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# SALMONELLA BREDENEY OUTBREAK IN EASTERN STATES

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Since February 21, 1995, health authorities in eastern Australia have been notified of more than 100 cases of *Salmonella bredeney*. The outbreak, which is concentrated in the Australian Capital Territory and south-western Sydney, is being investigated by the NSW Public Health Network in co-operation with the Communicable Diseases Network of Australia and New Zealand. NSW is taking a leading role and co-ordinating the investigation. Although no source has been identified yet, a range of food has been sampled. The results of all food sampling has been negative.

ACT Health reported 11 cases of *S bredeney* on February 21. The Institute of Clinical Pathology and Medical Research at Westmead simultaneously reported 34 cases in NSW. Further notifications were received from February 23-27 and March 1-13. The Microbiological Diagnostic Unit, Melbourne University, where the National Salmonella Surveillance Scheme (NSSS) is administered, provided five additional notifications for NSW and four for the ACT. Three cases have been notified in Victoria and one in Queensland.

## BACKGROUND

Each year about 1,000 notifications of *Salmonella* infection are received in NSW, and more than 5,000 are received nationally. *S bredeney* is one of 200-300 different serovars of *Salmonella* human pathogens isolated in Australia.

There is no evidence linking this organism with a single animal or environmental reservoir. Recent food, animal and environmental isolations of *S bredeney* received by the NSSS and Australian Salmonella Reference Laboratory (ASRL) were from meat meal (Victoria and Tasmania), pork, sewage and sewage sludge (NSW), egg pulp, meat, chicken layer, chicken feed and porcine intestine (Queensland) and bovine intestine (South Australia).

The average number of notifications for *S bredeney* a year in NSW between 1988 and 1994 was six. The Australia-wide figure was 22 cases (Table 1).

**TABLE 1**

***SALMONELLA BREDENEY* NOTIFICATIONS (1988-1993 NATIONAL SALMONELLA SURVEILLANCE SCHEME DATA, 1994 NSW HEALTH DATA)**

| | Total | NSW | Vic | Qld | SA | WA | Tas | NT |
|---|---|---|---|---|---|---|---|---|
| 1994 | 20 | 2 | na | na | na | na | na | na |
| 1993 | 27 | 12 | 1 | 6 | 3 | 4 | – | 1 |
| 1992 | 27 | 9 | 8 | 4 | 1 | 4 | – | 1 |
| 1991 | 14 | 3 | 2 | 5 | – | 4 | – | – |
| 1990 | 30 | 4 | 1 | 8 | 9 | 2 | – | 6 |
| 1989 | 21 | 2 | 4 | 2 | 3 | 4 | – | 6 |
| 1988 | 14 | 4 | 5 | 3 | – | 2 | – | – |
| Mean | 21.86 | 6 | 4 | 5 | 3 | 3 | 0 | 2 |

na = not available

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## Salmonella bredeney *outbreak*

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### Cases

Eighty notifications of *S bredeney* have been received for NSW, and 24 for the ACT. Five of the NSW notifications were received in January, 67 in February and six in March. The dates of isolation are not known for two cases. NSW notifications peaked on February 20 (Figure 1) but are spread throughout that month. Notifications continued to be received in March. Because there can be a delay of two-three weeks between laboratory confirmation and notification, the outbreak may be continuing.

The largest number of notifications has been in the 0-5 age group (30 cases, 38 per cent). Seventeen of these 30 cases (57 per cent) were females. Of the total notifications, 44 (55 per cent) were for females (Figure 2). Age is not known for two male cases.

The places of residence of the cases notified were widespread (Figure 3), making a single point source unlikely. Place of residence is not known for one case. The largest number of cases (18) was from south-western Sydney. The 24 ACT cases were likely to have been associated with the NSW cases. Water, other than packed water, is unlikely to be implicated given the wide distribution and the number of different water supplies. The distribution of cases suggests a food widely available in NSW and the ACT but with limited distribution in other States.

### Investigation

All NSW cases are being interviewed using a questionnaire from the *Outbreak Management Plan for Foodborne Illness and Gastroenteritis in an Institution*. This questionnaire seeks information on food history before onset of illness, foods which the cases associated with their illness, and history relating to consumption or routine purchase of a range of high-risk foods. Information is also sought on the retail outlets from which cases obtain food and social functions they may have attended. Contact with animals, pet foods, home-grown vegetables and associated fertilisers and water supplies is also being investigated. Telephone or personal interviews are being undertaken by Public Health Units in consultation with notifying doctors.

Data obtained using this questionnaire have suggested associations with several possible foods. These foods are being investigated by food sampling, both at retail level, to obtain a range of "use by" dates, and at factory level.

Information on the distribution of these foods has been obtained to try to match the distribution of foods with the place of residence of cases.

Results of food sampling have been negative.

A case-control study controlling for age, sex and geographical location is being undertaken.

**FIGURE 1**

***SALMONELLA BREDENEY* NOTIFICATIONS IN NSW, 1995, BY DATE OF ISOLATION**

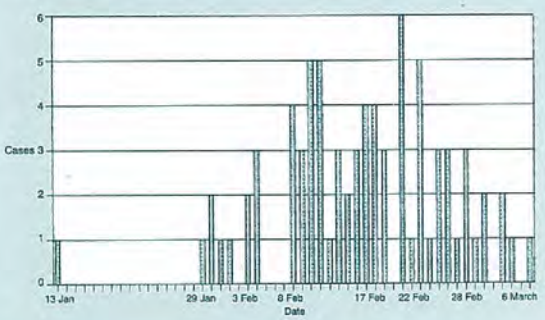



**FIGURE 2**

***SALMONELLA BREDENEY* NOTIFICATIONS IN NSW, 1995, BY AGE AND SEX**

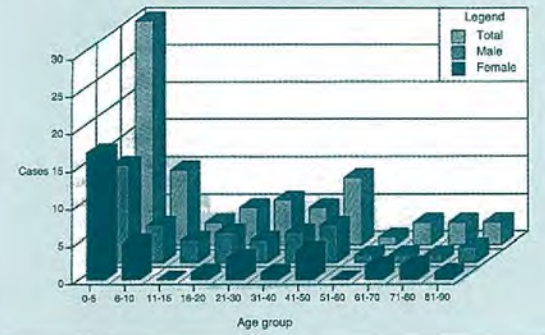
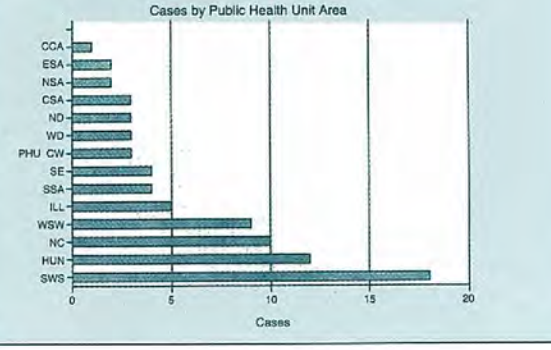



**FIGURE 3**

***SALMONELLA BREDENEY* NOTIFICATIONS IN NSW, 1995, BY PUBLIC HEALTH UNIT**



# TUBERCULOSIS SCREENING IN INMATES AND STAFF OF A NSW JAIL

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This article reports on a screening survey for tuberculosis (TB) infection in a NSW jail. Its objective was to determine the extent of TB infection and risk factors among inmates.

