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LEAD ISSUES

This issue of the *NSW Public Health Bulletin* focuses on lead.

- It contains two articles reporting investigations of lead exposure in primary school environments.
- On page 126 is the first in a new series of occasional reports giving a roundup of recent or current public health action being carried out by the NSW Public Health Network to assess or deal with specific problems. This month's Public Health Network Report is on lead. Readers' comments on the Network Report would be welcomed.
- The following is an invited commentary on the status of the lead problem in NSW, written by Garth Alperstein, Area Community Paediatrician, Central Sydney Area Health Service.

– Editor

LEAD – WHERE ARE WE NOW, AND WHERE TO FROM HERE?

Garth Alperstein
Area Community Paediatrician
Central Sydney Area Health Service

Over the past decade in Australia there has been a resurgence of interest in environmental lead contamination and lead exposure of children. This has resulted in several major initiatives which are having significant effects in reducing children's exposure. Perhaps the most important initiative was the introduction of unleaded petrol in Australia in 1986. About 55 per cent of vehicles use unleaded petrol, and recently the amount of lead in leaded petrol has been decreased.

In the past most research has focused on communities exposed to high levels of lead contamination from point sources such as smelters or mining operations. However, over the past five years the attention of researchers has begun to turn to the lead burden in large urban centres and its possible effects on children.

In June 1993 the National Health and Medical Research Council (NHMRC) revised its guidelines on Lead in Australians[1]. The NHMRC recommended that a goal be set to achieve a blood lead level below 0.48 µmol/l (10 µg/dl) for all Australians, particularly targeting the 1-4 year age group because of the adverse effects of lead exposure on intellectual development of young children. The strategy to achieve this goal included sample surveys of populations at increased risk of harmful lead exposure, and associated environmental surveys. The NHMRC also developed protocols for public health and individual management responses appropriate for various blood lead levels.

In November 1994 the NSW Government endorsed the NSW Lead Management Action Plan developed by the Interdepartmental Lead

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Correspondence

Please address all correspondence and potential contributions to:

The Editor,
NSW Public Health Bulletin,
Public Health Division,
NSW Health Department
Locked Bag No 961,
North Sydney NSW 2059
Telephone: (02) 391 9191
Facsimile: (02) 391 9029

Lead – where are we now?

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Taskforce[2]. This plan dealt comprehensively with the lead issue. Its recommendations accorded with those of the NHMRC for lead exposure of children and other high risk groups.

This issue of the *Bulletin* contains two articles reporting investigations of lead exposure in primary school environments (pages 121 and 124). Two very different instances of possible environmental lead contamination were investigated, and their relationship with children's blood lead levels was explored. Both reports have important implications for public health responses, research and policies in relation to lead exposure.

Other than studies of children living near point sources of lead (such as those at Broken Hill, Port Pirie and Boolaroo, near Newcastle), a limited number of surveys has targeted groups at increased risk of lead exposure. These groups include children aged 1-4 years living in, or frequently visiting, environments with the potential for high levels of lead exposure. These sources of exposure include houses built before the 1970s and painted with leaded paint (particularly houses being renovated, or having peeling, powdering of chalking paint); living on major roadways; and hobbies involving the handling of lead. The five published studies carried out in urban areas of Australia over the past decade provide some information on blood lead levels among children[3-7]. However, most have biases in sampling techniques and/or design, limiting the accuracy with which they characterise the distribution of blood lead levels in preschool children and identify target groups.

The investigation by Aldrich et al, reported on page 121 of this issue, was conducted as a public health response to a request from a school following concern by parents and teachers about lead exposure of children resulting from the construction of a four-lane motorway behind the school. In view of the public attention the lead issue has received over the past five years or so, the concern of the parents and school personnel was understandable. However, by many criteria, these children would be considered to have been at very low risk. Protective factors included the following:

- The children were older than the age at which hand-mouth behaviour frequently occurs.
- Physical barriers had been constructed to reduce noise pollution.
- Trees along the road also provided a physical barrier.
- The school grounds were some distance from the road.
- As a result of the local topography, the road was some metres below the level of the school.
- Although the estimates for the number of vehicles that would use the motorway were not available when the concerns were raised, it was unlikely that traffic would exceed 30,000 vehicles a day, and around 55 per cent of cars now use unleaded petrol.
- Research among preschool children in Eastern Sydney indicates that the blood lead level rises 0.05 µmol/l (1µg/dl) for every 10,000 vehicles a day, similar to results from overseas studies (personal communication: Dr Stephen Corbett, NSW Health Department, October 1995); the effect on school children is likely to be even less.

This survey highlights the problem where community concern demands a public health response, but the community's perception of risk does not have a scientific basis. The community's concern is understandable on the basis of mass media portrayals of the lead issue. However, the resources used to allay this concern could be used more constructively to survey a preschool population of children at risk of exposure from:

- Leaded paint; or
- Living near to a lead smelter.
- Close proximity to a roadway travelled by at least 30,000 cars a day; or
- Pica (repetitive ingestion of non-food substances) in high lead environments.

Further debate between the community, the public health system and the media is needed to moderate inaccurate perceptions of risk associated with lead exposure, promote an understanding of the risk and evolve appropriate forms of communication about it.

The report by Bawden-Smith et al (see page 124) provokes the question, how many children with aggressive or destructive behaviours or with "difficult temperaments" have, or have had, elevated blood lead levels, especially in the presence of pica? The article refers to an autistic child who required chelation. The child had exhibited aggressive and often violent behaviour for at least 18 months. The child's mother (and presumably also their doctor) had assumed this behaviour was part of autism. After chelation, the mother commented on the subsidence of her child's aggression and on how easy the child had become to manage, in comparison with behaviour before chelation.

The article should also stimulate debate and the formulation of a policy on blood lead testing for children with behavioural problems and significant developmental delay who attend special schools, and on environmental assessment of such schools. Because blood testing of these children can often be a difficult and traumatic experience, should all special schools be evaluated for lead contamination of paint and dirt? In response to the contamination discovered at the school assessed in the report, the NSW Health Department recommended to the Department of School Education that all Special Education schools be evaluated for lead contamination and remedied as appropriate.

The development of the Action Plan by the Interdepartmental Lead Taskforce was a significant step forward. The Taskforce recommended the establishment of a Lead Reference Centre within the Environmental Protection Agency to "co-ordinate the implementation of effective strategies by organisations identified in the Lead Management Action Plan which are aimed at reducing lead hazards, and to develop and implement the lead education strategies contained in the Lead Action Management Plan". Establishment of a functioning centre is a priority.

Finally, the two articles raise questions about directions for research. How much more research do we need on the contribution of lead in petrol to the environment, when this contribution will only decrease in the future? Leaded petrol will go away, but leaded paint which accounts for most of

ASSESSING THE IMPACT OF A NEW MOTORWAY ON CHILDREN'S BLOOD LEAD LEVELS

Rosemary Aldrich, Public Health Physician
Ruth Toneguzzi, Clinical Nurse Specialist
John Wlodarczyk, Medical Statistician
Michael J Hensley, Director
Newcastle Environmental Toxicology Research Unit
John Stephenson, Director
Hunter Public Health Unit

This article reports on surveys of blood lead levels in children attending a primary school adjacent to a new four-lane motorway. The surveys were carried out immediately after the motorway was opened in 1993, and again one year later.

It has been estimated that emissions from motor vehicles are responsible for the major part of Australian children's body burden of lead[1]. Other major sources of environmental lead exposure include point sources such as lead smelters, and deteriorating lead-based paint. The impact of leaded petrol on children has received increasing political, public and media attention in recent years and has been the subject of a federally-funded health promotion campaign[2]. International studies have detailed the contribution of heavy traffic to environmental lead and schoolchildren's blood lead levels[3,4].

The day before the four-lane M23 motorway opened on June 24, 1993, parents and teachers at a school in suburban Newcastle, NSW, raised concerns about possible environmental lead contamination from traffic using the motorway. The school principal asked the Newcastle Environmental Toxicology Research Unit (NETRU) to conduct a survey of children. The objectives were to measure baseline blood lead levels and to assess the impact of one year's traffic use of the motorway by repeating the blood lead testing in 1994. Estimates of the traffic volume on the motorway were not available.

NETRU and the Hunter Public Health Unit (HPHU) undertook to offer blood lead testing to the children at the school as a community service. However, the surveys also provided potential opportunities to examine dose-response relationships between traffic volumes and blood lead levels of children, thereby contributing to scientific understanding of the impact of lead exposure. Accordingly, information was collected on children's blood lead levels and on lead levels in soils and dusts.

METHOD

Initial survey

All children and teachers at the school were offered blood lead testing. The children's ages ranged from five to 13 years. Initial testing was carried out in July 1993, 10 school days after the motorway was opened. The test procedure was approved by the NSW Department of School Education, but the survey proposal was not submitted to a research ethics committee because the investigation was conducted as a matter of urgency. Children participated in the initial survey only with the written consent of their parents or legal guardians. Experienced blood collectors from the Hunter Area Pathology Service (HAPS) obtained samples by venipuncture over a three-day period, drawing sufficient

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the levels seen above 0.72 µmol/l (15 µg/dl) will not. There is a pressing need to obtain the baseline data specified in the NHMRC recommendations for the preschool population at high risk of lead exposure.

In summary, the articles highlight the need for clear policy regarding research and surveillance of blood lead levels among high risk exposure groups; policy regarding environmental evaluation, blood lead testing and abatement of lead contamination; and a commitment to action.

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EDITOR'S COMMENTS

With regard to Dr Alperstein's recommendations:

- During 1995 the NSW Department of School Education carried out environmental sampling in five Special Education schools with buildings constructed before 1970 and which had recently undergone cyclic maintenance. One of the schools was found to have high lead levels in carpet dust and the carpet was replaced. No excessive lead levels were found in the other four schools.
- A Lead Reference Centre is being established in December 1995. It is a joint initiative of the NSW Environmental Protection Authority and the NSW Health Department, with other participating agencies. The Centre is at the Gladesville Hospital premises of the Health Department's Public Health Division.
- A national survey of blood lead levels in preschool-aged children was completed in June 1995. The survey was carried out under the auspices of the Australian Institute of Health and Welfare. Only preliminary results were available at the time of writing, and a full report had not been published.

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blood from each child for a full blood count (FBC) and lead assay.

On the day of blood collection, children were given letters to their parents or guardians informing them of the anticipated date results would be available. Within three weeks all results had been processed and reviewed by a medical practitioner. Written results were given to parents and guardians, accompanied by an explanatory letter and information on how to contact a member of the survey team for further information and discussion if desired.

Soil and dust samples were collected by HPHU Environmental Health Officers, using an Environment Protection Authority (EPA) protocol adapted for the site[5]. The samples were sent to the analytical laboratory at Pasminco-Metals Sulphide, Boolaroo.

