NEW SOUTH WALES
Public Health Bulletin

Volume 8
Number 11-12
November-December 1997

NSW HEALTH DEPARTMENT

ISSN 1034 7674
State Health Publication No. 98-0003

THE LEAD REFERENCE CENTRE

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Lead Reference Centre

The need for a comprehensive and coordinated approach to the Statewide management of lead hazards in the environment was identified in 1992 and led to the release of the NSW Government Lead Issues Paper by the Environment Protection Authority (EPA) in 1993. The NSW Interdepartmental Lead Taskforce was established in June 1993 and chaired by the EPA throughout 1993 and 1994.

In June 1993 the National Health and Medical Research Council (NHMRC) announced a new public health goal for all Australians to have a blood lead level lower than 0.48 µmol/L (10 µg/dL) and a reduction schedule for blood lead levels by 1998.

The previous NSW Government endorsed the recommendations of the NSW Interdepartmental Lead Taskforce and its working groups and the NSW Lead Management Action Plan (LMAP) in November 1994. Just before the release of the LMAP, the NSW Parliament established a Parliamentary Select Committee Upon Lead Pollution to investigate lead issues in NSW. This committee met in the second half of 1994 and released its report in December 1994. The previous Government had not considered the Parliamentary Select Committee report before the change of government occurred. The committee report, which was tabled in the NSW Parliament on May 25, 1995, raises major issues that the committee considers were not adequately addressed in the LMAP. One of the significant differences between the Select Committee report and the LMAP is the policy response to lead paint hazards.

One goal of the LMAP was to determine the extent of childhood lead exposure in NSW. A system of mandatory notification of excessive blood lead levels has been established to work towards achieving this goal. Since December 1996 pathology laboratories in NSW have been required by law to notify the appropriate Public Health Unit when a blood level exceeds 15 µg/dL (0.72 µmol/L). The notification system will assist health care professionals, particularly general practitioners, to care for, and manage, people with elevated blood lead levels. PHU staff may be able to assist GPs and patients in identifying sources of lead exposure and provide professional remediation advice.

Both the LMAP and the Parliamentary Select Committee report strongly supported the establishment of the Lead Reference Centre, to be administered by the EPA. Establishing of the Lead Reference Centre was publicly announced early in 1995.

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The Lead Reference Centre

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The Lead Reference Centre has been established within the Environment Protection Authority to coordinate the NSW Government's policy response to lead hazards in the environment and the implementation of the Lead Management Action Plan. It is funded until June 1999 by six NSW Government agencies: the Department of Housing, the Department of Public Works and Services, the Environment Protection Authority, the NSW Health Department, Roads and Traffic Authority and the WorkCover Authority. Its goal is to achieve a lead-safe NSW by minimising the health, environmental and economic impacts of lead contamination and other lead hazards for all population groups, occupations and ecosystems. A reduction in the impact of lead contamination will result in the continuing reduction of blood lead levels in the population.

The Lead Reference Centre has a broad role in coordinating the development, evaluation and reporting on NSW Government initiatives and policy responses across all levels of the NSW Government, as well as the management of lead hazards by the community, the private sector and industry. To support this effort, the centre will work with appropriate NSW Government agencies and the private sector to develop Statewide research, public education and industry training programs. The Lead Reference Centre will coordinate the development of a broad policy response and a large number of related standards, methods and guidelines required for the effective management of lead hazards in NSW.

Major projects of relevance to health care professionals include:

- the development of guidelines to manage lead hazards for environmental health officers;
- running regional briefing programs on lead for health and planning officers within local government, PHUs, WorkCover and the EPA;
- working with the Royal College of General Practitioners to develop lead teaching materials;
- the development of a major community education campaign planned for early 1998, targeting parents, renovators and lead industry workers; and
- the dissemination of educational materials for health care professionals through the health networks.

The Lead Reference Centre has six staff and is located with the Environmental Health, Food and Nutrition Branch of the NSW Health Department in the grounds of the Gladesville Hospital.