Prison populations have been identified as having an increased incidence of active TB in overseas studies[1]. The NSW Health Department has identified control of TB as an important public health issue[2]. A major part of the NSW TB control strategy is the identification of high-risk settings for TB transmission, appropriate monitoring of people in these settings and supervised treatment of cases.

Development of multi-drug resistant strains of *Mycobacterium tuberculosis* and concurrent HIV/TB infection has led to increased concern about TB and the need for implementation of control strategies[3].

Prison inmates and staff are a mobile population. Introduction of infectious diseases into the prison system can occur at any time. Inmates are frequently moved between jails and remain in detention for variable lengths of time, living in close proximity and sharing cells.

There is little information about the extent of exposure to *Mycobacterium tuberculosis* in the NSW prison system. A review of TB notifications in NSW undertaken in 1993 revealed no active cases in prison inmates. But no routine monitoring of TB infection has been conducted and there is a need to develop appropriate screening and TB management practices in prisons.

## METHODS

All inmates of Grafton Correctional Centre during June 1993 were invited to have Mantoux tuberculin tests. Prison staff were examined to assess their exposures. Tests were conducted on five days between June 15 and July 5, 1993. Reactions of 10mm or greater in people who had not had a BCG, and 15mm or greater in vaccinated people, on the third day after the test were regarded as positive[4]. Inmates were interviewed about BCG vaccination status. Those found to be Mantoux positive were offered chest x-rays. Medical notes of inmates were reviewed by staff of the Corrections Health Service for further relevant details.

All information was entered into a database (Epi-Info) for analysis. Individual associations between Mantoux positivity and age, length of time in jail, BCG status, Aboriginality, country of birth, and alcohol and injecting drug use were examined. Logistic regression modelling (SAS) was also used to assess the combined influence of these variables on Mantoux positivity.

## RESULTS

The prison's maximum capacity was 260 inmates and it had a full-time staff of 115. In all, 140 inmates (54 per cent) and 20 staff (17 per cent) were screened.

All but two of the screened inmates were male. The age range was 19-69 years (the mean age was 35, with a standard deviation of 9.7 years). Only three inmates were positively identified as Aboriginal, but Aboriginality was not specifically ascertained for a further 47 (34 per cent). Eighty-two inmates (59 per cent) reported that they were Australian-born, 14 (10 per cent) were known to be born overseas and country of origin was not ascertained for the remaining 44 (31 per cent).

Mantoux results were available for all but one of the inmates screened. Of the 140 inmates tested, 66 (47 per cent) had a reaction of at least 5mm and 47 (34 per cent) were defined as Mantoux positive (10+mm reactions, or 15+mm with previous BCG). Mantoux positivity was not related to age (Table 2).

**TABLE 2**

**MANTOUX TEST RESULTS BY AGE OF INMATES**

| Age (years) | No. of inmates | Positive Mantoux (%) |
|---|---|---|
| 15-24 | 20 | 3 (15%) |
| 25-29 | 18 | 9 (50%) |
| 30-34 | 31 | 12 (39%) |
| 35-39 | 30 | 9 (30%) |
| 40-44 | 17 | 6 (35%) |
| 45+ | 22 | 8 (36%) |
| Total | 138 | 47 (34%) |

NB: Mantoux result unknown for one inmate 40-44 years of age. Age unknown for one Mantoux negative inmate.

Twenty-two (27 per cent) of the 82 Australian-born inmates were Mantoux positive. Nine (64 per cent) of the 14 inmates born overseas were also positive, a significantly increased risk of positivity in overseas-born inmates (relative risk = 2.40; 95% CI 1.41 to 4.07). These nine inmates were born in New Zealand, Fiji, Kenya, Lebanon, Papua New Guinea, Sicily, Slovak Republic, Spain and the United States of America. Two of the three known Aboriginal inmates had positive results.

The length of time spent in jail was recorded for 70 inmates, and was not related to Mantoux positivity (Table 3).

**TABLE 3**

**TIME SPENT IN JAIL BY MANTOUX POSITIVITY**

| Time (years) | Mantoux result +ve | Mantoux result −ve | Total |
|---|---|---|---|
| 1 | 8 | 18 | 26 |
| 2-4 | 9 | 15 | 24 |
| 5-7 | 1 | 7 | 8 |
| 8+ | 5 | 7 | 12 |
| Not recorded | 24 | 44 | 68 |
| Total | 47 | 91 | 138 |

Only 15 (11 per cent) of the inmates screened reported having had a BCG vaccination. Seventy-seven (55 per cent) were known not to have been vaccinated and BCG status was unknown for a further 48 inmates (34 per cent). Of the 66 inmates with Mantoux reactions of at least 5mm, 10

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## Tuberculosis in a NSW jail

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(15 per cent) reported having had a BCG. Only five BCG positive inmates were regarded as having Mantoux positive (15+mm) reactions.

Ten (12 per cent) of the 82 Australian-born prisoners had been vaccinated with BCG. Sixty-eight (83 per cent) had not been vaccinated and four prisoners were of unknown BCG status. None of the Aboriginal prisoners had received a BCG. Of the 14 prisoners born overseas, four (29 per cent) were known to have been vaccinated.

All the 66 inmates with Mantoux reactions had chest x-rays. These showed clear lung fields in all but one inmate, who had evidence of past lung damage.

Certain other factors influencing health status were examined by interview or by medical chart review.

Thirty-three inmates (24 per cent) had a history of injecting drug use (IDU), 38 (27 per cent) were known not to inject drugs and IDU status was unknown for 69 (49 per cent). Six injecting drug users had received a BCG. Thirteen (39 per cent) had a positive Mantoux test, compared with 29 per cent of non-users.

Twenty-three inmates (16 per cent) had a medical history of alcohol use, while 50 (36 per cent) did not and alcohol involvement was unknown for 69 prisoners (49 per cent). Six inmates with a history of alcohol use had a positive Mantoux (26 per cent), compared with 36 per cent of non-users.

All inmates were screened for HIV infection on admission to the jail. None of the 140 prisoners screened was known to have HIV infection or to have an AIDS-defining condition.

Logistic regression modelling showed no significant combined influence of age, BCG status, Aboriginality, length of time in jail, place of birth, injecting drug use and alcohol use on Mantoux positivity.

Of the 20 staff examined, seven (35 per cent) were Mantoux positive (two female and five male officers). Both the female officers had 20-29mm Mantoux reactions. One of the male officers had a 10-14mm reaction, three had 15-19mm reactions and one had a 30+mm reaction. Five of the Mantoux positive officers had had BCGs; all five had 15+mm reactions. All seven officers with a positive Mantoux had clear chest x-rays, with no evidence of TB.

### Discussion

This survey examined the prevalence of TB infection (past and present), associated risk factors and BCG vaccination status in inmates and staff of a NSW jail. The survey was limited by the fact that only half the prisoners were screened and because data were missing on many items for screened inmates. Many of the inmates had been relocated or released before all relevant details were collected so, for some, information had to be gleaned from medical records. Furthermore, the screening was voluntary and it is unknown how representative study subjects were of the general prison population.