Follow-up survey

NETRU and HPHU conducted the repeat survey in July 1994, again offering blood lead testing to all children and teachers at the school. Three weeks before the 1994 survey, letters were sent to the children's parents or guardians informing them of the second round of testing and seeking written consent for venipuncture, FBC and blood lead estimation. The schedule and procedure for blood testing and distribution of results followed the 1993 protocol.

The follow-up testing was also offered to children who had attended the school in 1993 but had since left. With the consent of their parents or guardians, these children had venipunctures at the school or at the John Hunter Hospital Outpatients Department.

HPHU Environmental Health Officers repeated soil and dust sampling.

Aggregate results of the two surveys were not reported or published until all individual results were distributed and parents had received a summary of the aggregate results in a school newsletter.

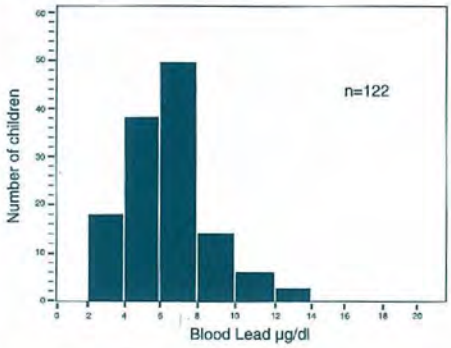
To avoid confusion, results were explained (by letter or in person) to parents and guardians in micrograms per decilitre (µg/dl), although results were also presented in micromoles per litre (µmol/l). It was believed that there had been substantial community discussion about "goals" of 10µg/dl and "actions" at 15µg/dl to warrant a need for consistency. Results in this article are reported in µg/dl, followed by µmol/l in square brackets. (Results in µg/dl may be converted to µmol/l by dividing by 20.714.)

Results

Ninety-five schoolchildren had a test in 1993, and 122 in 1994, giving response rates of 60 per cent and 65 per cent respectively. Of the 73 children who had a test in 1993 and were still at the school in 1994, 65 had a second test in 1994 – a follow-up rate of 89 per cent. Of the 22 children who had tests in 1993 and had left the school by July 1994, only three were retested – a response rate of 14 per cent. In 1994, 57 children had a blood lead test for the first time, so a total of 152 children had at least one test, either in 1993 or in 1994.

The mean blood lead level of the 122 children at the school in 1994 was 6.23µg/dl (95% confidence interval: 5.87-6.6) [0.30µmol/l; 95% CI: 0.28-0.32]. This was very similar to the mean for the 95 children tested in 1993 of 6.16µg/dl (95% CI: 5.73-6.58) [0.30µmol/l; 95% CI: 0.28-0.32]. The levels in 1993 ranged from 1.7µg/dl to 14.1µg/dl [0.08µmol/l to 0.68µmol/l], and in 1994 from 2.5µg/dl to 13.9µg/dl [0.12µmol/l to 0.67µmol/l]. The distribution of the results in 1994 is illustrated in Figure 1.

DISTRIBUTION OF RESULTS, SCHOOL BLOOD LEAD SURVEY, 1994



The same proportion of children in 1993 and 1994 (4 per cent) had blood lead levels of 10µg/dl [0.48µmol/l] or more, the National Health and Medical Research Council (NHMRC) goal for all Australians. No child required individual attention, which is recommended by the NHMRC in children with a blood lead level of 15µg/dl or greater.

The mean blood lead of the children tested twice was 6.39µg/dl (95% CI: 5.81-6.97) [0.31µmol/l; 95% CI: 0.28-0.34] in 1993 and 5.95µg/dl (95% CI: 5.44-6.46) [0.29µmol/l; 95% CI: 0.26-0.31] in 1994. This reduction of 0.44µg/dl is significant (95% CI: -0.77 to -0.096µg/dl), but is consistent with the decrease in blood lead seen in children with increasing age.

Environmental lead levels were assayed from samples collected at seven sites in 1993 and six sites in 1994 (Table 1). Site 2 could not be retested because no loose dirt or dust was present in 1994. The mean concentration in 1993 (arithmetic mean 182ppm, geometric mean 87ppm, 95% CI: 26-293) was very close to that in 1994 (AM=183ppm, GM=112, 95% CI: 34-362); confidence intervals were very wide because of the small numbers of samples. These data suggest no major change in lead burden in soils and dusts over the year. Importantly, the upper confidence limit for 1993 did not exceed the EPA threshold for investigation (300 parts per million), and the upper confidence limit for 1994 exceeded the threshold only slightly.

According to the NSW Roads and Traffic Authority (RTA), a daily average of 14,400 vehicles have used the M23 motorway since it opened.

Discussion

The results of the surveys indicate that the burden of lead in the school population and the school environment did not increase over the first year after the motorway was opened. There are three possible explanations for this finding.

- Significant lead pollution of the school environment did not occur. A daily average of fewer than 15,000

TABLE 1

ENVIRONMENTAL SAMPLES FROM THE SCHOOL – LEAD CONTENT IN PARTS PER MILLION, 1993 AND 1994

Sample # and Type	Result (ppm) (µg/g)	
Date	15 July, 1993	11 August, 1994
1. Soil 320 130		
2. Soil	160	Not available
3. Soil	25	65
4. Soil	50	30
5. Soil	30	375
6. Dust	660	450 (only 0.02µg/dl sample)
7. Soil	30	45
Average	182.14	182.50

* Note: Results in µg/dl may be converted to µmol/l by dividing by 20.714.

cars does not represent heavy traffic, and about half the vehicles were likely to have been using unleaded petrol. Furthermore, the RTA had built physical barriers between the school and the road in the early stages of the motorway's construction to minimise the potential impact of visual, noise and air pollution. Fortunately, the local topography enabled the road surface to be some metres below the level of the school grounds, and hidden from view by a 3m high timber fence and trees planted on the roadside embankment. The trees were about 3m tall in 1993 and 4-5m in 1994. Any lead emitted from motor vehicles was probably restricted in its fall to the immediate vicinity of the roadway and would not have become sufficiently airborne to settle on the school side of the fence, thereby causing little or no pollution in the school grounds.

- Significant lead pollution did occur, but the study did not detect it. The response rates of 60 per cent in 1993 and 65 per cent in 1994 were disappointing. However, it was unlikely that the blood lead levels of non-participants would have risen more than those of participants.

The environmental sampling was on a small scale. A more comprehensive sampling schedule would have had more power to detect a difference in environmental lead between 1993 and 1994, if a real difference had existed. However, the mean soil and dust lead levels for 1993 and 1994 were so similar as to make it unlikely that a major effect was missed.

- It is possible (but unlikely) that the follow-up survey was conducted too soon after the initial survey and that a longer follow-up period would have shown a different pattern.

Questions about accumulated lead could be addressed by serial blood lead and environmental lead surveys at the school. More information on environmental pollution could be obtained by measuring air lead levels with a high-volume sampler. However, such surveys seem unwarranted in this setting.

Lyngbye et al[3] and von Schirnding et al[4] have found that proximity of schools or residences (respectively) to heavy traffic was associated with higher blood lead levels in school children. We plan to assess the dose-response relationship in children living or attending schools near main roads, using data from more than 2,100 children whom we have tested in the Hunter area since 1991.

The present investigation was carried out in response to community concern. It was an efficient way to examine the impact of a new motorway on children's and environmental lead levels. The methods used could be applied in another setting should the public health need arise.

ACKNOWLEDGMENTS

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Address for correspondence:
Newcastle Environmental Toxicology Research Unit
Level 1, Royal Club Building
Royal Newcastle Hospital
PO Box 664J
Newcastle NSW 2300.
Phone: 049 23 6214
Fax: 049 23 6654

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LEAD EXPOSURE AT A SCHOOL FOR CHILDREN WITH DEVELOPMENTAL DISABILITIES

Jason Bawden-Smith, Environmental Health Officer
Jeremy McAnulty, Director
Southern Sydney Public Health Unit
Mark Bek, Acting Director
Central Sydney Public Health Unit

In July 1994 a paediatrician reported that an autistic child aged six years had been admitted to hospital for chelation therapy with a blood lead level of 3.86 micromoles per litre. The child had ingested some granular material containing 23 per cent lead. This material was found in the child's home, where it was possibly used to treat rising damp. In September 1994 the child was enrolled at a school for children and young people with developmental disabilities. In view of the child's history of pica and elevated blood lead level, the school environment was assessed for possible lead contamination. Because it was believed that many of the students at the school engaged in hand-to-mouth activities, they were also offered blood lead testing. This article reports the findings of the environmental and blood lead surveys, which were conducted in October 1994, and outlines interventions designed to reduce the lead burden.

METHODS

Environmental survey

The school principal identified areas in the school grounds where children played and attended classes. Environmental samples were collected from these areas, which included carpet, window wells, wooden floors, bare surface soil and peeling paint. The samples were tested for total lead using standard methods.

Survey of children

Parents received letters advising them to have their children tested for lead levels, either through their general practitioners or at special clinics held at the school. Class teachers completed a form giving demographic information about the children and information on the frequency with which they put fingers or objects into their mouths. Data analysis focused on the relationship between blood lead levels and children's behaviour and demographic characteristics.

RESULTS

Environmental study

The school's administration building was a refurbished two-storey Victorian-style erected in the 1880s. The classrooms and other facilities were located in buildings constructed in the mid- to late 1970s. A month before this investigation began, contractors were employed to repaint the administration building. They sanded back lead-painted exterior timber and masonry surfaces.

Very high levels of lead contamination were found on interior and exterior surfaces of the administration building. The classrooms and associated facilities contained low levels of lead, except for carpets in classrooms 2 and 4 (Table 2).