A GUIDE FOR HEALTH CARE PROFESSIONALS ON LEAD

Richard Birdsey
Lead Reference Centre

The first major publication developed by the Lead Reference Centre is *Lead Safe: A guide for health care professionals*. The booklet was launched by the Minister for the Environment, Ms Pam Allan, in September, and has been distributed through the NSW health network by the NSW Health Department's Better Health Centre.

The guide is aimed at a range of health care professionals, including general practitioners, specialists, environmental health officers, early childhood nurses and parent educators. Information is provided on lead issues facing health care professionals, including:

- identifying groups vulnerable to the effects of lead, such as pregnant women, young children, renovators and lead industry workers;
- sources of environmental lead contamination;
- health effects of lead and how to identify them;
- testing for lead; and
- management and prevention of exposures (this includes guidelines based on the National Health and Medical Research Council action guidelines).

A comprehensive information section provides health care professionals with advice to give to patients on what to do to reduce their exposure to lead. A plain-English patient fact sheet – *Lead, your health and the environment* – has been developed to accompany the booklet.

Feedback provided by focus groups indicated that most health care professionals are deluged with information and would probably not read a large booklet. To assist readers, key information has been summarised into a single short section at the front of the booklet. Important points in the summary are cross-referenced to the main body of the text, where they are expanded on. This enables the user to find out more about topics of interest quickly and easily. A comprehensive reading list on lead and a directory of contacts for more information is also provided.

Copies of the booklet and accompanying fact sheet can be obtained by contacting the NSW Environment Protection Authority's Pollution Line on 131 555 during business hours.

LEAD COMMUNITY GROUPS IN NSW

Since the 1960s health, environment, advocacy and community groups have campaigned on lead issues in Australia (Table 1). Some groups have dealt with lead contamination as part of broader health or environmental issues, including:

- historical and contemporary contamination of the environment;
- the effects on children's health;
- the export of lead waste to developing countries; and
- the regulation of industry emissions.

Several groups have focused on specific locations, concentrating their attention on developments or industries such as waste disposal facilities or chemical factories.

Four NSW community groups focus specifically on lead issues:

THE LEAD EDUCATION AND ABATEMENT DESIGN (LEAD) GROUP

The Lead Education and Abatement Design (LEAD) Group, based in Sydney, was established in 1991 by a group of parents whose children's health had been affected by lead. The group's goal is "to eliminate childhood lead poisoning by 2002 and protect the environment from lead". The LEAD Group's two main areas of activity are community education and advocacy on lead issues. The NSW Government currently funds the Group to run the Lead Advisory Service (NSW), which provides a range of free services to parents and the community, including:

- free information and advice via a free call phone service;
- referrals to a range of lead specialists;
- parent education seminars; and
- publication of a range of information materials and a newsletter.

The free call phone of the Lead Advisory Service (NSW) (1800 626 086) receives more than 300 calls a month from concerned parents.

NORTHERN LAKES ENVIRONMENTAL ACTION DEFENCE (NO LEAD)

Northern Lakes Environmental Action Defence (NO LEAD) was formed in 1990, following residents' concerns of environmental contamination from a lead-zinc smelter at North Lake Macquarie. The smelter has been in operation since last century. The group focuses on child health and the environment and has been campaigning for the reduction of emissions from the smelter and the remediation of North Lake Macquarie.

MARAYONG ACTION GROUP

The Marayong Action Group was formed in 1993 by residents living near a proposed lead-acid battery factory plant in the western Sydney suburb of Marayong. As a result of the high level of community concern, conditions were attached to the development proposal to deal with lead contamination issues. The group is still represented on the community forum set up as part of the development conditions.

ILLAWARRA RESIDENTS AGAINST TOXIC ENVIRONMENTS (IRATE)

The IRATE group was formed by residents concerned about the health and environment effects of current emissions and historical contamination from heavy industry in the Port Kembla area, south of Wollongong.

The organisation participates in a range of committees that consider environmental issues associated with lead and other pollutants. It is also involved in developing community awareness about the effects of lead.

Further information is available from Elizabeth O'Brien, 9716 0014.