Despite these shortcomings, the survey revealed that one-third (34 per cent) of screened inmates had been infected with TB at some time in their lives. No information was available to indicate whether infection was atypical TB or not. No active TB cases were found on chest x-ray of Mantoux positive individuals. Fifty-three per cent of positive inmates were known not to have been vaccinated, indicating a high rate of exposure to infection. None of the three known Aboriginal inmates was vaccinated, and two of them were Mantoux positive. Of the 14 prisoners born overseas, nine (64 per cent) were Mantoux positive, and two-thirds were known not to have been vaccinated.

BCG vaccination was associated with Mantoux reactions (5+mm) but not with Mantoux positivity. Overseas origin was significantly associated with Mantoux positivity. There was no association observed with age, alcohol use, length of incarceration or HIV. Logistic regression modelling was inconclusive because of the small sample size and missing details.

Jail inmates often come from the lower socioeconomic strata, have low levels of education and are over-represented by minority groups. As such, their risk factors for TB infection are high. Despite this, no active cases of TB were found in this screening survey and no TB cases in inmates have been notified to the NSW Health Department since revised notification procedures were introduced in 1991[5]. The survey indicates that currently there is a low infection risk in the NSW prison system. This is very different from the situation observed in the US, where TB infection rates in inmates are more than three times higher than in the general population[6]. Although results of this survey are encouraging, the changing spectrum of TB infection – with the rise of drug-resistant TB strains and HIV/TB co-infection – creates a need for continued vigilance. The confined and ever-changing nature of the prison population increases the opportunity for infection, should diseases such as TB be introduced.

Given that groups at risk of TB are known to be over-represented in the prison population, there is an opportunity for routine screening and follow-up of inmates as a primary health care initiative. This approach would partially address screening recommendations made by the NSW TB Advisory Committee[1] and also satisfy occupational health and safety concerns of prison staff. It is important that any active TB cases going into the prison system are detected and reported promptly in order for effective public health response to occur. Diagnosis of active disease would precipitate tracing and screening of contacts within the prison population and the general community to prevent the risk of secondary spread.

### Acknowledgments

David Norris of Grafton Correctional Centre Corrections Health Service coordinated testing and data collection of inmates and staff. Dr Philip Brown, Chief Executive Officer, Corrections Health Service, provided critical comment on the paper.

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## LETTER TO THE EDITOR

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In the November 1993 edition of the *NSW Public Health Bulletin*, Corbett and Cowie referred to lead in petrol (Pb-P) and to children's blood lead (Pb-B) levels and recommended the increased use of unleaded petrol (ULP)[1]. When Pb-P is phased down the aromatic hydrocarbons in petrol are increased (Figure 4), resulting in higher air levels of carcinogenic benzene and other air toxics. In some countries this has increased the risks of leukaemias in the general community and occupationally. Therefore, it is not medically appropriate to consider lead only when changes are made to the composition of petrol.

### Lead

The *Bulletin* article stated that from 1976 to 1990 in the US, Pb-P phasedown was "associated with a significant reduction" in mean Pb-B levels[1]. As shown in Figure 5, Pb-Bs in the US have been declining and continue to decline independently of the increased use of Pb-P[2]. After the UK phasedown from 0.4 grams/litre to 0.15 grams/litre, tests showed that, although a 63 per cent decline in air lead levels (Pb-A) reduced Pb-B levels in those living near major roads, the data suggest that dietary lead intake, rather than Pb-A or leaded dust, was the "major contributor" to Pb-B levels[3].

By 1988 Sydney's Pb-As (Figure 6) were below the National Health and Medical Research Council (NHMRC) recommendation[4] of 1.5 µg/m³.

Corbett and Cowie expressed concern about raised Pb-B levels[1]. They did not state that the average level in a 1993 survey of 252 Melbourne children was only 5.4 µg/dL[5]. Other recent studies[6,7], not involving contaminated soil, have reported Pb-B levels similar to Victoria[5].

Reference was made[1] to a review by the South Australian Health Commission, tabulating Pb-B levels from 1975-1990[8], and an estimate that "45 per cent of preschool children have Pb-B levels about 10 µg/dL"[9]. The commission referred to the limitations of its estimates and stressed the need for caution. The findings of the commission's review – in which more than two-thirds of the surveys were taken from industrial or contaminated sites[8] – are not applicable to current situations. When discussing the "IQ deficit" caused from Pb-B levels, the *Bulletin* article should have stated that Pb-B levels relate to group averages and therefore cannot be applied to individual Pb-B level[10].

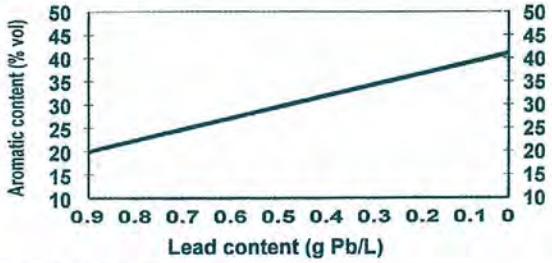
Corbett and Cowie stated that a NSW cost-benefit analysis was being prepared incorporating "loss of IQ"[1]. The US Environmental Protection Agency (EPA) 1989 statistical lead uptake model[11] used in many Australian calculations and cost-benefit analyses has been superseded[12]. The validity of Australian estimates[9] must be re-examined in light of this revision and should include estimates of petrol-related cancers. In California the cost per "case avoided ranges from $22 to $40 million"[13].

### Aromatic hydrocarbons

As lead is phased down, aromatic hydrocarbons are usually increased[2]. During the combustion process benzene and other carcinogens are produced in the exhaust gases.

**FIGURE 4**

**AUSTRALIAN REFINERIES**
**AROMATIC CONTENT INCREASES AS LEAD IS PHASED OUT**




Source: Octel Gasoline Surveys 1974-1993

**FIGURE 5**

**TRENDS IN LEAD IN BLOOD AND LEAD IN PETROL 1930-90**

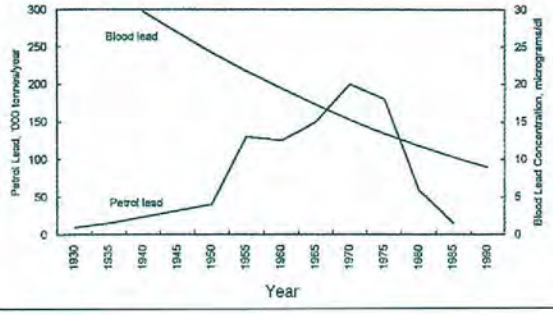
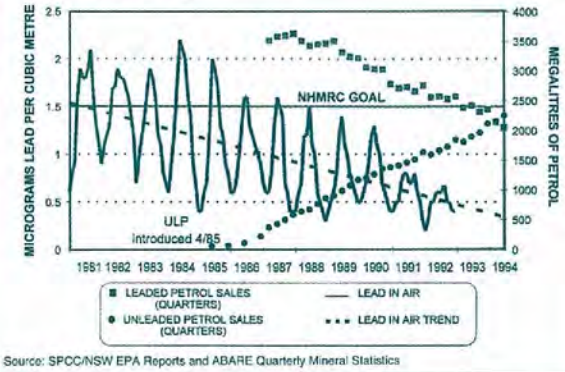



**FIGURE 6**

**LEAD IN AIR TRENDS – SYDNEY SUBURBS (90 DAY AVERAGES)**




Source: SPCC/NSW EPA Reports and ABARE Quarterly Mineral Statistics

Benzene does not have to be present in petrol for benzene to be produced[14]. Benzene, toluene and xylenes are carcinogenic[15]. In 1993 the NHMRC recommended that substantial octane enhancement of fuels should not be achieved by using carcinogenic additives[16].

