | – | 2 |
| Meningoccocal infection (NOS) | – | – | – | – | 1 | – | – | 1 | – | – | – | – | – | – | – | – | 2 |
| Mycobacterial tuberculosis | – | – | 1 | – | – | – | 1 | – | – | – | – | – | – | – | – | – | 2 |
| Mycobacterial infection (NOS) | – | – | – | – | – | – | 1 | – | – | – | – | – | – | – | – | – | 1 |
| Rubella | – | – | – | – | 1 | – | – | – | – | – | – | – | – | – | – | – | 1 |
| Salmonella infection (NOS) | 1 | – | – | 2 | 2 | – | – | – | – | – | 1 | 1 | – | 3 | – | – | 10 |
| Syphilis | 2 | 2 | – | 4 | 1 | 1 | 1 | – | – | – | 3 | – | 11 | 1 | – | – | 26 |
| Typhoid & paratyphoid | – | – | – | – | 1 | – | – | – | – | – | – | – | – | – | – | – | 1 |

### TABLE 9

**INFECTIOUS DISEASE NOTIFICATIONS, BY HEALTH AREA & REGION**
**For period January 1 to September 30, 1991**

| CONDITION | CSA | SSA | ESA | SWS | WSA | WEN | NSA | CCA | ILL | HUN | NCR | NER | OFR | CWR | SWR | SER | OTH | U/K | TOTAL |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| AIDS* | 30 | 6 | 79 | 3 | 17 | 7 | 17 | 5 | 3 | 6 | 9 | – | – | – | – | – | – | 2 | 184 |
| Arboviral infection (NOS) | – | – | 4 | – | 1 | – | – | – | 1 | 8 | – | 138 | 188 | 4 | 35 | 5 | – | – | 384 |
| Brucellosis | – | – | 2 | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | 2 |
| Foodborne illness (NOS) | 175 | 333 | 480 | 123 | 229 | 140 | 1 | 35 | 16 | 89 | 277 | 125 | 109 | 24 | 106 | 2 | 17 | – | 2281 |
| Gastroenteritis (instit.) | – | – | – | 5 | 10 | 6 | 4 | 2 | – | – | – | 7 | 3 | – | – | – | – | – | 37 |
| Gonorrhoea | 16 | 6 | 118 | 23 | 16 | – | 7 | 1 | 11 | 4 | 6 | 4 | 43 | – | 7 | 1 | 1 | – | 264 |
| H. influenzae epiglottitis | 1 | – | – | 3 | 1 | – | 2 | – | – | – | – | – | – | – | – | – | – | – | 7 |
| H. influenzae meningitis | 1 | 3 | – | 6 | 2 | 1 | 8 | – | – | 7 | – | – | – | – | 2 | – | – | – | 30 |
| H. influenzae septicaemia | – | 2 | – | – | – | – | 3 | – | – | 1 | – | – | – | – | – | – | – | – | 6 |
| H. influenzae infection (NOS) | 11 | 18 | 8 | 2 | 13 | 12 | 1 | 2 | 9 | 2 | – | 1 | 5 | 2 | 9 | 4 | – | – | 99 |
| Hepatitis A | 81 | 38 | 297 | 28 | 21 | 2 | 102 | – | 5 | 13 | 3 | 9 | 4 | – | 3 | 5 | 1 | – | 612 |
| Hepatitis B — Acute | 11 | 4 | – | – | – | – | – | – | – | – | – | – | – | – | – | 2 | – | – | 17 |
| Hepatitis B — Carrier | 9 | 11 | 1 | – | 1 | – | – | – | – | – | – | – | – | – | – | – | – | – | 22 |
| Hepatitis B — Unspecified | 103 | 66 | 67 | 164 | 84 | 5 | 86 | – | 5 | 33 | 17 | 36 | 56 | – | 3 | 16 | 2 | – | 743 |
| Hepatitis C | 67 | 22 | – | 7 | 13 | 1 | 40 | 1 | 2 | 12 | 8 | 12 | – | – | 1 | – | 1 | – | 187 |