Survey of children

At the time of the survey 76 children were attending the school in years 1-11. Their ages ranged from 3 to 18 years (median 9 years), and 57 (75 per cent) were male. Teachers reported that 26 (34 per cent) children frequently, 8 (11 per cent) sometimes and 41 (54 per cent) rarely placed objects in

TABLE 2

ENVIRONMENTAL LEAD RESULTS FOR THE SCHOOL, OCTOBER 1994 – BEFORE AND AFTER ABATEMENT

Sample location and description (units)		Before abatement	After abatement	US EPA guidelines
Administration building				
Library	vacuum cleaner dust (mg/kg)	49,423	carpet removed	500-1,000
	dust wipe floor (µg/m²)	9,389	2,725[a]	1,076
	dust wipe window well (µg/m²)	16,451	9,941	8,608
	paint interior wall (% w/w)[b]	0.22	N/A	0.5
Staffroom	dust wipe floor (µg/m²)	8,050	2,619[a]	1,076
	dust wipe window well (µg/m²)	6,324	1,490	8,608
Office dust	wipe floor (µg/m²)	2,144	1,219[a]	1,076
	dust wipe window well (µg/m²)	3,818	1,611	8,608
Classroom	dust wipe floor (µg/m²)	2,736	1,756[a]	1,076
	dust wipe window well (µg/m²)	3,667	2,373	8,608
Paint	exterior wall (% w/w)	10.53	removed/covered	0.5
	interior bathroom wall (% w/w)	30.60	removed/covered	0.5
Soil	perimeter of building (mg/kg)	4,649	371	400
Classrooms and common areas				
Classroom 2	vacuum dust (mg/kg)	1,033	521	500-1,000
	paint hallway wall (% w/w)	5.45	removed/covered	0.5
Classroom 4	vacuum dust (mg/kg)	1,156	460	500-1,000
Other	vacuum dust (mg/kg)	184-563	248-417	500-1,000
classrooms	paint interior wall (% w/w)	0.06	N/A	0.5
Soil	(mg/kg)	77[c]	N/A	400
Paint	handrails (% w/w)	0.45	N/A	0.5
Water	first flush from bubbler (µg/l)	5	N/A	10

a new carpet on top
b percentage weight for weight
c arithmetic mean of 6 soil samples
N/A Not applicable since before abatement results are below standards

their mouths, and that 27 (36 per cent) frequently, 12 (16 per cent) sometimes and 37 (49 per cent) rarely placed their fingers in their mouths.

Forty-two (55 per cent) of the children had venipuncture for blood lead level determination. The median blood lead was 0.22 µmol/l (range 0.10-2.57 µmol/l), and five children (12 per cent) had elevated lead levels (>0.48 µmol/l). There were no significant differences between children with elevated lead levels and others by age, sex or grade. Children reported by their teachers to place objects or their fingers in their mouths frequently were significantly more likely to have lead levels above 0.48 µmol/l than children who rarely placed objects or fingers in their mouths (Table 3).

TABLE 3

CHARACTERISTICS OF CHILDREN TESTED FOR BLLs AT THE SCHOOL, OCTOBER 1994

Characteristic	Total	No. >0.48 µmol/l (%)
Age		
<10 years	21	3 (14)
≥10 years	21	2 (10)
Sex		
male	30	5 (17)
female	11	0 (0)
unknown	1	0 (0)
Class		
1-2	9	2 (22)
3-5	6	1 (17)
6-7	10	0 (0)
8-9	9	2 (22)
10-11	8	0 (0)
Objects in mouth[a]		
frequently	15	4 (27)
sometimes	5	1 (20)
rarely	21	0 (0)
unknown	1	0 (0)
Fingers in mouth[b]		
frequently	17	4 (24)
sometimes	5	1 (20)
rarely	20	0 (0)
Total	42	5 (12)

a chisquare for trend 5.8, p<0.02
b chisquare for trend 4.8, p<0.03

INTERVENTION

To reduce lead exposure school staff restricted student access to potentially contaminated areas. Temporary fences were erected around the contaminated soil, carpets were replaced, and floors, windows, cupboards and other surfaces and objects (including books in the library) in and around the administration building were thoroughly cleaned. Soil around the perimeter of the administration building to a thickness of 10cm was removed and the area was paved. All deteriorating paint was removed or encapsulated. Carpet in the classrooms was professionally cleaned. Cleaners were asked to wipe dust from painted wooden window wells and sills as a routine.

Repeat sampling after the abatement program showed soil lead contamination had been substantially reduced (Table 2).

The home environments of children with blood lead levels >0.72 µmol/l were also investigated for lead contamination, and their parents were advised on ways to reduce their children's lead exposure and to minimise absorption.

DISCUSSION

This investigation showed there was significant lead contamination in the school environment. Our findings indicate that children with developmental disabilities may be at greater risk of lead exposure due to their behavioural characteristics such as pica and frequent hand-to-mouth activity.

The remedial work undertaken at the school reduced environmental lead contamination, minimising the risk for students. While dust lead levels on the floor in the administration building remained high despite intensive cleaning, they were covered with new carpet to prevent access. This finding demonstrated the difficulty in removing lead dust from surfaces that are not smooth and reinforces the importance of minimising the dispersal of dust when removing lead-based paints. Lead dust levels remained high in window wells in the administration building due to the dust created by friction when the sash windows previously painted with a lead-based paint were opened and closed. Higher carpet dust levels in classrooms located closest to the administration building may be attributed to students and staff "tracking" contaminated soil and dust on their shoes from the administration building.

Lead is a potent neurotoxin that adversely affects many systems in the body, especially the central nervous system, the renal system and blood-forming tissues[1]. Recent studies have shown adverse neuro-psychological effects associated with elevated blood lead levels in children[2], including reduced intelligence quotient[3], attention deficits[4], aggression and destructive behaviours[5]. Damage to the central nervous system may be permanent, resulting in school failure and anti-social behaviour, and ultimately impairing productivity[4].

Levels of environmental lead contamination found in areas around the administration block of the school were well above North American intervention guidelines[6] (<0.5% in paint, 400 mg/kg in accessible bare soil in play areas, lead dust loading for floor <1076 µg/m², window sills, 5380 µg/m² and window wells <8608 µg/m²).

Twelve per cent of the 42 children tested had blood lead levels above the National Health and Medical Research Council goal (<0.48 µmol/l). This was no higher than levels found in preschool children in Central and Southern Sydney[7] but may be higher than expected for primary school children. Children in this study who frequently placed their fingers and other objects in their mouths were more likely to have high blood lead levels.

A major limitation of the survey was the disappointing response to the offer of blood lead testing, with only 55 per cent of students being tested. The extent to which this group represented the school's entire enrolment is unknown. Another limitation was the reliance on teacher reports of hand-to-mouth activity after the blood screening of students.

This investigation was initiated in response to a need to protect a child with a history of an elevated blood lead level.

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NSW PUBLIC HEALTH NETWORK REPORT

LEAD

While overt childhood lead poisoning from environmental sources has been long recognised in Australia, concern over the more subtle effects of chronic low-level exposure to lead has arisen only over the past decade or so. Communities clearly at high risk in NSW are those near lead industries – such as Boolaroo in Newcastle and Broken Hill. In both these communities the Public Health Network has determined the proportion of children with a high blood level, and some of the risk factors. Children in older urban areas face risks from old lead-based paint and soil that has become contaminated from leaded petrol exhausts. The research by the Public Health Network has assessed the extent of risk among these children as well. Work is now addressing interventions to reduce children's blood lead level. In Broken Hill, the Environment Protection Authority (EPA) and Western NSW Public Health Unit are collaborating to evaluate these efforts. In Sydney, Public Health Units look forward to working with the EPA's recently upgraded Lead Reference Centre to develop interventions and conduct further research to reduce lead poisoning among urban children.
The following list describes the action of the NSW Public Health Network to address lead problems since 1991.

Publications and reports

Title: **Community prevalence survey of children's blood lead levels and environmental contamination in inner Sydney**
Authors: Fett M, Mira M, Smith J, Alperstein G, Causer J, Brokenshore T, Gulson B, Cannata S.
Publication: *Med J Aust 1992*; 157: 441-445
Contact: CS PHU

Title: **Mort Bay, Balmain pilot study of blood lead in children**
Authors: Smith J, Mira M, Fett M, Alperstein G, Gulson B, Brokenshore T, Cannata S.
Publication: *Central Sydney Public Health Unit* 1992
Contact: CS PHU

Title: **Living near a lead smelter: an environmental health risk assessment in Boolaroo and Argenton, New South Wales**
Authors: Galvin J, Stephenson J, Wlodarczyk J, Loughran R, Waller G.
Publication: *Aust J Public Health* 1993; 17: 373-8
Contact: HUN PHU

Title: **The Newcastle lead study: how straight is the critical path for a multisectoral approach towards a public health outcome?**
Authors: Galvin J, Stephenson J.
Publication: Conference Proceedings, Public Health Association Conference, Canberra, 1992
Contact: HUN PHU

Title: **Tackling childhood lead poisoning: The Newcastle lead study**
Authors: Galvin J, Stephenson J, Corbett S.
Publication: *NSW Public Health Bulletin* 1991; 2:98-99
Contact: HUN PHU

Title: **Blood lead levels in 1-4 year old children attending child care centres in the Eastern Sydney Area**
Authors: Cowie C, Black D, Ferson M, Fraser I.
Publication: *NSW Public Health Bulletin* (Supplement) 1994; 5(S-1): 9
Contact: ES PHU

Title: **Illawarra child blood lead study 1994**
Authors: Kreis I, Calvert G, Gan I, Westley-Wise V, Willison R.
Publication: *Illawarra Environmental Health Unit/Illawarra Public Health Unit* 1994.
Contact: ILL PHU

Title: **Evaluation of possible environmental sources of lead affecting children in Port Kembla, Kemblawarra, Warrawong and Cringila**
Authors: Williams C, Calvert GD, Gan I, Kacprzak J, Kreis I, Westley-Wise V, Willison R.
Publication: *Illawarra Environmental Health Unit/Illawarra Public Health Unit*, 1995.
Contact: ILL PHU

Title: **Risk factors for blood lead levels in preschool children in Broken Hill**
Authors: Phillips A, Hall J.
Publication: *Western NSW Public Health Unit*
Contact: WN PHU

Public health programs and research

Title: **1991 Prevalence study of blood lead levels in Broken Hill** (completed)
Contact: WN PHU

Title: **1992 Cross-sectional study of blood lead levels in Broken Hill** (completed)
Contact: WN PHU

Title: **Survey of blood lead level in children** (completed)
Contact: CS PHU

Title: **1993 Broken Hill cross-sectional blood lead survey** (completed)
Contact: WN PHU

Title: **Case control study of risk factors for elevated lead in Broken Hill** (completed)
Contact: WN PHU

Title: **Case control study of environmental factors and high blood lead levels in the Port Kembla area** (in progress)
Contact: ILL PHU