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There are worrying reports about the possibilities of leukaemias in children partly related to travelling in cars[17], of breast cancers from aromatics[18,19] and, as estimated by an Italian Government committee, possibly large numbers of additional leukaemias and lung cancers[20]. There are American estimates of additional cancers from benzene and 1,3-butadiene from vehicle emissions[21]. Previous Australian occupational exposures to total hydrocarbons and benzene may have caused haematological malignancies[22].

Associated Octel commissioned a university to determine air levels of benzene and toluene in Sydney's Central Business District (CBD). The benzene results are shown in Table 4.

**TABLE 4**

**BENZENE IN AIR (PARTS PER BILLION – PPB)**

| Period | Daily average | Peak | % of "time" ≥5 |
|---|---|---|---|
| 15/1/94 to 14/2/94 | 4.1 | 10.2 | 27 |
| 25/6/94 to 14/7/94 | 7.6 | 25.9 | 87 |

Toluene air levels were much higher[23].

A British expert panel recommended an air quality standard (running annual average) of 5 ppb of benzene which later should be reduced to 1 ppb[24]. Thus Sydney's CBD measurements should be cause for concern.

In 1994 the Federazione Nazionale Pro Natura requested the Commission of the European Communities (CEC) to initiate proceedings against the Italian Government which "encourages the use of high aromatic and unleaded fuels even in cars not equipped with catalysts", showing "a complete disregard for health". It also requested the CEC urgently "to authorise a temporary return to 0.3 g Pb/L maximum level for lead (in low-leaded petrol) in the event that this should be the only practical way to reduce the aromatic content of petrol below 30-32 per cent by weight, and to ban the use of unleaded petrol with a high aromatic content in cars"[25] with no catalysts.

As lead has been phased out, fuels have become more polluting[21]. The NRMA has stated that "if the oil industry cannot reduce lead without increasing benzene, the timing of this stage of the [phasedown] program should be reviewed"[26].

Because only 40 per cent of Australian vehicles have catalysts and because the annual level of benzene in air and some other carcinogens are not known, a moratorium on promoting unleaded petrol for pre-1986 cars should be implemented. At the current switch-over rates, the use of Pb-P will be eliminated by 2007.

Clearly, protecting health is not as simple as only taking the lead out of petrol.

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## AUTHORS' REPLY

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We would like to offer the following comments in reply to Dr Bell's letter about our article[1] on lead in petrol.

Dr Bell correctly raises concerns about the possible health hazards of using benzene or other polycyclic aromatic

hydrocarbons (PAHs) as alternatives to lead in petrol. There is concern in Italy and in some other European countries that the use of these alternative octane boosters will increase ambient benzene levels and may lead to an increased incidence of some cancers, particularly leukaemia.

We believe that the reduction of lead in petrol in Europe is not directly comparable to the Australian situation. Dr Bell has ignored the issue of octane rating, which is critical to his debate. Octane rating is a measure of the compression of the petrol-air mixture in a car engine: a higher octane rating ensures higher compression and greater engine efficiency. Lead or aromatic compounds can be added to petrol to increase octane rating. In Australia, unleaded petrol has a specified Research Octane Number (RON) of 91 and leaded petrol has a RON of 97. In Europe the RON of unleaded petrol is 95 and 98.

In Europe the removal of lead from petrol was accompanied by the addition of benzene and other aromatic hydrocarbons to achieve the higher octane rating for unleaded petrol. Figure 4 in Dr Bell's letter illustrates the increase in the volume of aromatic compounds used when lead in petrol is reduced. This graph is not relevant in the Australian context. In Australia, we are seeking to lower lead in petrol while at the same time lowering octane demand, thereby avoiding the use of alternative additives to petrol. Figure 7, provided by the Australian Institute of Petroleum (AIP), indicates that aromatic and olefin (a hydrocarbon) content of unleaded petrol has, if anything, fallen slightly over the period 1992-1994.

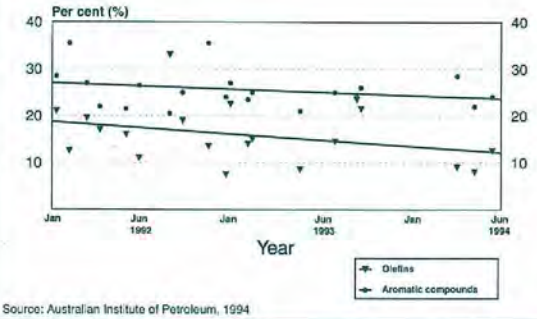
When the lead level in leaded petrol in NSW was reduced from 0.4 to 0.3 g/L in February 1994, octane rating was also decreased from 97 to 96 RON. Further reductions to lead in petrol to 0.02 g/L occurred in late 1994. Benzene content in petrol varies depending on the fuel batch, because of variations in the crude fuel stock and blending compositions. However, the AIP[2] advises that there will be little or no noticeable increase in benzene content of petrol with lead at 0.2 g/L. Reducing the lead in petrol while maintaining octane rating requires changes to refinery operations which can be achieved at cost to the refinery. One Australian refinery has been able to produce petrol with a lead content of 0.15 g/L by improving its refining technologies[3].

In Australia the average level of benzene in both leaded petrol (LP) and unleaded petrol (ULP) is between 2.6 per cent and 2.8 per cent[2]. Although premium unleaded petrol (PULP) has a much higher benzene content (4 per cent) than ULP or LP, the large price differential (PULP costs up to 8-15 cents/litre more) ensures there is little incentive to use PULP in pre-catalyst vehicles. This is reflected in the very small proportion (1 per cent) that PULP contributes to total sales of petrol in Australia. Furthermore, spot measurements of petrol taken by Octel indicate that the total aromatic contents of LP (20-33 per cent) and ULP (24-34 per cent) are similar[4].

Overseas experts have claimed[5] that ULP should not be used in cars without catalytic converters. Dr Bell has upheld this claim in his letter. In Europe ULP with a much higher aromatic content was introduced without the requirement for catalytic converters. In Australia, the introduction of ULP in 1986 was accompanied by the requirement for all new vehicles to be fitted with emission reduction equipment. Vehicle manufacturers achieved this by the use of catalytic converters.

**FIGURE 7**

**OLEFIN AND AROMATIC CONTENT OF ULP FROM NSW REFINERIES**




Source: Australian Institute of Petroleum, 1994

For the above reasons and because aromatic contents of LP and ULP are similar, a switch-over to ULP for the pre-1986 vehicles which can operate on ULP is not expected to increase emissions of benzene or other aromatics in Australia, irrespective of the presence or absence of a catalytic converter.