TABLE 1

ORGANISATIONS INVOLVED WITH LEAD ISSUES

Group	Issue
ADI Residents Action Group	Pollution at Australian Defence Industry sites
Ardeer Residents Group	Lead-acid battery factory
Australian Chemical Trauma Alliance	Toxic chemicals and contamination
Australian Conservation Foundation	Environment
Broken Hill Residents Action Group	Lead issues in Broken Hill
Consumers Health Forum	Consumer advocacy
Friends of the Earth	Environment
Greenpeace	Environment
Illawarra Residents Against Toxic Environments (IRATE)	Heavy industry in Port Kembla
Inner Sydney Regional Council for Social Development	Leaded petrol and urban environment
Inner City Residents Assisting their Environment	Waterloo incinerator
Lead Education and Abatement Design (LEAD) Group	Community education and advocacy
Marayong Action Group	Lead-acid battery factory
National Toxics Network	Chemicals
Northern Lakes Environmental Action Defence (NO LEAD)	Lead–zinc smelter at North Lake Macquarie
Public Interest Advocacy Centre	Public interest and advocacy issues
Total Environment Centre	Environment

MEASURING THE AMOUNT OF LEAD IN INDOOR DUST: LONG-TERM DUST-FALL ACCUMULATION IN PETRI DISHES (A PILOT STUDY)

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Lead from dust is potentially the major contributor to blood lead levels in children living in urban environments in Australia[1] and is likely to make a substantial contribution to blood lead levels of children living in the vicinity of lead smelters. We undertook a trial of measuring dust in petri dishes to estimate the long-term flux of lead in indoor dust.

Conventional methods of assessing risk from lead exposure have concentrated on outdoor sampling of lead in soil and in air. Although these pathways may be important in delivering lead to a child's house, the most likely source of directly ingestible lead is dust that settles inside homes.

Risk to neurological development is assumed to be greatest in toddlers and preschool children who spend much of their time indoors. The natural actions of putting into the mouth hands, toys and other objects carrying dust are unavoidable in this age group. The risk associated with these actions is more relevant to potential interventions than measures of outdoor lead concentrations.

Among the methods used for measuring lead in indoor dust are passive wipe methods, vacuum cleaners and long-term dust-fall accumulation. Passive wipe methods (wiping a surface with a cloth) have limitations in that it is technically difficult to remove the lead from the cloth for analysis, and in that the cloths do not work well on rough surfaces (such as carpets). Vacuum cleaners need to be especially designed to pick up fine particulates and must be mains powered[2,3]. Neither of these methods allows an assessment of the time taken for the dust to accumulate.

In Australia, Gulson has pilot-tested a method for measuring dust-fall accumulation, using petri dishes.[4] In his study the petri dishes were left in suburban houses in Sydney and Broken Hill for three months. The method was useful for measuring total lead deposition (load) and sufficient dust was collected for speciation (that is, detailed analysis of lead isotopes and scanning electron microscopy). The isotopic fingerprints (identifying sources of lead) in the dust found in petri dishes were strongly correlated with the isotopic fingerprints found in children's blood.

Australia's second largest lead smelter, operated by Pasminco Metals–Sulphide, is in the suburb of Boolaroo on the northern shores of Lake Macquarie. Boolaroo and the adjacent suburbs of Argenton and Speers Point developed around the smelter. The site gradually expanded its works so that now the closest house is only 150 m from the main stack.

For years the community has been concerned about the effects of lead pollution in the area. The aims of this pilot study were to adapt and trial the petri dish method of measuring long-term dust accumulation and to quantify the amount of lead in dust falling on interior surfaces.

METHODS

Selection and setting up of petri dishes

Fifteen households were recruited for the pilot study – nine from North Lake Macquarie (Boolaroo and Speers Point) and six from other urban areas of the Hunter region.

The households at North Lake Macquarie were a convenience sample willing to cooperate with the study plan. The nearest house in the sample was 400 m from the lead smelter stack. Other households were recruited at approximately 100 m intervals from the smelter stack.