TABLE 4

NSW PUBLIC HEALTH UNITS

Code	Unit	Address	Phone	Facsimile
CC PHU	Central Coast Public Health Unit	PO Box 361 GOSFORD 2250	043 20 4545	043 20 4550
CS PHU	Central Sydney Public Health Unit	PO Box 374 CAMPERDOWN 2050	02 550 6810	02 565 1690
CW PHU	Central West Public Health Unit	PO Box 143 BATHURST 2795	063 32 8505	063 32 8577
ES PHU	Eastern Sydney Public Health Unit	Locked Bag 88 RANDWICK 2031	02 313 8322	02 313 6291
HUN PHU	Hunter Public Health Unit	PO Box 11A NEWCASTLE 2300	049 29 1292	049 29 4037
ILL PHU	Illawarra Public Health Unit	PO Box 66 KEIRAVILLE 2500	042 26 4677	042 26 4917
NC PHU	North Coast Public Health Unit	PO Box 498 LISMORE 2480	066 21 7231	066 22 2151
ND PHU	Northern Districts Public Health Unit	PO Box 597 TAMWORTH 2340	067 66 2288	067 66 3003
NS PHU	Northern Sydney Public Health Unit	Hornsby Ku-ring-gai Hospital Palmerston Road HORNSBY 2077	02 477 9400	02 482 1650
SE PHU	South Eastern Public Health Unit	Locked Mail Bag 11 GOULBURN 2580	048 27 3428	048 27 3438
SS PHU	Southern Sydney Public Health Unit	PO Box 482 KOGARAH 2217	02 350 3377	02 350 3474
SW CPH	South West Centre for Public Health	PO Box 503 ALBURY 2640	060 58 1700	060 58 1701
SWS PHU	South Western Sydney Public Health Unit	Locked Bag 17 LIVERPOOL 2170	02 828 5944	02 828 5955
WN PHU	Western NSW Public Health Unit	PO Box M61 EAST DUBBO 2830	068 81 2235	068 84 7223
WS PHU	Western Sector Public Health Unit	13 New Street NTH PARRAMATTA 2151	02 840 3603	02 840 3608

(Compiled by the South West Centre for Public Health on behalf of the Public Health Network)

Lead exposure at a school

► *Continued from page 125*

As a consequence an environmental assessment of the child's new school was carried out. Subsequently, other students at the school were offered blood lead testing and public health action was taken to reduce lead exposure within the school grounds. The study highlighted the benefits of notification of elevated blood lead levels to public health authorities, following NHMRC recommendations. Public health action can then be taken to reduce exposures. The study also drew attention to the value of targeting children with developmental disabilities for lead screening, especially those in potentially contaminated environments and those who frequently engage in hand-to-mouth activity.

1. National Research Council. Measuring lead exposure in infants, children and other sensitive populations. Committee on Measuring Lead in Critical Populations. Board of Environmental Studies and Toxicology. Commission on Life Sciences. Washington DC: National Academy of Press, 1993.
2. Needleman H, Bellinger D. The health effects of low pressure to lead. *Ann Rev Pub Hlth* 1991; 12:111-140.
3. McMichael AJ, Baghurst PA, Wigg NR et al. Port Pirie cohort study: Environment exposure to lead and children's intelligence at the age of seven years. *N Engl J Med* 1992; 327:1279-84.
4. Needleman H, Schell A, Bellinger D, Leviton A, Allred EN. The long-term effects of exposure to low doses of lead in childhood. An 11-year follow-up report. *N Engl J Med* 1990, 322:82-88.
5. Sciarillo WG, Alexander G, Farrell KP. Lead exposure and child behaviour. *Am J Public Health* 1992; 82:1356-1360.
6. National Centre for Lead Safe Housing. Guidelines for the Evaluation of Lead-based Paint Hazards in Housing. February 1995.
7. Mira M, Bawden-Smith J, Causer J, Alperstein A, Karr M, Snitch P, Waller G, Fett J. Blood lead concentrations of urban Australian children. Submitted to *Am J Paed.*

UPDATE ON THE WAITING LIST REDUCTION PROGRAM

Nick Shiraev and Jean McGarry
Evaluation and Monitoring Branch
Performance Management Division
NSW Health Department

The October issue of the *Public Health Bulletin* contained an article describing the initial results of the Waiting List Reduction Program to the end of September 1995[1]. This report updates the results to the end of October.

The Program was introduced to improve access to elective surgery services in public hospitals throughout NSW. The aim is to reduce the March 31, 1995 elective surgery waiting lists by 50 per cent within 12 months, concentrating on people who have been waiting more than six months for surgery.

RESULTS

By October 31 the elective surgery waiting list had decreased from 44,707 to 29,780 patients, a reduction of 14,927 patients (33 per cent) (Table 5).

Since March 31 the number of people waiting longer than 12 months has declined by 1,343 (59 per cent), and the number waiting between six and 12 months has fallen by 3,674 (58 per cent) (Table 6). The waiting list has already been halved for people waiting more than six months; the most significant effect of the Program has been the reduction in the number of people waiting more than six months by over 5,000.

Particularly significant were reductions in numbers of people waiting longer than six months for procedures such as cholecystectomy (down by 207 or 70 per cent), cataract extraction (down by 771 or 63 per cent), tonsillectomy (down by 416 or 67 per cent) varicose vein stripping and ligation (down by 303 or 72 per cent) and total hip replacement (down by 78 or 43 per cent).

The average waiting time of patients treated in the course of a month rose from 1.4 months in March to 1.7 months in October. This reflects large numbers of admissions of patients who have been waiting long periods. The clearance time has reduced to 1.4 months from 1.8 months, indicating patients have been admitted more quickly. The average time on the list has fallen from 3.6 months to 2.8 months, indicating patients are waiting on the list almost a month less (Table 7). This overall pattern is reflected in most Areas and Districts (Table 7).

In the period from July 1, 1994 to March 31, 1995 admissions increased by 2.5 per cent compared with the corresponding period a year previously. Then between April

TABLE 5

REDUCTIONS IN WAITING LISTS BY AREA AND DISTRICT HEALTH SERVICE, MARCH TO OCTOBER, 1995

Area/District/Institution	Number on list		Total reduction	
	March 31, 1995	October 31, 1995*	Number	%
Central Sydney	2,779	2,004	–775	–27.9
Northern Sydney	2,708	1,689	–1,019	–37.6
Western Sydney	4,650	2,168	–2,482	–53.4
Wentworth	2,122	1,475	–647	–30.5
South West Sydney	4,514	3,127	–1,387	–30.7
Central Coast	2,317	1,660	–657	–28.4
Hunter	4,178	3,408	–770	–18.4
Illawarra	2,778	2,064	–714	–25.7
South Eastern Sydney	7,190	4,152	–3,038	–42.3
Royal Alexandra Hospital	792	713	–79	–10.0
Barwon	43	29	–14	–32.6
Castlereagh	51	5	–46	–90.2
Central Western	588	560	–28	–4.8
Clarence	199	11	–188	–94.5
Evans	330	225	–105	–31.8
Far West	204	149	–55	–27.0
Hume	413	226	–187	–45.3
Lachlan	111	59	–52	–46.8
Lower North Coast	1,071	740	–331	–30.9
Macleay-Hastings	385	200	–185	–48.1
Macquarie	813	636	–177	–21.8
Mid North Coast	887	531	–356	–40.1
Monaro	59	50	–9	–15.3
Murray	16	20	4	25.0
Murrumbidgee	–	–	–	–
New England	404	279	–125	–30.9
North West	1,213	695	–518	–42.7
Orana	33	30	–3	–9.1
Richmond	681	432	–249	–36.6
Riverina	1,276	850	–426	–33.4
South Coast	89	24	–65	–73.0
Southern Tablelands	187	57	–130	–69.5
Tweed Valley	824	574	–250	–30.3
Port Macquarie Base Hospital	802	938	136	17.0
New South Wales	**44,707**	**29,780**	**–14,927**	**–33.4**

Source: Department of Health Reporting System, November 9, 1995

* Excludes transfers. List transfers, in general, are an administrative change and not a change in local demand. They are therefore not taken into account when estimating changes in the number of patients on a list.

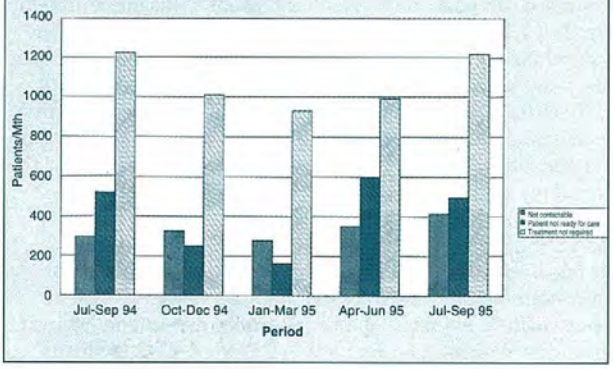
and October 1995, the total number of admissions to hospitals in NSW rose by 35,744 – or 4.8 per cent – compared with the corresponding period in 1994. On an annual basis this is equivalent to more than 60,000 extra admissions. Thus the increase in admissions attributable to the Waiting List Reduction Program to October 31, 1995 is estimated to have been 2.3 per cent, representing 28,000 admissions on an annual basis.

It is also estimated that, to October 31, 1995, at least 16,386 extra procedures were performed as part of the Waiting List Reduction Program. This is likely to be an underestimate because, in mid-1994, a large number of medical procedures, such as endoscopies and chemotherapy, was erroneously coded as surgery. Most of the increase occurred in the period June-October 1995, but there was some minor increase in April and May. The rate of increase of elective surgery was 11.2 per cent, more than double the general increase (total all programs) of 4.8 per cent in the same period (Table 8).

The changes in listing status over the past five quarters are shown in Figure 2. The main reason for removal of patients from waiting lists was that the treatment was no longer

FIGURE 2

LISTING STATUS CHANGES FROM READY FOR CARE (EXCLUDING ADMISSIONS)



Continued on page 130 ▶

TABLE 6

REDUCTIONS IN PATIENTS ON AREA AND DISTRICT WAITING LISTS, OCTOBER, 1995
PATIENTS WAITING LONGER THAN SIX MONTHS

	NUMBER ON LIST							
	Waiting 6-12 months				Waiting > 12 months			
			Reduction				Reduction	
Area/District/Institution	Mar 31, 1995	Oct 31, 1995	Number	%	Mar 31, 1995	Oct 31, 1995	Number	%
Central Sydney	116	19	–97	–84	15	0	–15	–100
Northern Sydney	404	107	–297	–74	173	100	–73	–42
Western Sydney	836	139	–697	–83	295	74	–221	–75
Wentworth	340	125	–215	–63	293	134	–159	–54
South West Sydney	555	309	–246	–44	191	143	–48	–25
Central Coast	398	254	–144	–36	107	25	–82	–77
Hunter	768	415	–353	–46	179	94	–85	–47
Illawarra	463	181	–282	–61	173	74	–99	–57
South Eastern Sydney	1,150	467	–683	–59	559	161	–398	–71
Royal Alexandra Hospital	25	8	–17	–68	11	–	–11	–100
Barwon	–	–	–	–	–	–	–	–
Castlereagh	–	–	–	–	–	–	–	–
Central Western	80	57	–23	–29	8	8	–	–
Clarence	1	–	–1	–100	–	–	–	–
Evans	2	–	–2	–100	–	–	–	–
Far West	1	–	–1	–100	–	–	–	–
Hume	25	14	–11	–44	4	–	–4	–100
Lachlan	13	–	–13	–100	–	–	–	–
Lower North Coast	207	146	–61	–29	52	1	–51	–98
Macleay-Hastings	101	10	–91	–90	46	6	–40	–87
Macquarie	164	58	–106	–65	41	42	1	2
Mid North Coast	150	31	–119	–79	4	1	–3	–75
Monaro	–	–	–	–	–	–	–	–
Murray	–	–	–	–	–	–	–	–
Murrumbidgee	–	–	–	–	–	–	–	–
New England	26	–	–26	–100	1	–	–1	–100
North West	169	61	–108	–64	16	2	–14	–88
Orana	4	1	–3	–75	–	2	2	–
Richmond	95	31	–64	–67	1	4	3	300
Riverina	199	82	–117	–59	77	5	–72	–94
South Coast	4	2	–2	–50	–	–	–	–
Southern Tablelands	1	1	–	–	–	–	–	–
Tweed Valley	79	20	–59	–75	19	–	–19	–100
Port Macquarie Base Hosp	3	167	164	5,467	–	46	46	–
New South Wales	**6,379**	**2,705**	**–3,674**	**–58**	**2,265**	**922**	**–1,343**	**–59**

Source: Department of Health Reporting System, November 9, 1995

Update on waiting lists

► *Continued from page 129*

required (for clinical or personal reasons); this accounted for around 1,200 patients, virtually unchanged from the same period last year. The next major reason for removal from the list was that the patients were not ready for care (around 500 patients a month, again similar to last year). The third reason for removal from the list was that the patient was no longer contactable. This has increased by about 100 over the same period last year.