There has been limited air monitoring of ambient benzene levels in Sydney. The levels quoted by Dr Bell are based on a limited number of samples collected from George Street, one of Sydney CBD's busiest streets. The NSW Environment Protection Authority will shortly begin a pilot air toxics monitoring program including benzene and other volatile organic compounds. Preliminary monitoring by the EPA has shown that ambient benzene levels are unlikely to be above 1 part per billion in most of metropolitan Sydney.

Benzene is an acknowledged cause of leukaemia. Although exposure to ambient benzene levels is hypothesised to be associated with increased levels of childhood leukaemia, further sophisticated studies are required to determine whether this association is causal.

Dr Bell challenges the information provided in our article which discusses comparative rates of decline of lead in petrol in the US and Australia and the mean blood lead in the US population. Figure 5 in Dr Bell's article is very misleading. The repeated surveys on which it is based are a mixture of occupational and population groups and are not comparable. The National Health & Nutrition Examination Surveys (NHANES) data which we quoted were based on repeated random surveys.

There have been drastic decreases in blood lead in the US[6]. Data from the US indicate there was a decrease of 77 per cent in blood lead levels of non-Hispanic white children aged 1-5 years from 13.7 µg/dL in 1976 to about 3.2 µg/dL in 1991; and a 72 per cent decrease in blood lead levels of non-Hispanic black children from 20.2 to 5.6 µg/dL. The change

*Continued on page 24 ▶*

► Continued from page 23

was attributed to the removal of 99.8 per cent of lead from fuel and the removal of lead from soldered cans.

There is little doubt that blood lead levels have been decreasing over the past few decades. This is due to the significant decline of lead in food through the virtual elimination of lead soldered cans, decreasing air lead levels, and the banning of lead-based paint.

The association of blood lead with decreasing cognitive development has been measured on a population basis. Although the effect of an average loss of 2-3 IQ points may be difficult to detect in an individual, its consequence on the general child population is considered to be significant. The proportion of children with very high IQs will fall, while the proportion of children with low IQs requiring remedial teaching will increase.

Dr Bell asserts that the review of blood lead data made by the South Australian Health Commission[7] stressed the need for caution in interpretation of its results. In our article we agreed that the prevalence of children with blood lead levels above 10 µg/dL was likely to be lower than 45 per cent. Recent NSW studies of children living in non-point source areas have found slightly higher mean blood lead levels: 7.5 µg/dL in Eastern Sydney[8] with 12.6 per cent above 10 µg/dL, and a mean of 11.4 µg/dL in the inner western suburbs of Sydney[9] with about 50 per cent of children with a blood lead level above 10 µg/dL. An opportunistic survey[10] of blood lead levels in a paediatric population in Newcastle found a mean blood lead level of 5.9 µg/dL, with blood lead levels 1.4 µg/dL higher in the inner city compared with non-metropolitan areas.

Despite the fall in blood lead levels, recent surveys indicate there is still a significant number of children with blood lead levels above the national goal for blood lead of 10 µg/dL. US data indicate that even further reductions in blood lead can be achieved. This is important as there is yet no evidence of a threshold level of lead below which no effects occur.

We maintain that it would be irresponsible to allow continued dispersion of lead from a known source, while alternatives are available. However, if it is decided that additives to petrol other than lead are required to sustain octane demand, we strongly echo the NHMRC's statement that "such enhancement should not be achieved by the use of known carcinogenic additives"[11].

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3. Shell Australia Ltd. Shell introduces half-lead petrol (media release). July 1994.
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6. Pirkle JL, Brody DJ, Gunter EW et al. The decline in blood lead levels in the United States: The National Health and Nutrition Examination Surveys (NHANES). *JAMA* 1994; 272(4):284-291.
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10. Aldrich RA, Toneguzzi R, Wlodarzyk J et al. Opportunistic blood lead testing in a paediatric in-patient population. Presented at the International Epidemiology Association Conference, Sydney University, November 1993.
11. National Health & Medical Research Council (NHMRC). Carcinogenic additivies in low and unleaded petrol. November 1993.

***Correspondence on this issue is now closed – Editor.***

## CHANGES IN PUBLIC HEALTH UNITS

There have been several changes in the State's Public Health Units over recent months. The Southern Sydney and Central Sydney Public Health Units have been established as separate entities. Dr Jeremy McAnulty has been appointed Director of the Southern Sydney PHU, while Dr Mark Bek continues as Acting Director of the Central Sydney PHU. Dr Peter Lewis has been appointed Director of the Central Coast PHU, freeing Dr Rod Kennedy for other duties in the Central Coast Area Health Service. Ms Christine Robertson has been acting as Director of the Northern Districts PHU in Tamworth while Dr John Rooney serves as Director of the Centre for Health Promotion and Disease Prevention at the NSW Health Department in Sydney. Dr Michael Douglas has succeeded Dr John Hall in the Western NSW PHU in Dubbo and Dr Paul van Buynder is the Director of the South Eastern PHU in Goulburn. Table 5 (opposite) gives updated contact information on all Public Health Units.

## PUBLIC HEALTH EDITORIAL STAFF

The editor of the Public Health Bulletin is Dr Michael Frommer, Director, Research and Development, NSW Health Department.

The Bulletin aims to provide its readers with population health data and information to motivate effective public health action. Articles, news and comments should be 1,000 words or less in length and include a summary of the key points to be made in the first paragraph. References should be set out using the Vancouver style, the full text of which can be found in *British Medical Journal* 1988; 296:401-5.

Please submit items in hard copy and on diskette, preferably using WordPerfect 5.1, to the editor, NSW Public Health Bulletin, Locked Mail Bag 961, North Sydney 2059. Facsimile (02) 391 9029.

Please contact your local Public Health Unit to obtain copies of the NSW Public Health Bulletin.