The six households from outside the North Lake Macquarie area were made up of volunteers from staff members of the Newcastle Environmental Toxicology Research Unit (NETRU). The houses were in the industrial and inner-city areas of Mayfield and Cooks Hill, and the suburban areas Adamstown, Charlestown and New Lambton.

Four petri dishes were placed in each house, in the four most-used rooms. The dishes were placed 0-2 m from floor level on objects where the dishes would not be disturbed and would not be obstructed from dust fall. The position of each dish was recorded. The dishes were secured with a piece of plasticine on the base of the dish after the mounting surface had been wiped with a tissue.

The dishes were left in the houses for 5-12 weeks. They were removed, placed flat in individual airtight bags, secured in a box and transported to the laboratory for analysis.

Laboratory methods

Polystyrene petri dishes 85 mm in diameter were prepared by the Trace/Toxic Element Unit, Division of Clinical Chemistry, Hunter Area Pathology Service. Petri dishes were soaked overnight in an alkaline detergent, thoroughly rinsed with deionised water, then soaked for a further 48 hours in ultra-pure 2 per cent nitric acid. Finally, the petri dishes and their lids were removed from the acid, rinsed three times with ultra-pure deionised water (resistivity >18M), and dried in a low temperature drying oven. When dry, each dish (with its corresponding lid) was numbered and accurately weighed.

Assays of lead content were carried out in accordance with draft international standard ISO/DIS 9855, except that the acids were used in a concentration of 7M nitric acid and 6M hydrochloric acid. This procedure is based on nitric acid and hydrochloric acid digestion and final determination by graphite furnace atomic absorption spectrometry.

If the amount of collected dust was sufficient to allow accurate weighing, the lead content was reported as:

- concentration, in µg Pb/gm dust
- loading, in µg Pb/area (m^2)
- flux, in µg Pb/area (m^2) per month

If insufficient dust was collected to allow accurate weighing, only lead loading and flux were reported.

Statistical analysis

Lead loading and concentrations were approximately log normally distributed and were transformed accordingly for analysis. Since the petri dishes were left out for various periods, loading estimates were firstly standardised to a four-week period: thus, if the dish was placed for five weeks, the flux was calculated as four-fifths of that measured.

Initial graphical review of data also highlighted a relationship between the height of the dish from the floor and the lead loading: the lower the dish, the higher the lead level. In order to standardise for differences in height, a linear regression model was used to estimate the relationship between ln lead loading and height. This regression equation was then used to standardise loading measures to a height of 1 m.

Therefore, the final results are presented as µg lead/m² per month at 1 m. The arithmetic average of results from each house is also presented.

The relationship between distance from the smelter and lead flux was estimated by simple linear regression.

RESULTS

Lead flux in relation to distance from the smelter

The main results are presented in Figures 1 and 2. Figure 1 shows a scattergram of the lead flux (µg lead/m² per month) levels for each dish, while Figure 2 shows a scattergram of the average lead flux (µg lead/m² per month) for each household. The results for houses in North Lake Macquarie are shown on the left of both scattergrams, plotted against distance from the smelter. The results for houses from other Newcastle areas are shown at the right of the scattergrams.

There was a clear trend towards decreasing lead flux with distance from the smelter ($P<0.0001$, $r^2=0.94$). The average lead flux levels for houses 600 m to 1 km from the smelter were approximately 30 per cent of those from houses 400-600 m from the smelter. The average lead flux for houses in North Lake Macquarie was 1,148 µg lead/m² per month.

The houses from other Newcastle areas had an average lead flux of 237µg lead/m² per month. Interestingly, one house was extensively repainted during the study, which included sanding back very old paint. The lead flux results for this house were considerably above those for other areas of Newcastle and comparable to those in North Lake Macquarie. The second highest readings came from a house at Mayfield between two major highways.

DISCUSSION

This study has shown the potential usefulness of using petri dishes to measure long-term dust accumulation in a community setting. The method was relatively simple and caused minimal inconvenience to householders. The results obtained over 4-12 weeks appear to reflect the gradients in air and soil lead concentrations in the vicinity of a lead smelter. The lower levels in Newcastle homes away from the smelter are consistent with less lead exposure.