DISCUSSION

It has been suggested that reductions in waiting lists stimulate an increase in demand for services. At present there is little evidence of an accelerated demand following from the Waiting List Reduction Program. This confirms recent overseas findings[2] that induced demand resulting from substantial reductions in waiting lists is, at least in the short term, rather small. It will be interesting to see whether a surge in demand is apparent early in 1996.

Predicted changes observed in the flow patterns are the result of growing and relatively under-resourced areas being able to provide more of their residents with services locally. Examples are the Wentworth and Central Coast Areas.

It is now appropriate to direct the focus of attention to waiting times and the achievement of best practice in the management of elective patients. Future issues for consideration may include development of benchmarks for:

- patients allocated clinical urgency codes of 1 (should be admitted within seven days) or 2 (should be admitted within one month);
- theatre utilisation;
- admissions delayed by hospital constraints; and
- same day surgery.

1. Shiraev N, McGarry J. Waiting List Reduction Program: initial results. *NSW Public Health Bulletin* 1995; 6(10):106-109.
2. Martin S, Smith P. Modelling waiting times for elective surgery. Centre for Health Economics, The University of York, 1995.

TABLE 7

VARIATIONS IN WAITING TIMES BY AREA AND DISTRICT HEALTH SERVICE, MARCH TO OCTOBER, 1995

	WAITING TIMES (Months)								
	Expected waiting time			Average waiting time			Average time on list		
Area/District/Institution	Mar 31, 95	Oct 31, 95	Variation*	Mar 31, 95	Oct 31, 95	Variation*	Mar 31, 95	Oct 31, 95	Variation*
Central Sydney	1.1	0.9	–0.3	1.0	0.9	0.1	1.5	1.0	–0.5
Northern Sydney	1.3	1.0	–0.3	0.9	2.3	1.4	3.5	2.8	–0.7
Western Sydney	1.9	1.3	–0.6	1.6	1.5	0.0	4.1	2.6	–1.5
Wentworth	2.6	1.7	–0.9	2.1	2.6	0.4	5.9	4.4	–1.4
South West Sydney	1.7	1.5	–0.2	1.4	1.6	0.2	3.3	3.4	0.2
Central Coast	2.5	2.1	–0.4	1.9	2.2	0.3	4.0	3.0	–1.0
Hunter	2.0	1.8	–0.2	1.6	1.8	0.2	3.7	3.1	–0.6
Illawarra	2.4	1.7	–0.6	2.3	2.3	0.0	3.9	2.9	–1.1
South Eastern Sydney	1.7	1.4	–0.3	1.2	1.5	0.2	4.2	3.2	–1.0
Royal Alexandra Hospital	1.3	2.3	0.9	1.3	1.0	–0.3	1.6	1.4	–0.2
Barwon	0.6	0.4	–0.2	0.5	0.4	0.0	0.7	0.3	–0.4
Castlereagh	0.6	0.4	–0.2	0.5	0.8	0.3	0.5	3.1	2.6
Central Western	1.4	1.4	0.0	1.1	1.7	0.6	2.6	2.7	0.1
Clarence	1.6	0.5	–1.1	0.4	0.4	0.0	1.9	0.4	–1.5
Evans	0.9	0.8	–0.1	0.8	0.8	0.0	1.1	1.0	–0.1
Far West	1.4	1.1	–0.2	0.9	1.1	0.2	1.4	1.1	–0.3
Hume	0.8	0.8	0.0	0.9	0.7	–0.2	2.0	1.9	–0.2
Lachlan	0.9	0.5	–0.4	0.3	0.7	0.3	2.1	1.4	–0.8
Lower North Coast	5.4	2.5	–2.9	3.8	4.3	0.5	3.8	3.2	–0.7
Macleay-Hastings	2.2	1.3	–0.9	2.1	1.2	–0.8	4.8	2.0	–2.8
Macquarie	2.6	2.3	–0.3	1.5	2.1	0.6	4.0	3.6	–0.4
Mid North Coast	3.5	1.4	–2.2	2.4	1.8	–0.6	2.9	2.2	–0.7
Monaro	0.7	0.8	0.1	0.9	0.9	0.0	1.0	0.5	–0.5
Murray	0.6	0.7	0.2	0.4	0.4	0.0	0.5	0.4	–0.1
Murrumbidgee	0.0	0.7	0.7	0.0	0.6	0.6	0.0	1.0	1.0
New England	1.5	1.2	–0.4	1.6	1.0	–0.6	1.9	0.9	–1.0
North West	2.4	1.8	–0.6	2.0	2.0	0.0	3.0	2.3	–0.7
Orana	1.6	1.7	0.1	1.6	3.1	1.4	2.9	2.8	–0.1
Richmond	1.1	0.9	–0.2	1.0	0.9	–0.1	2.2	1.8	–0.4
Riverina	3.0	2.3	–0.7	2.9	2.6	–0.4	3.9	2.6	–1.3
South Coast	0.6	1.1	0.5	0.5	0.7	0.2	1.3	2.5	1.2
Southern Tablelands	1.1	0.9	-0.3	0.9	0.9	0.0	3.3	1.1	–2.2
Tweed Valley	2.9	1.8	–1.1	1.9	2.2	0.3	3.0	1.9	–1.1
Port Macquarie Base Hosp	3.0	3.2	0.2	2.0	4.5	2.4	3.0	4.0	0.9
New South Wales	**1.8**	**1.4**	**–0.4**	**1.4**	**1.7**	**0.3**	**3.6**	**2.8**	**–0.8**

Source: Department of Health Reporting System, November 9, 1995

* Rounding during addition may produce minor inconsistencies in totals.

TABLE 8

ADMISSIONS TO HOSPITAL, APRIL-OCTOBER 1995

Area/District/Institution	ADMISSIONS – ALL PROGRAMS*			ELECTIVE SURGERY ADMISSIONS		
		Increase			Increase due to Waiting List Reduction Program	
	Total	Number	%	Total	Number	%
Central Sydney	79,187	3,406	4.5	16,953	1,637	10.7
Northern Sydney	73,958	2,881	4.1	13,906	771	5.9
Western Sydney	76,706	2,969	4.0	13,863	1,432	11.5
Wentworth	26,760	3,238	13.8	5,558	836	17.7
South Western Sydney	74,123	7,242	10.8	16,672	1,494	9.8
Central Coast	32,560	2,440	8.1	5,930	887	17.6
Hunter	66,335	1,353	2.1	13,004	1,625	14.3
Illawarra	37,340	1,503	4.2	7,366	699	10.5
South Eastern Sydney	109,088	2,595	2.4	24,381	370	1.5
Royal Alexandra Hospital	11,490	638	5.9	3,290	216	7.0
Barwon	4,548	–132	–2.8	735	33	4.7
Castlereagh	4,567	266	6.2	655	100	18.0
Central West	10,661	–48.5	–4.4	2,068	–551	–21.0
Clarence	5,445	334	6.5	684	172	33.6
Evans	9,859	226	2.3	2,445	475	24.1
Far West	4,392	113	2.6	973	214	28.2
Hume	11,288	856	8.2	3,636	839	30.0
Lachlan	6,168	485	8.5	987	–17	–1.7
Lower North Coast	8,295	405	5.1	2,377	601	33.8
Macleay-Hastings	10,006	334	3.5	2,645	861	48.3
Macquarie	12,939	804	6.6	2,495	420	20.2
Mid North Coast	9,422	977	11.6	1,675	756	82.3
Monaro	4,047	–139	–3.3	732	61	9.1
Murray	3,439	–96	–2.7	501	–77	–13.3
Murrumbidgee	7,653	–	–	1,320	64	5.1
New England	8,345	153	1.9	1,930	407	26.7
North West	13,676	26	0.2	3,348	193	6.1
Orana	3,624	175	5.1	114	5	4.6
Richmond	20,543	921	4.7	4,254	657	18.3
Riverina	12,211	528	4.5	2,878	374	14.9
South Coast	8,602	1,101	14.7	1,643	433	35.8
Southern Tablelands	7,937	234	3.0	1,459	50	3.5
Tweed Valley	9,139	393	4.5	2,303	349	17.9
New South Wales	**784,353**	**35,744**	**4.8**	**162,780**	**16,386**	**11.2**

Source: Department of Health Reporting System, Waiting List Information System: Area and District returns.

* Total all admissions, all NSW public hospitals

PUBLIC HEALTH EDITORIAL STAFF

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

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, to the editor, NSW Public Health Bulletin, Locked Mail Bag 961, North Sydney 2059. Facsimile (02) 391 9029.

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THE SEDUCTION OF MEDICINE BY HEALTH OUTCOMES: FROM MEANING TO MEASUREMENT

Graham Vimpani
Director, Child, Adolescent and Family Health Service
Professor of Community, Child and Family Health
Newcastle University Wallsend Campus

The pattern of contemporary morbidity in young Australians (15-20 per cent mental health problems) is indicative of widespread community, social and family dysfunction.

Much political and professional energy is devoted to identifying strategies which will deal with the problems of contemporary morbidity. But these efforts are often controlled by those who have been heavily influenced by the advances of modern 'scientific' medicine. This has been dominated in the past hundred years by the widespread application of the germ theory of disease, with its focus on interrelationships between single causes and effects. Even when it is clear that health and social problems – or outcomes – have multiple causes, e.g. intellectual disability, interventions usually focus on changing things that make a measurable but marginal contribution. Consequently, rather than unravelling what it is about the quality of a domestic environment that has a major impact on this outcome we tend to focus on things like lead.