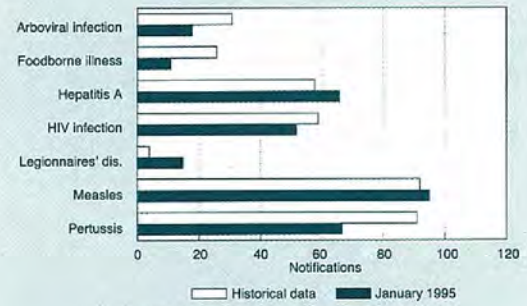
**TABLE 5**

| Public Health Unit | Director | Telephone | Facsimile | Street Address | Postal Address |
|---|---|---|---|---|---|
| Central Coast | Dr Peter Lewis | 043 20 4545 | 043 20 4550 | "Birralee", Wyoming Hospital<br>Cnr Pacific Highway and Kinarra Avenue<br>Wyoming NSW 2250 | "Birralee"<br>PO Box 172W<br>Gosford NSW 2250 |
| Central Sydney | Dr Mark Bek<br>(Acting Director) | 02 550 6810 | 02 565 1690 | Level 6 West<br>Queen Mary Building<br>Grose Street<br>Camperdown NSW 2050 | PO Box 374<br>Camperdown NSW 2050 |
| Southern Sydney | Dr Jeremy McAnulty | 02 350 3377 | 02 350 3474 | Level 1, James Laws House<br>St George Hospital<br>Kogarah NSW 2217 | PO Box 482<br>Kogarah NSW 2217 |
| Central Western | Dr Dan Russell | 063 32 8505 | 063 32 8577 | Webb's Chambers, 175 George Street<br>Bathurst NSW 2795 | PO Box 143<br>Bathurst NSW 2795 |
| Eastern Sydney | Dr Mark Ferson | 02 313 8322 | 02 313 6291 | Royal South Sydney Hospital<br>Ground Floor, Esme Cahill Building<br>Joynton Avenue<br>Zetland NSW 2017 | Locked Bag 88<br>Randwick NSW 2031 |
| Hunter | Dr John Stephenson | 049 29 1292 | 049 29 4037 | Irene Hall<br>Pacific Highway<br>Newcastle NSW 2300 | PO Box 11A<br>Newcastle NSW 2300 |
| Illawarra | Dr Victoria Westley-Wise<br>(Acting) | 042 26 4677 | 042 26 4917 | 18 Madoline Street<br>Gwynneville NSW 2500 | PO Box 66<br>Keiraville NSW 2500 |
| Northern Districts | Ms Christine Robertson<br>(Acting) | 067 66 2288 | 067 66 3003 | Suite 7, Second Floor, Parry Shire Building<br>470 Peel Street<br>Tamworth NSW 2340 | PO Box 597<br>Tamworth NSW 2340 |
| North Coast | Dr John Beard | 066 21 7231 | 066 22 2151 | No. 31 Uralba Street<br>Lismore NSW 2480 | PO Box 498<br>Lismore NSW 2480 |
| Northern Sydney | Dr Don Holt | 02 477 9400 | 02 482 1650 | c/- Hornsby Ku-ring-gai Hospital<br>Palmerston Road<br>Hornsby NSW 2077 | Same as street address |
| Western NSW | Dr Michael Douglas | 068 81 2235 | 068 84 7223 | 62 Windsor Parade<br>Dubbo NSW 2830 | PO Box M61<br>East Dubbo 2830 |
| South Eastern | Dr Paul Van Buynder | 048 27 3432 | 048 27 3438 | Kenmore Hospital, Taralga Road<br>Goulburn NSW 2580 | PO Box 300<br>Goulburn NSW 2580 |
| South West | Mr Tony Kolbe | 060 23 0350 | 060 23 0168 | 475 Townsend Street<br>Albury NSW 2640 | PO Box 503<br>Albury NSW 2640 |
| South Western Sydney | Dr Greg Stewart | 02 828 5944 | 02 828 5955 | Hugh Jardine Building<br>c/- Liverpool Hospital<br>Liverpool NSW 2170 | Private Bag 17<br>Liverpool NSW 2170 |
| Western Sydney<br>and Wentworth | Dr Anthony Capon | 02 840 3603 | 02 840 3608 | 13 New Street<br>North Parramatta NSW 2151 | Same as street address |

# INFECTIOUS DISEASES

**FIGURE 8**

**SELECTED INFECTIOUS DISEASES JANUARY 1995 AND HISTORICAL DATA**




(Average January notifications 1992-1994)

**TABLE 6**

**SUMMARY OF NSW INFECTIOUS DISEASE NOTIFICATIONS FEBRUARY 1995**

| Condition | Number of cases notified | | | |
|---|---|---|---|---|
| | Period | | Cumulative | |
| | Feb 1994 | Feb 1995 | Feb 1994 | Feb 1995 |
| Adverse reaction | 5 | 1 | 9 | 3 |
| AIDS | 47 | 3 | 85 | 18 |
| Arboviral infection | 70 | 11 | 96 | 29 |
| Brucellosis | – | – | – | – |
| Cholera | – | – | – | – |
| Diphtheria | – | – | – | – |
| Foodborne illness (NOS) | 8 | 21 | 29 | 32 |
| Gastroenteritis (instit.) | 11 | – | 12 | 2 |
| Gonorrhoea | 26 | 17 | 63 | 42 |
| H influenzae epiglottitis | 1 | – | 3 | – |
| H influenzae B – meningitis | – | – | 1 | 2 |
| H influenzae B – septicaemia | 1 | 1 | 2 | 1 |
| H influenzae infection (NOS) | 1 | – | 3 | – |
| Hepatitis A | 53 | 22 | 104 | 88 |
| Hepatitis B | 326 | 126 | 673 | 485 |
| Hepatitis C | 780 | 241 | 1,389 | 894 |
| Hepatitis D | 2 | – | 3 | 1 |
| Hepatitis, acute viral (NOS) | 1 | – | 2 | 19 |
| HIV infection | 47 | 43 | 85 | 95 |
| Hydatid disease | 2 | – | 2 | – |
| Legionnaires' disease | 5 | 4 | 8 | 19 |
| Leprosy | – | – | – | – |
| Leptospirosis | 2 | – | 4 | 1 |
| Listeriosis | 2 | 3 | 4 | 3 |
| Malaria | 25 | – | 51 | 9 |
| Measles | 70 | 28 | 226 | 123 |
| Meningococcal meningitis | 3 | 5 | 8 | 7 |
| Meningococcal septicaemia | 2 | 4 | 3 | 5 |
| Meningococcal infection (NOS) | – | 2 | 1 | 5 |
| Mumps | – | – | 1 | 2 |
| Mycobacterial tuberculosis | 27 | 3 | 86 | 24 |
| Mycobacterial – atypical | 40 | 1 | 90 | 11 |
| Mycobacterial infection (NOS) | 4 | 1 | 5 | 9 |
| Pertussis | 133 | 25 | 320 | 92 |
| Plague | – | – | – | – |
| Poliomyelitis | – | – | – | – |
| Q fever | 23 | 5 | 50 | 24 |
| Rubella | 13 | 6 | 34 | 14 |
| Salmonella infection (NOS) | 135 | 70 | 246 | 200 |
| Syphilis | 92 | 19 | 185 | 100 |
| Tetanus | – | – | – | – |
| Typhoid and paratyphoid | 6 | 4 | 7 | 7 |
| Typhus | – | – | – | – |
| Viral haemorrhagic fevers | – | – | – | – |
| Yellow fever | – | – | – | – |

## AMENDMENTS TO NOTIFICATION REQUIREMENTS

Three amendments have been made to the Public Health Regulation 1991. From March 17, 1995:

- rabies is notifiable by laboratories and hospital Chief Executive Officers;
- typhoid may give rise to a public health order; and
- leprosy can no longer give rise to a public health order.

Under Section 21-36 of the Public Health Act 1991 a public health order may be served on a person who has tuberculosis, typhoid (as of March 17) or HIV/AIDS, and is behaving in a way that may endanger the health of the public. The order may require a person to have a medical examination, undergo treatment, refrain from certain conduct, undergo counselling, submit to supervision or be detained while having treatment. In the case of HIV or AIDS, a person may be detained while the order is in force. Only the Chief Health Officer or a medical practitioner specifically authorised by the Director-General can issue a public health order. Orders are issued only in extreme circumstances when all other approaches have failed.