The method also appeared to be sensitive to sources of lead such as sanding leaded paint during renovations.

FIGURE 1

Scattergram of log lead flux, standardised to a four-week period and 1m height: individual dishes

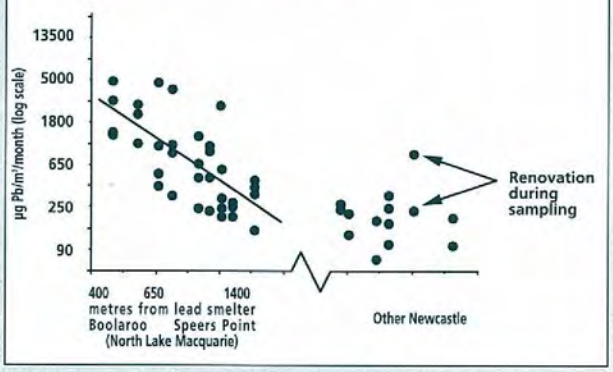
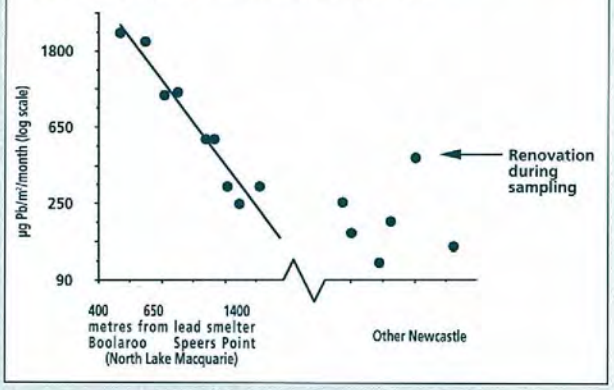


FIGURE 2

Scattergram of log lead flux, standardised to a four-week period and 1m height: mean for each participant



Although the study was not designed to include an investigation of the effects of leaded paint on lead loading, the level for the one house known to have been renovated during the study was markedly higher than those for other houses in the Newcastle area, and deserve further investigations.

The petri dish method has the advantage of measuring lead loading and flux in addition to concentration, that is, it gives an estimate of the total amount of lead accumulating in a home over a specific period in addition to the amount of lead in each gram of dust.

One way to conceptualise the difference between lead loading and lead concentration is to consider the effect of cleaning a house with a wet cloth. While this would reduce the total amount of lead available for ingestion by reducing the amount of dust in the house, it would not alter the concentration of lead in the dust remaining. The lead loading measure done after cleaning would reflect the lower levels of lead remaining.

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LEAD IN DUST AND SOIL FROM DAY-CARE CENTRES

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Lead is a neurotoxin and, even at low levels in the blood, is considered to result in lowering of IQ, behavioural problems and learning difficulties in children[1].

More than 90 per cent of the lead in Sydney urban air is still derived from motor vehicle emissions[2]. With many schools and day-care centres located on major traffic thoroughfares, and often with traffic lights located outside the schools for pedestrian safety, there is a potential risk of rooms and play areas becoming contaminated with lead. The main pathways of petrol lead to children are via direct airborne transport from the roads, or indirectly by re-entrainment of dust/soil by the wind. This airborne lead may be deposited onto surfaces such as desks, floors, benches and paved areas, and hence ingested via hand-to-mouth activity.

Airborne lead may also be deposited in ceiling cavities and, if disturbed, result in contamination of surrounding areas. Contamination from ceiling dust may also occur in older buildings in which ceiling rosettes or cornices are in poor condition. In addition to petrol, lead and other toxic metals may also be introduced into ceiling cavities (and other areas) from previous industrial activities including incinerators and power stations.

Of even greater concern in older areas of inner Sydney is the contribution from leaded paint, with any dwelling built before 1970 having the potential to contain lead-bearing paints[3]. If these paints are in poor condition or the dwelling is being renovated, there is a chance to contaminate not only the immediate dwelling but also the surrounding neighbourhood[4]. Such contamination can result in elevated blood lead levels in children and even in adults.