The health outcomes approach focuses attention on "interventions in the health – or other – social systems which have measurable effects and are able to demonstrate health gains". Sometimes, however, the gains are both minuscule and peripheral. The assumption that adolescent suicide in rural areas will be reduced by reducing the level of gun ownership, improving skills of community health workers and increasing the numbers of mental health workers ignores the fact that gun ownership has always been more common in rural communities and that it has not been a decline in community mental health services which has resulted in the rising suicide rates. Community mental health services have always been deficient in rural areas. But it is easy to demonstrate improvements in these proxy measures and continue to ignore the underlying social malaise that has given rise to the problem. Indeed, it is a clinician's reductionist response to a public health problem which needs to be tackled closer to its source rather than so far downstream.

This approach is another example of the consequences of reductionism and specialisation, of people acting like carpenters and seeing every problem as a nail waiting to be hit with a hammer. What is needed is an integrative approach. For this to occur people will need to break out of the mould into which specialisation has placed them and begin to collaborate with a much broader range of disciplines. By becoming "feral scientists" (to use the term coined by Charles Birch) and attempting to achieve transdisciplinary academic synthesis they may gain an understanding of how the different styles of discourse, research and scholarship can contribute to a deeper, more holistic, understanding of the contemporary human dilemma.

Society finds it difficult to deal with many contemporary social health problems because of their complexity. Further, effective solutions pose a challenge to existing social norms and values. To deal with them effectively would require a profound social and ethical change which would be especially threatening to those who control resource allocation. These are mostly middle-aged and usually male decision makers and politicians whose agenda reflects that of much of society – to enable the pursuit of the virtue of selfishness (to quote Ayn Rand) and the continuing growth of personal autonomy and self-actualisation while hoping that any consequent social and family costs won't be too great. If as much money and energy as has been directed towards immunisation coverage and environmental lead reduction was spent on translating the knowledge we have about the roots of family and community dysfunction into developing strategies to address these problems, the overall level of community well-being would be much better.

The focus on health outcomes is often at the top of the pyramid rather than at its base. What is needed, instead, is the development of reliable and valid ways of understanding, describing and measuring the characteristics of individuals, families and communities (including schools). Through this not only can the links between good environments and good outcomes be more clearly demonstrated (and vice versa) but also what it is about some individuals that grants them resilience. Sometimes these characteristics are best described rather than measured. In many instances, therefore, qualitative approaches to problem solving are more likely to help our understanding rather than the quantitative approaches which most health professionals have inherited as the only way to interpret reality.

The challenge for the "health outcomes movement" is to incorporate this approach into its theory and practice.

PUBLIC HEALTH ABSTRACTS

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

BRAIN DAMAGE AND SHEEP DIP

Sheep dip commonly contains organophosphate compounds which in repeated low doses may result in damage to the nervous system. A study of British farmers has demonstrated that they performed significantly worse than controls in tests to assess sustained attention and speed of information testing.

Stephens R, Spurgeon A, Calvert IA et al. Neuropsychological effects of long-term exposure to organophosphates in sheep dip. *Lancet* 1995; 345:1135-1139.

SMOKING INCREASES RISK OF BREAST CANCER

It has been suggested that cigarette smoking may reduce cancer of the breast. This hypothesis is based on alterations to hormone levels associated with smoking. But there appears to be a direct carcinogenic effect on the breast associated with smoking. This has been confirmed by a Danish-based study, which shows there is a pronounced increased risk of breast cancer in women who have smoked for more than 20 years (odds ratio 1:6).

Bennicke K, Conrad C, Sabroe S et al. Cigarette smoking and breast cancer. *Br Med J* 1995; 310:1431-1433.

EATING FRUIT AND VEGETABLES – HOW MUCH?

Health information should be clear and unambiguous. Advice to eat more fruit and vegetables gives consumers no indication of the quantities involved. If advice is being given that say five portions of fruit and vegetables are good for you, these portions need to be defined. A portion might equal a cupful of berries, two spoonfuls of beans, two apples and a small bowl of salad. On the other hand, a couple of slices of tomato in a sandwich or a few mushrooms on a piece of chicken should not count as a portion.

Williams C. Healthy eating: clarifying advice about fruit and vegetables. *Br Med J* 1995; 310:1453-1455.

FOLIC ACID AND PREVENTION OF NEURAL TUBE DEFECTS

The evidence appears conclusive that an increase in the intake of folic acid among women planning pregnancy will prevent most neural tube defects. Some are recommending a population approach to this issue. It is proposed that folic acid be added to common foodstuffs such as flour, in the same way that white flour is commonly fortified with vitamins and minerals. Such an approach might substantially reduce the problem. About 350 infants with neural tube defects are born in Australia each year.

Wald NJ, Bower C. Folic acid and the prevention of neural tube defects. *Br Med J* 1995; 310:1019-1020.

SUICIDE IN OLDER AUSTRALIANS

Recent concern about the increase in youth suicide has overshadowed the fact that suicide rates remain highest in men aged 75 years and over. More than 90 per cent of such victims have a mental disorder, predominantly depression (75 per cent). Most of those with depression are inadequately treated. Early recognition and treatment are priorities.

Draper BMJ. Prevention of suicide in old age. *Med J Aust* 1995; 162:533-534.

FIREARM CONTROL IN QUEENSLAND REDUCES SUICIDE

Most firearm deaths are suicides and most homicides due to firearms are triggered by personal distress. More than 85 per cent of 587 firearm deaths in Queensland between 1980 and 1989 were associated with personal distress as opposed to crime or accidents. In January 1992 a new Weapons Act took effect. The essential feature of the Act is a 28-day "cooling off" period between applying for and receiving approval to buy a weapon. Although the evidence is preliminary, there appears to be a statistically significant reduction in suicides due to firearms of about 35 per cent. However, there was little change in rural areas.

Cantor CH, Slater PJ. The impact of firearm control legislation on suicide in Queensland: preliminary findings. *Med J Aust* 1995; 162:583-585.

THE VALUE OF GERIATRIC INTERVENTIONS

Ten years ago Rubenstein et al reported the astonishing effectiveness of a geriatric evaluation and assessment unit. Two recent US studies produced conflicting results. One study confirmed the value of geriatric assessment and rehabilitation, while the other showed no difference to standard care. The authors argued that the lack of a difference in the second study was due to general physicians now possessing the special skills of geriatricians. Their overall conclusion was that specialised geriatric programs are of real value.

Capion EW. The value of geriatric interventions. *N Engl J Med* 1995; 332:1376-1377.

INFECTIOUS DISEASES

NOTIFICATION TRENDS

Notification rates were higher than historical levels in September 1995 (Figure 3) and October 1995 (Table 9) for gastroenteritis and rubella. Notifications for foodborne illness (not otherwise specified) were also elevated in October (Table 9). There were lower notification rates in September 1995 for *Haemophilus influenzae* type b (Hib), measles, pertussis and Q fever.

Hib notifications continue a pleasing long-term downward trend. It appears the trend for pertussis notifications to increase, which has been observed since April 1995, may have peaked in August 1995. The usual spring and early summer peak in measles notifications has not been observed in 1995, and the number of measles notifications has been significantly lower than the historical average (Figures 3 and 4).

High notification rates were reported from the North Coast Public Health Unit (PHU) for arboviral infection, hepatitis C, Q fever, pertussis and rubella.

RUBELLA

Rubella notification rates continued a marked upward trend (Figure 5). The highest rates for the period January 1-October 31, 1995 were reported from the Central West PHU (65 cases per 100,000 population), followed by the North Coast (28/100,000), Western NSW (17/100,000) and Western Sydney (14/100,000). As expected, the largest proportion of cases occurred in adolescent and young adult males.

GASTROENTERITIS IN AN INSTITUTION

As described in the October 1995 issue of the *Public Health Bulletin*, the numbers of notifications for gastroenteritis in an institution have been high since July 1995. There appears to have been a peak in notifications in August, with large numbers of notifications also observed in September. At the time of writing notifications were still being received for October.

Q FEVER

Q fever was discussed in the July issue of the *Bulletin*. Seventeen notifications were received in August from the South East PHU, involving abattoir workers at Young. The highest rate for the period January 1-October 31 was observed in Western NSW (31/100,000).

EQUINE MORBILLI VIRUS

In October 1995 a horse breeder in Queensland died from equine morbilli virus (EMV). The appearance of EMV highlights the importance of innovative surveillance systems capable of detecting unusual and severe illness patterns and triggering prompt and appropriate public health responses.

Dr Jeremy McAnulty, the Acting Medical Adviser in Infectious Diseases for the NSW Health Department, has prepared an article outlining the key facts on EMV. This has been distributed to all PHUs for further communication to general practitioners and to emergency department and other hospital staff, in a proactive attempt to find any further cases. The text of the article is reprinted below.

Open letter to general practitioners, hospital emergency departments and infectious diseases departments

The recent well-publicised case of equine morbilli virus (EMV) in a Queensland horse breeder represents the third reported human case. Available evidence indicates the disease is rare and not very infectious. However, more work needs to be done before we can fully understand the epidemiology of EMV. Here we review some features of reported cases and request that doctors report suspected cases to their public health unit.

Case history

On October 21, 1995 a 35-year-old man from Mackay, Queensland, died with encephalitis. The man had been ill more than a year before — in August or September 1994 — with a mild meningo-encephalitis. In mid-September 1995 he developed personality change and encephalitis. Later he developed status epilepticus, became comatose and required ventilation. An MRI scan indicated widespread brain inflammation. He had no respiratory symptoms until he developed aspiration pneumonia. Serology indicated he had a rapidly rising antibody titre to EMV in blood and cerebrospinal fluid, indicating infection at the time of death. No other cause of his illness was identified.

Exposures

In August 1994 the horse breeder reportedly had close contact with two sick horses and assisted his wife, a veterinary surgeon, in their post-mortem examinations. The first horse had died on the man's property within 24 hours of developing a respiratory illness with renal involvement (at the time thought to be avocado poisoning). The second horse from the property died 7-10 days later after developing neurological symptoms (at the time thought to be due to snake bite). Serological testing since the man's diagnosis has indicated the second horse was infected with EMV.

Review of veterinary records from the man's property revealed no evidence of other possible EMV infections. The property has about 90 horses and has been secured voluntarily. All these horses and others in the Mackay area are being surveyed for evidence of EMV. Four horses from the Mackay property were moved to NSW in the past 15 months. All are well and have no serological evidence of past or present EMV infection.

The Hendra outbreak

In September 1994 a well-publicised outbreak of severe respiratory illness was reported among two men and 21 horses from a racing stable at Hendra, a Brisbane suburb. Illness among the horses was characterised by acute onset of high fevers and severe respiratory difficulty. Fourteen horses died. A 49-year-old trainer and 40-year-old stable hand who had contact with a dying mare developed severe influenza-like illnesses two weeks later. The trainer died after six days in intensive care and post-mortem examination revealed severe interstitial pneumonia.