## NEW REPORTING FORMAT

A new feature has been introduced this month to compare recent numbers of notifications with historical data for selected diseases (see figure 8). In this context the term "historical data" refers to the average number of notifications for a particular condition notified in the same month in the previous three years. The month selected is one month before the cut-off date for other infectious disease tables. Thus, in this issue of the *Public Health Bulletin*, the tables display data up to the end of February, while the new figure displays data for January only. Delays in receiving and entering notifications result in the latest month's figures always being an underestimate, so comparisons with historical data would not be appropriate.

## LEGIONNAIRES' DISEASE

Notifications of legionnaires' disease are considerably higher in 1995 compared with the same period in previous years (Figure 8, Table 6). This is due to the cluster in Western Sydney Area (WSA) in January 1995, reported in last month's issue of the *Bulletin*. WSA Public Health Unit (PHU) has reported 15 cases with onset in January, but none in February.

## HIV

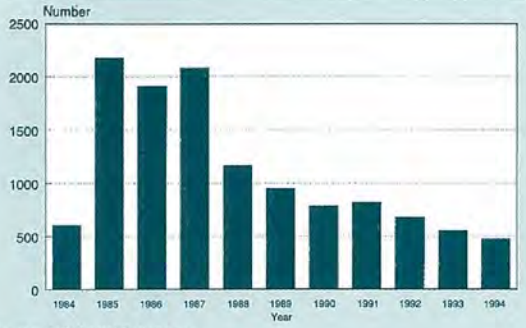
The HIV notification rate this year (1.53/100,000 population) is higher than for the same months in 1994 (1.37/100,000, Table 6), but slightly lower than the average over the past three years (Figure 8). The annual NSW notification rate for HIV has decreased almost every year since testing began in 1985 (see Figure 9). The number of notifications in January and February may diminish later when more information from callback to doctors is received, as cases discovered to have tested positive previously are "denotified". It is too early to determine whether the decline in notification rates in recent years is continuing.

## HEPATITIS A

Central West Public Health Unit reported the highest hepatitis A notification rate so far in 1995 with 29.2/100,000 population for the Evans District. This small outbreak was reported in last month's *Bulletin*. PHU staff are continuing to deal with possible causative factors.

**FIGURE 9**

**HIV NOTIFICATIONS NSW, 1984-1994**




Source: NSW HIV Database

Eastern Sydney had the next highest notification rate with 7.0/100,000 population, compared to the Statewide figure of 1.4/100,000. An outbreak originally among injecting drug users in Eastern and Central Sydney was reported previously. As most transmission is through the person-to-person route, hepatitis A outbreaks are difficult to control, typically evolving slowly and continuing for many months. PHU staff continue to follow up every case and have organised training for council employees whose work is related to food inspection or environmental health.

## GONOCOCCAL SENSITIVITY SURVEILLANCE, OCTOBER-DECEMBER 1994

The following information is based on information from the Gonococcal Reference Laboratory, Prince of Wales Hospital.

There were 111 gonococci isolated and referred in this quarter. Of these, 106 remained viable for further examination.

### Antibiotic sensitivity patterns

Thirty-nine isolates (36.8 per cent) were penicillin-resistant, either by virtue of lactamase production (PPNG) (17 strains – 16 per cent) or as a consequence of chromosomally mediated mechanisms (22 strains – 20.8 per cent).

The high proportion of isolates resistant to the penicillins means this group of antibiotics is inappropriate for therapy of gonorrhoea in NSW.

*Ceftriaxone*

All isolates examined were sensitive to this antibiotic. This injectable cephalosporin is very active against gonococci. An oral third-generation cephalosporin has been released in Australia and the likely outcome of therapy with this antibiotic can be inferred from in vitro sensitivity data on ceftriaxone.

*Specinomycin*

All strains tested were susceptible in vitro to this injectable antibiotic.

*Quinolones (Ciprofloxacin)*

Seven isolates (7 per cent) had some level of resistance to the oral quinolone antibiotics. These strains have been present in low numbers in NSW since 1984. The recommended treatment regimen of a single dose of 500mg of ciprofloxacin is adequate to deal with almost all isolates, including those with the levels of chromosomal resistance usually encountered. There is no plasmid-mediated resistance to quinolone antibiotics.

However, in this quarter, three isolates from two patients had high levels of quinolone resistance. Patients infected with these types of gonococci usually do not respond to quinolone therapy.

World Health Organisation sources indicate that quinolone resistance is increasing rapidly in countries visited frequently by Australians. Continued monitoring of resistance in this group of antibiotics is essential, especially in patients entering or returning to Australia. Strains from apparent treatment failures warrant close examination.

*Tetracyclines*

Tetracyclines are NOT recommended for treatment of gonorrhoea in NSW. Some of the chromosomal mechanisms that increase resistance to the penicillins at the same time increase resistance to the tetracycline group. Additionally, tetracyclines are not suitable for single-dose therapy. For these reasons isolates are not routinely tested for chromosomal resistance to the tetracyclines.

**TABLE 7**

**INFECTIOUS DISEASE NOTIFICATIONS FOR 1994/1995 BY SELECTED MONTH OF ONSET FOR NOTIFICATIONS RECEIVED BY FEBRUARY 28, 1995**

| Condition | Nov | Dec | Jan | Feb |
|---|---|---|---|---|
| Adverse event after immunisation | 5 | 5 | 2 | 1 |
| AIDS | 30 | 18 | 13 | 3 |
| Arboviral infection | 8 | 7 | 18 | 11 |
| Foodborne illness (NOS) | 58 | 7 | 11 | 21 |
| Gastroenteritis (instit.) | 8 | 31 | 2 | – |
| Gonorrhoea infection | 25 | 29 | 25 | 17 |
| H influenzae septicaemia | – | 1 | – | 1 |
| H influenzae meningitis | 2 | 1 | 2 | – |
| H influenzae infection (NOS) | – | 2 | – | – |
| Hepatitis A – acute viral | 79 | 39 | 66 | 22 |
| Hepatitis B – acute viral | 5 | 3 | 1 | 1 |
| Hepatitis B – chronic/carrier | 45 | 29 | 42 | 9 |
| Hepatitis B – unspecified | 407 | 281 | 316 | 116 |
| Hepatitis C – acute viral | 5 | 1 | – | 2 |
| Hepatitis C – unspecified | 886 | 519 | 653 | 239 |
| Hepatitis D | – | – | 1 | – |
| HIV infection | 44 | 29 | 52 | 43 |
| Hydatid disease | 4 | 1 | – | – |
| Legionnaires' disease | – | 2 | 15 | 4 |
| Leptospirosis | – | 1 | 1 | – |
| Listeriosis | 1 | 2 | – | 3 |
| Malaria | 8 | 14 | 9 | – |
| Measles | 342 | 260 | 95 | 28 |
| Meningococcal infection (NOS) | 2 | 2 | 3 | 2 |
| Meningococcal meningitis | 5 | 5 | 2 | 5 |
| Meningococcal septicaemia | 3 | 3 | 1 | 4 |
| Mumps | – | 1 | 2 | – |
| Mycobacterial atypical | 15 | 11 | 10 | 1 |
| Mycobacterial infection (NOS) | 15 | 10 | 8 | 1 |
| Mycobacterial tuberculosis | 18 | 16 | 21 | 3 |
| Pertussis | 95 | 66 | 67 | 25 |
| Q fever | 23 | 19 | 19 | 5 |
| Rubella | 7 | 7 | 8 | 6 |
| Salmonella (NOS) | 72 | 88 | 130 | 70 |
| Syphilis infection | 77 | 47 | 81 | 19 |
| Typhoid and paratyphoid | 1 | – | 3 | 4 |