| Hepatitis, acute viral (NOS) | – | – | 5 | 4 | 198 | 14 | 1 | 2 | 8 | 5 | – | 3 | 28 | – | 7 | 20 | – | – | 295 |
| HIV infection | 56 | 14 | 139 | 16 | 24 | 11 | 15 | 5 | 2 | 15 | 15 | – | 2 | 2 | 1 | 1 | 5 | 269 | 611 |
| Hydatid disease | 1 | 1 | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | 2 |
| Legionnaires' disease | – | – | – | 5 | 6 | 2 | 5 | – | – | 2 | – | – | – | – | 1 | – | 1 | – | 22 |
| Leptospirosis | 1 | – | – | – | – | – | – | – | – | 9 | 1 | 3 | 4 | – | 4 | – | 3 | – | 25 |
| Listeria | 2 | 1 | – | – | – | – | – | – | – | 1 | – | – | – | – | – | – | – | – | 4 |
| Listeriosis | – | – | – | – | – | – | 2 | – | – | – | – | – | – | – | – | – | – | – | 2 |
| Malaria | 7 | 7 | 4 | 3 | 10 | 3 | 45 | 3 | 3 | 4 | 1 | 3 | – | – | 4 | 2 | 1 | – | 100 |
| Measles | 64 | 5 | 12 | 12 | 20 | 5 | 32 | 11 | 9 | 55 | 13 | 2 | 4 | – | 1 | 9 | – | – | 254 |
| Meningococcal meningitis | 2 | 3 | – | 11 | 1 | – | 1 | 1 | 2 | 5 | 1 | 2 | – | – | – | 1 | – | – | 30 |
| Meningococcal septicaemia | – | 1 | – | – | 1 | – | – | 1 | – | – | 4 | 2 | – | – | – | 1 | – | – | 10 |
| Meningococcal infection (NOS) | – | – | 4 | 3 | 3 | 1 | 2 | 3 | 5 | – | 1 | 5 | 2 | – | 2 | 1 | – | – | 32 |
| Mumps | – | – | – | – | 1 | – | 1 | – | – | – | – | – | – | – | 1 | – | – | – | 3 |
| Mycobacterial atypical | 20 | 19 | – | – | 1 | – | – | – | – | – | – | – | – | – | – | – | 3 | – | 43 |
| Mycobacterial tuberculosis | 22 | 15 | 36 | 21 | 8 | – | 7 | 2 | 7 | 13 | – | – | 3 | – | – | 1 | 1 | – | 136 |
| Mycobacterial infection (NOS) | – | – | 7 | 1 | 29 | 8 | 46 | – | 11 | – | 3 | 6 | 2 | 3 | 3 | 2 | – | – | 121 |
| Pertussis | – | 2 | 5 | 3 | 4 | 1 | 1 | – | – | 1 | 3 | 1 | 9 | – | 3 | 1 | – | – | 34 |
| Q Fever | – | 1 | – | 1 | – | 1 | – | – | – | 5 | 7 | 44 | 93 | 3 | 2 | 1 | – | – | 158 |
| Ross River fever | 1 | – | 4 | – | – | – | 4 | – | – | 1 | 11 | 71 | 23 | – | 6 | – | 7 | – | 128 |
| Rubella | – | 1 | 6 | – | 6 | 1 | 6 | 1 | 1 | 1 | – | – | – | – | 2 | – | – | – | 25 |
| Salmonella infection (NOS) | 67 | 90 | 74 | 112 | 124 | 62 | 72 | 1 | 39 | 18 | 63 | 61 | 63 | 14 | 22 | 11 | 18 | – | 911 |
| Syphilis | 36 | 12 | 33 | 47 | 30 | 7 | 26 | – | 6 | 16 | 64 | 17 | 116 | 3 | 14 | 1 | 3 | – | 431 |
| Tetanus | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | 2 | – | – | 2 |
| Typhoid & paratyphoid | 10 | 4 | 11 | – | 2 | – | 2 | – | 1 | 2 | – | 2 | – | – | – | – | 1 | – | 35 |

*Data from January to August only.

*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.