As a result of the Hendra outbreak, the Queensland Department of Primary Industries tested 292 race horses from the central Queensland coast in October 1994. None had evidence of EMV infection. A large percentage of horses in the Hendra area was also tested and seven — all associated with the original outbreak — were positive and were destroyed. Until the report of the Mackay case, subsequent surveillance did not detect any other cases in horses or other animals. Investigations of suspected horses and cats and limited serosurveys of local wildlife were all negative.

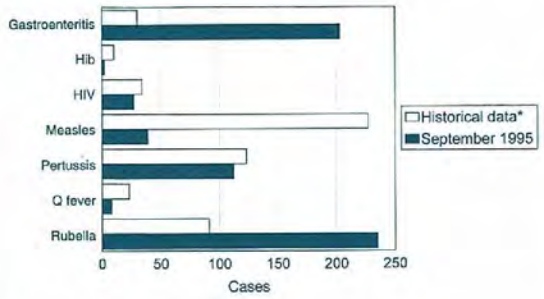
TABLE 9

INFECTIOUS DISEASE NOTIFICATIONS FOR NSW, 1995 BY SELECTED MONTH OF ONSET FOR NOTIFICATIONS RECEIVED BY OCTOBER 31, 1995

Condition	Jul	Aug	Sep	Oct	Total
Adverse event after immunisation	3	3	4	3	13
AIDS	23	23	13	15	74
Arboviral infection	13	14	9	2	38
Cholera	1	–	–	–	1
Foodborne illness (NOS)	14	8	17	37	76
Gastroenteritis (instit.)	230	430	203	42	905
Gonorrhoea infection	16	34	35	14	99
H. influenzae epiglottitis	–	1	1	–	2
H. influenzae meningitis	2	–	1	1	4
H. influenzae septicaemia	–	2	–	–	2
Hepatitis A – acute viral	17	32	36	31	116
Hepatitis B – acute viral	5	3	3	–	11
Hepatitis B – chronic/carrier	29	47	36	11	123
Hepatitis B – unspecified	302	332	344	105	1,083
Hepatitis C – acute viral	6	7	–	–	13
Hepatitis C – unspecified	525	637	544	209	1,915
Hepatitis D – unspecified	–	–	2	–	2
Hepatitis, acute viral (NOS)	1	–	–	1	2
HIV Infection	32	36	46	38	152
Hydatid disease	1	–	1	–	2
Legionnaires' disease	7	2	1	1	11
Leprosy	–	–	1	–	1
Leptospirosis	1	–	–	–	1
Listeriosis	–	–	2	–	2
Malaria	3	4	1	–	8
Measles	29	53	39	30	151
Meningococcal infection (NOS)	1	1	3	1	6
Meningococcal meningitis	12	8	6	5	31
Meningococcal septicaemia	2	5	2	–	9
Mumps	2	1	1	–	4
Mycobacterial atypical	27	12	4	2	45
Mycobacterial infection (NOS)	6	5	1	1	13
Mycobacterial tuberculosis	37	26	18	3	84
Pertussis	118	162	112	71	463
Q fever	18	31	8	3	60
Rubella	64	119	235	65	483
Salmonella (NOS)	71	75	57	41	244
Syphilis infection	72	57	55	25	209
Tuberculosis – non active	7	10	3	2	22
Typhoid and paratyphoid	1	–	2	–	3

FIGURE 3

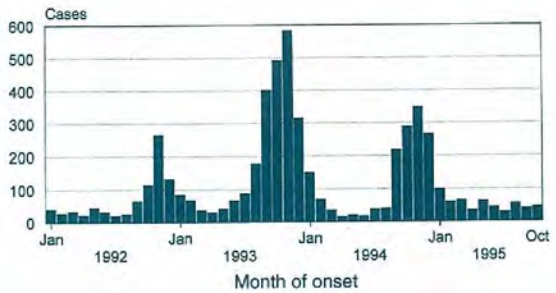
SELECTED INFECTIOUS DISEASES: NSW SEPTEMBER NOTIFICATIONS, 1995 COMPARED WITH HISTORICAL DATA



* Historical data: the average number of notifications diagnosed in the same month in the previous three years.

FIGURE 4

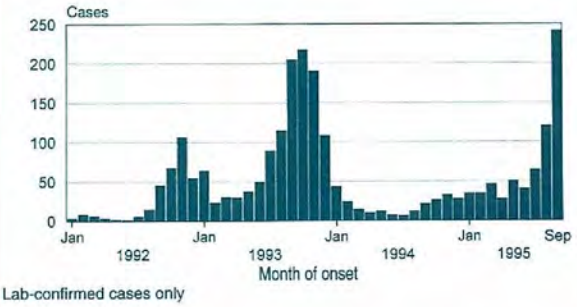
MEASLES NOTIFICATIONS NSW 1992-1995, BY DATE OF ONSET



Source: IDDS

FIGURE 5

RUBELLA NOTIFICATIONS NSW 1992-1995, BY DATE OF ONSET



Lab-confirmed cases only

Source: IDDS

No link has been found between the September 1994 outbreak and the Mackay property.

The virus

EMV was first identified in viral studies of samples taken from the Hendra outbreak. EMV is a newly recognised morbilli virus, distantly related to measles, canine distemper and rinderpest[1].

The disease in horses[2]

EMV infection in horses seems to lead to anorexia, depression, fever and increasingly severe respiratory disease, terminating in frothy nasal discharge. Post-mortem changes include very heavy, grossly oedematous and congested lungs with thick tenacious frothy exudates in the airways. Microscopically, the lung changes are consistent with interstitial pneumonia with proteinaceous alveolar oedema, haemorrhage, alveolar necrosis and necrosis of the walls of small blood vessels. Syncytial giant cells are present in the epithelium of the lung capillaries and arterioles.

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The disease in humans
With only three reported cases, no typical clinical picture of EMV infection has yet emerged in humans. Clearly, two distinct syndromes are likely — one characterised by an influenza-like illness and one by encephalitis. Two of the three reported cases have died.

What is the natural host?
Little is known about the pathogenesis and epidemiology of EMV infection. In vitro, the virus is able to grow in cell cultures in a number of mammalian species, birds, reptiles, amphibians and fish. However, after inoculation of mice, guinea pigs, chickens, rabbits, cats and dogs, only cats and guinea pigs developed disease. A serosurvey of 500 cats from metropolitan Brisbane found none was positive for EMV.

How contagious is it?
All reports suggest EMV is not very contagious. It appears close contact is required for transmission to occur. Horses sharing paddocks and in adjacent stalls to infected horses in the Hendra outbreak remain free from infection. There is no evidence of person-to-person transmission.

So why are you telling me this?
All indications are that EMV infection is extremely rare and that infection in humans is very difficult to acquire. However, to evaluate the implications of EMV for human health, we need more information. We must determine the prevalence (if any) of disease and infection among people; who is at greatest risk and why; and how can any risk be minimised? Please report suspected cases!

We have reports of three people ill with EMV but with different clinical pictures. To help identify possible cases for surveillance purposes, we have developed a preliminary case definition. Suspected cases are defined as a person:

- aged >1 and <50 years critically ill with an acute respiratory illness or encephalitis;
- who has been previously healthy, with no underlying conditions; and
- in whom preliminary testing has failed to reveal the cause of illness.

All doctors are urged to report suspected cases (diagnosed in the past or present) to their Public Health Unit. The PHU will help arrange appropriate serological testing if required.

Conclusion
There is no indication that EMV poses a significant public health risk. Nevertheless, reporting suspected cases (if any), together with studies to determine the natural animal reservoir of EMV, and the prevalence (if any) of EMV in animal and human populations, will be vital for evaluating the epidemiology and public health significance of this newly recognised pathogen.

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1. Murray K et al. A morbilli virus that caused fatal disease in horses and humans. *Science* 1995; 268:94-97.
2. Queensland Department of Primary Industries. Update on equine morbilli virus infection (acute equine respiratory syndrome). Animal Health Bureau, July 12, 1995.

GONOCOCCAL ISOLATE SURVEILLANCE

The following report, focusing on the three-month period from July 1 to September 30, 1995, is based on information provided by the Gonococcal Reference Laboratory in the Microbiology Department of the Prince of Wales Hospital, Randwick, Sydney.

In the nine months to September 30, 458 gonococcal isolates were referred to the laboratory. By comparison, 496 isolates were reported for the whole of 1994. An increase in the number of isolates was particularly evident in the six months to June 30, with a decline to more typical numbers in the quarter to September 30.

A total of 125 gonococci isolates was referred in the quarter to September 30. Of these, 120 remained viable for further examination. This compares with the 168 isolates in the quarter to June 30, 1995 and 132 isolates examined in the quarter to September 30, 1994.

It is widely recognised that the number of cases of gonorrhoea tends to decrease in the colder months. The decrease has been attributed to factors including the prevalence of winter colds and respiratory tract infections which are presumed to limit social activity and attract the intercurrent use of antibiotics.

Of the 125 isolates examined in the quarter to September 30, 1995, 115 were from males and nine from females. Sex and site of infection were not stated in one instance.

The sites of infection in males were as follows:

Urethra	88
Pharynx	5
Ano-rectum	20
Disseminated gonococcal infection	1
Not stated	1

The endocervix/vagina was the site of infection for all nine isolates from females.

The male-to-female ratio of infection in the quarter to September 30, 1995 was 12.8:1, compared with 6.3:1 in the quarter to June 30, 1995 and 5.6:1 in the quarter to September 30, 1994.

In the quarter to September 30, 1995, 18 per cent of isolates from men were ano-rectal in origin while 4 per cent were pharyngeal. In the equivalent period in 1994 the respective figures were 11 per cent and 6 per cent.

Antibiotic sensitivity patterns

Penicillins (including penicillin, ampicillin and amoxycillin)
For many years a high proportion of the gonococci isolated in NSW has been resistant to the penicillins. In the quarter to September 30, 1995, 38 isolates (32 per cent) were penicillin resistant, either because they were penicillinase-producing *Neisseria gonorrhoeae* (PPNG) (11 strains – 9 per cent) or through chromosomally mediated mechanisms (27 strains – 23 per cent). Details of the acquisition of PPNG infection were available in six instances and all six cases were imported.

Only a very small proportion of isolates is fully sensitive to penicillin, with low minimum inhibitory concentrations (MIC) in the range 0.004-0.03 mg/l. In 1990 these strains reappeared and came to comprise about one-third of all isolates. They represent about 6-7% of isolates now.

Penicillins should not be used for routine treatment of gonorrhoea in NSW.

Ceftriaxone
All isolates examined in this quarter were sensitive to ceftriaxone. This injectable cephalosporin remains very active against gonococci.