## TABLE 8

**INFECTIOUS DISEASE NOTIFICATIONS FOR 1995 BY PUBLIC HEALTH UNIT, RECEIVED BY FEBRUARY 28, 1995**

| Condition | CSA | SSA | ESA | SWS | WSA | WEN | NSA | CCA | ILL | HUN | NC | ND | WN | CW | SW | SE | U/K | Total |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| AIDS | 4 | 5 | 3 | – | 1 | – | 1 | – | – | 1 | 3 | – | – | – | – | – | – | 18 |
| Arboviral infection | – | – | 1 | – | – | – | – | 1 | 1 | 2 | 13 | 5 | 2 | – | 2 | 2 | – | 29 |
| Gonorrhoea infection | 3 | 1 | 20 | – | 4 | 1 | – | 1 | 1 | 2 | 2 | 2 | 2 | – | – | 3 | – | 42 |
| Hepatitis B – acute viral | – | 1 | – | – | – | – | – | – | – | – | – | 1 | – | – | – | – | – | 2 |
| Hepatitis B – chronic/carrier | – | 1 | 35 | – | 8 | 2 | – | – | – | – | – | 3 | 1 | 1 | – | – | – | 51 |
| Hepatitis B – unspecified | 40 | 65 | 2 | 175 | 53 | 2 | 62 | 1 | 6 | 9 | 3 | 2 | 2 | 3 | 4 | 3 | – | 432 |
| Hepatitis C – acute viral | – | – | – | – | 1 | – | – | – | – | – | – | – | 1 | – | – | – | – | 2 |
| Hepatitis C – unspecified | 77 | 62 | 161 | 96 | 70 | 26 | 85 | 1 | 25 | 67 | 89 | 17 | 4 | 33 | 41 | 38 | – | 892 |
| Hepatitis D – unspecified | – | – | – | 1 | – | – | – | – | – | – | – | – | – | – | – | – | – | 1 |
| HIV infection | 13 | 1 | 27 | 5 | 1 | – | 1 | 2 | 2 | – | 3 | – | – | – | – | – | 40 | 95 |
| Legionnaires' disease | – | – | 1 | – | 12 | – | 3 | – | 1 | 2 | – | – | – | – | – | – | – | 19 |
| Leptospirosis | – | – | – | – | – | – | – | – | – | 1 | – | – | – | – | – | – | – | 1 |
| Malaria | – | – | 1 | 1 | 2 | 1 | 1 | 2 | – | – | 1 | – | – | – | – | – | – | 9 |
| Meningococcal infection | – | – | 1 | 1 | – | – | – | – | – | 2 | 1 | – | – | – | – | – | – | 5 |
| Meningococcal meningitis | 1 | 2 | – | 1 | – | – | – | – | 1 | 1 | 1 | – | – | – | – | – | – | 7 |
| Meningococcal septicaemia | 1 | – | – | – | – | – | – | – | – | 4 | – | – | – | – | – | – | – | 5 |
| Mycobacterial atypical | 1 | – | – | 4 | – | 1 | – | – | – | 4 | 1 | – | – | – | – | – | – | 11 |
| Mycobacterial infection (NOS) | 1 | – | – | – | – | – | 6 | – | – | 2 | – | – | – | – | – | – | – | 9 |
| Mycobacterial tuberculosis | 1 | 11 | 1 | 1 | 3 | – | 4 | 1 | – | 1 | – | – | – | – | – | 1 | – | 24 |
| Q fever | – | – | – | – | 1 | – | – | – | 1 | 2 | 1 | 7 | 11 | 1 | – | – | – | 24 |
| Syphilis infection | 12 | 5 | 27 | 19 | 7 | 2 | 2 | 1 | 2 | 3 | 1 | 9 | 5 | 4 | – | – | – | 100 |

## TABLE 9

**VACCINE PREVENTABLE AND RELATED CONDITIONS, NOTIFICATIONS FOR 1995 BY PUBLIC HEALTH UNIT, RECEIVED BY FEBRUARY 28, 1995**

| Condition | CSA | SSA | ESA | SWS | WSA | WEN | NSA | CCA | ILL | HUN | NC | ND | WN | CW | SW | SE | Total |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Adverse event after immunisation | – | – | – | – | – | – | – | – | – | – | – | – | – | – | 3 | – | 3 |
| H. influenzae meningitis | 1 | – | – | – | – | – | – | – | – | – | 1 | – | – | – | – | – | 2 |
| H. influenzae septicaemia | – | 1 | – | – | – | – | – | – | – | – | – | – | – | – | – | – | 1 |
| Measles | 6 | 8 | 13 | 7 | 13 | 10 | 1 | 4 | 18 | 17 | 2 | 16 | – | 1 | 3 | 4 | 123 |
| Mumps | – | – | 1 | – | 1 | – | – | – | – | – | – | – | – | – | – | – | 2 |
| Pertussis | 3 | 3 | 4 | 4 | 4 | 8 | 11 | 4 | 7 | 4 | 18 | 1 | 2 | 5 | 11 | 3 | 92 |
| Rubella | – | 3 | – | – | 5 | 3 | 1 | – | – | 1 | – | 1 | – | – | – | – | 14 |

## TABLE 10

**FOODBORNE INFECTIOUS DISEASE NOTIFICATIONS FOR 1995 BY PUBLIC HEALTH UNIT, RECEIVED BY FEBRUARY 28, 1995**

| Condition | CSA | SSA | ESA | SWS | WSA | WEN | NSA | CCA | ILL | HUN | NC | ND | WN | CW | SW | SE | Total |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Foodborne illness (NOS) | – | – | – | 13 | 12 | – | – | – | – | – | – | 1 | 6 | – | – | – | 32 |
| Gastroenteritis (instit.) | – | – | – | – | – | | – | – | – | 2 | – | – | – | – | – | – | 2 |
| Hepatitis A – acute viral | 10 | 7 | 22 | 3 | 3 | 1 | 6 | 1 | 1 | 5 | 4 | – | 2 | 20 | 3 | – | 88 |
| Listeriosis | – | – | – | 1 | – | – | – | 1 | – | – | – | – | – | – | – | – | 2 |
| Salmonella (NOS) | 7 | 16 | 17 | 20 | 16 | 13 | 29 | – | 12 | 23 | 8 | 16 | 4 | 7 | 3 | 9 | 200 |
| Typhoid and paratyphoid | – | 2 | 1 | 1 | 2 | 1 | – | – | – | – | – | – | – | – | – | – | 7 |

*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, NC North Coast Public Health Unit, ND Northern District Public Health Unit, WN Western New South Wales Public Health Unit, CW Central West Public Health Unit, SW South West Public Health Unit, SE South East Public Health Unit, 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.