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

Quinolones (ciprofloxacin, norfloxacin, enoxacin)
Since October 1994 there has been an increase in the number of quinolone-resistant gonococci isolated. Furthermore, the levels of resistance, as determined by quantitative sensitivity testing, have reached unprecedented levels.

However, in the quarter to September 30, 1995 the number and proportion of quinolone resistant isolates has returned to those seen before the October-December 1994 quarter. Five isolates (4 per cent) were quinoline resistant, and all of these were in the high MIC range (4-16 mg/l).

Before these strains appeared, the recommended treatment regimen of a single 500mg dose of ciprofloxacin had been adequate to cure nearly all infections encountered. But no dose of quinolone antibiotic (however high) would eliminate infections with such high levels of resistance.

World Health Organisation sources continue to report that quinolone resistance is increasing rapidly in nearby countries visited frequently by Australians.

A recent report from Canada noted a similar increase in quinolone-resistant gonococci in British Columbia with the cases being derived from contacts in Asia.

Continued monitoring of gonococcal resistance to quinolones is essential. Strains from apparent treatment failures and those from patients entering or returning to Australia warrant particularly close examination.

Tetracycline
Recent reports from the Gonococcal Reference Laboratory have highlighted an interesting form of plasmid-mediated high-level tetracycline resistance in gonococci which has also emerged in the past decade.

While tetracyclines are not recommended for treatment of gonorrhoea in NSW, the spread of tetracycline resistant *N gonorrhoeae* (TRNG) throughout the world has been of particular concern. Strains are examined routinely for the presence of this tetracycline resistance.

In the quarter to September 30, 1995 the number and proportion of TRNG isolated in NSW fell. Nine TRNG (8 per cent) were detected, a rate more in keeping with the experience at the same time last year, but much less than the 15 per cent of TRNG seen in the quarter to June 30, 1995.

Again it should be remembered that some countries close to Australia have high numbers of TRNG. Five of the nine TRNG were also PPNG.

Comment
In general, the great majority of antibiotic-resistant isolates seen in NSW have been acquired from contacts overseas. In the quarter to September 30, 1995 the numbers and proportions of PPNG, TRNG and quinolone-resistant gonococci were all lower than in the previous quarter.

TABLE 10

SUMMARY OF NSW INFECTIOUS DISEASE NOTIFICATIONS OCTOBER 1995

Condition	Number of cases notified			
	Period		Cumulative	
	Oct 1994	Oct 1995	Oct 1994	Oct 1995
Adverse reaction	–	3	32	29
AIDS	59	15	489	264
Arboviral infection	6	2	366	497
Brucellosis	–	–	4	2
Cholera	–	–	–	1
Diphtheria	–	–	–	–
Foodborne illness (NOS)	10	37	157	353
Gastroenteritis (instit.)	13	42	267	1,004
Gonorrhoea	32	14	307	319
H influenzae epiglottitis	1	–	21	5
H influenzae B – meningitis	1	1	14	9
H influenzae B – septicaemia	–	–	11	6
H influenzae infection (NOS)	1	–	9	2
Hepatitis A	53	31	464	424
Hepatitis B	521	116	3,881	3,799
Hepatitis C	831	209	7,804	6,410
Hepatitis D	4	–	19	14
Hepatitis, acute viral (NOS)	–	1	1	2
HIV infection	35	38	372	406
Hydatid disease	3	–	15	11
Legionnaires' disease	4	1	57	60
Leprosy	–	–	3	2
Leptospirosis	–	–	13	4
Listeriosis	1	–	7	9
Malaria	10	–	166	85
Measles	288	30	892	513
Meningococcal meningitis	12	5	70	61
Meningococcal septicaemia	5	–	35	20
Meningococcal infection (NOS)	4	1	17	16
Mumps	4	–	10	9
Mycobacterial tuberculosis	33	3	358	300
Mycobacterial – atypical	38	2	435	292
Mycobacterial infection (NOS)	3	1	34	51
Pertussis	126	71	1,241	1,029
Plague	–	–	–	–
Poliomyelitis	–	–	–	–
Q fever	21	3	219	161
Rubella	26	65	175	713
Salmonella infection (NOS)	87	41	907	968
Syphilis	92	25	919	671
Tetanus	1	–	3	–
Typhoid and paratyphoid	1	–	29	31
Typhus	–	–	–	–
Viral haemorrhagic fevers	–	–	–	–
Yellow fever	–	–	–	–

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TABLE 11

INFECTIOUS DISEASE CUMULATIVE NOTIFICATIONS FOR NSW, 1995 RECEIVED BY OCTOBER 31, 1995

	PUBLIC HEALTH UNIT																	
Condition	**CCA**	**CSA**	**CW**	**ESA**	**HUN**	**ILL**	**NC**	**ND**	**NSA**	**SE**	**SSA**	**SW**	**SWS**	**WEN**	**WN**	**WSA**	**U/K**	**Total**
AIDS	2	63	1	84	10	4	25	–	29	–	14	–	12	7	–	13	–	264
Arboviral infection	7	4	–	7	12	24	196	46	5	155	4	13	1	2	18	3	–	497
Brucellosis	1	–	–	–	–	–	–	–	–	–	–	–	1	–	–	–	–	2
Cholera	–	–	–	1	–	–	–	–	–	–	–	–	–	–	–	–	–	1
Gonorrhoea infection	2	41	11	139	7	13	15	6	16	8	18	1	15	3	15	9	–	319
Hepatitis B – acute viral	–	3	1	13	–	–	3	2	–	1	1	–	3	–	11	5	–	43
Hepatitis B – chronic/carrier	15	–	14	222	–	–	8	10	3	–	13	–	–	9	8	99	–	401
Hepatitis B – unspecified	18	374	9	58	77	86	55	12	438	27	492	20	1,190	29	9	461	–	3,355
Hepatitis C – acute viral	1	–	1	5	–	–	–	–	–	1	–	–	–	2	41	1	–	52
Hepatitis C – unspecified	154	681	267	965	396	394	665	188	460	192	412	177	728	114	26	537	–	6,358
Hepatitis D – unspecified	–	–	–	1	–	–	5	1	1	–	1	1	3	–	–	1	–	14
Hepatitis, acute viral (NOS)	–	–	–	1	–	–	–	–	–	–	–	–	–	–	–	1	–	2
HIV infection	9	72	3	133	15	11	6	2	21	–	17	5	24	6	1	18	63	406
Hydatid disease	–	–	1	1	–	1	1	–	1	–	–	2	3	–	–	1	–	11
Legionnaires' disease	1	2	–	6	10	6	1	2	8	–	–	–	3	2	1	18	–	60
Leprosy	–	1	–	–	–	–	–	–	1	–	–	–	–	–	–	–	–	2
Leptospirosis	–	–	–	–	1	–	1	2	–	–	–	–	–	–	–	–	–	4
Malaria	4	5	–	8	9	4	8	1	20	2	3	3	4	3	–	11	–	85
Meningococcal infection (NOS)	1	–	–	3	1	–	3	–	–	–	4	1	2	–	1	–	–	16
Meningococcal meningitis	7	1	5	5	8	9	4	2	7	3	2	–	5	1	–	2	–	61
Meningococcal septicaemia	–	3	–	–	5	–	1	1	2	1	1	1	3	2	–	–	–	20
Mycobacterial atypical	8	28	2	71	24	6	13	8	32	3	22	3	35	12	6	18	–	292
Mycobacterial infection (NOS)	4	5	–	1	–	–	4	1	4	–	4	–	19	2	–	7	–	51
Mycobacterial tuberculosis	4	21	1	16	8	5	4	3	33	1	33	3	92	3	4	69	–	300
Q fever	–	1	9	–	12	4	40	28	–	17	–	2	1	–	46	1	–	161
Salmonella infection	–	–	–	–	1	–	–	–	–	–	–	–	–	–	–	–	–	1
Syphilis infection	5	53	11	141	15	13	58	40	33	6	41	3	99	14	96	43	–	671

TABLE 12

VACCINE PREVENTABLE AND RELATED CONDITIONS, CUMULATIVE NOTIFICATIONS FOR NSW, 1995 BY PUBLIC HEALTH UNIT, RECEIVED BY OCTOBER 31, 1995

	PUBLIC HEALTH UNIT																
Condition	**CCA**	**CSA**	**CW**	**ESA**	**HUN**	**ILL**	**NC**	**ND**	**NSA**	**SE**	**SSA**	**SW**	**SWS**	**WEN**	**WN**	**WSA**	**Total**
Adverse event after immunisation	–	–	–	1	1	–	6	2	–	5	3	4	–	4	–	3	29
H. influenzae epiglottitis	–	–	1	1	–	–	1	–	–	–	1	–	–	–	1	–	5
H. influenzae infection (NOS)	1	–	–	–	–	–	1	–	–	–	–	–	–	–	–	–	2
H. influenzae meningitis	–	1	–	–	–	–	3	–	–	–	–	–	1	1	–	3	9
H. influenzae septicaemia	–	–	–	–	1	–	1	–	1	–	1	–	1	–	–	1	6
Measles	13	26	12	54	55	66	43	46	13	6	38	10	38	37	7	49	513
Mumps	–	–	–	1	–	2	2	–	2	–	–	–	–	–	–	2	9
Pertussis	35	19	18	25	56	79	293	15	83	25	48	61	67	98	11	96	1,029
Rubella	28	43	114	24	67	39	120	20	53	5	32	4	18	29	25	92	713

TABLE 13

FOODBORNE INFECTIOUS DISEASE CUMULATIVE NOTIFICATIONS FOR NSW, 1995 BY PUBLIC HEALTH UNIT, RECEIVED BY OCTOBER 31, 1995

	PUBLIC HEALTH UNIT																
Condition	**CCA**	**CSA**	**CW**	**ESA**	**HUN**	**ILL**	**NC**	**ND**	**NSA**	**SE**	**SSA**	**SW**	**SWS**	**WEN**	**WN**	**WSA**	**Total**
Foodborne illness (NOS)	16	9	3	3	188	–	3	3	4	–	1	8	70	–	23	22	353
Gastroenteritis (instit.)	7	155	–	–	132	–	47	–	132	1	141	–	–	206	2	181	1,004
Hepatitis A – acute viral	8	51	34	127	22	9	25	3	41	–	28	10	34	3	3	26	424
Listeriosis	–	1	1	3	–	–	–	1	1	1	–	–	–	–	–	1	9
Salmonella (NOS)	20	41	20	71	68	53	106	58	103	37	90	24	87	52	34	103	967
Typhoid and paratyphoid	–	2	–	8	–	–	3	–	4	–	5	–	4	1	–	4	31
Vibrio infection (non cholera)	–	–	–	1	–	–	–	–	–	–	–	–	–	–	–	–	1

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.