Because many day-care centres are in older buildings and in areas of moderate traffic, we undertook a pilot investigation to determine the extent of lead contamination in several day-care centres, especially within the inner Sydney area.

METHODS

Six day-care centres – five from inner Sydney suburbs – agreed to participate in the study. These centres were in relatively old buildings with the possibility of lead paints (and lead from other sources) being present.

A variety of samples was taken at each of the centres depending on the nature of ambient lead sources (internal and external) and their accessibility, especially to children. The interior areas targeted for identification of lead dust contamination included windowsills, shelves and carpeted floors. Potentially contaminated surfaces were sampled using commercially available moistened wipes such as Diaparene Baby Wash Cloths. Dust from carpets and rugs was collected over a known surface area using a Volta vacuum cleaner dedicated to such sampling. Long-term dust (passive dustfall accumulation) was collected using petri dishes[5] placed in specific locations for two to four months.

Exterior samples were taken of surface soil in playground areas and from the sandpit or from areas potentially contaminated by lead paint such as under the eaves. Interior and exterior samples of paint flakes were taken from any painted surface which was in poor condition, especially in areas which were accessible to children.

Further details of sampling and analytical methods, and complete analyses, may be obtained from the first author.

RESULTS

Results for soil and dust are displayed in Figures 1, 2 and 3. The results of this study can be compared with "guidelines" provided by regulatory authorities. For example, the "action level" of the NSW Environment Protection Authority in soils is 300 µg/g Pb (ppm or mg/kg). No guidelines are available for dustfall accumulation (petri dish dust) but the US Department of Housing and Urban Development[6] requires that the "clean-up" levels for lead loading (amount of Pb per unit area) using dust from surface wipes on floors after lead abatement is 1,076 µg/m^2. Likewise, there are no guidelines for vacuum cleaner dust, although Bawden-Smith et al.[7] give values of 500–1,000 µg/g.

Day-Care Centre A was a new building (built in 1987) containing no obvious internal lead sources and was located away from any main roads. The levels found in the playground soil and the sandpit were considered equivalent to "background" levels (Figure 1).

Day-Care Centre B was also in a low traffic zone, and again the sandpit and soil lead levels were very low (Figure 1). The building itself was old, though the interior had recently been repainted and recarpeted so the vacuum dust, petri dish dust and surface wipe lead levels were also low.

Day-Care Centre C was in an older suburb of Sydney in a low traffic zone. The playground soil and sandpit lead levels were again low. In the older section of the centre, there were high ceilings with rosettes which may have allowed dust entry. As surfaces were regularly cleaned, the lead levels in the dust wipes were generally low. However, dust accumulation for longer periods was higher as measured in the vacuum cleaner dust, a music room surface wipe (taken along a high ledge), and the dustfall accumulation over a 30-day period from the petri dish dust.

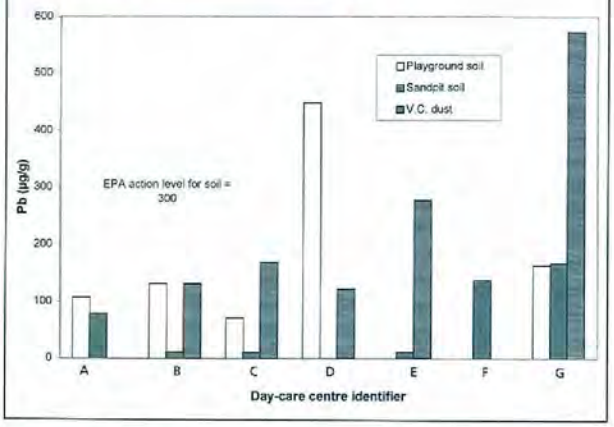
Day-Care Centre D proved to be the most contaminated day-care centre in this study. Paint under the eaves was in poor condition and contained about 45 per cent lead. Deterioration of this paint contaminated the soil beneath the eaves and probably the playground soil. The sandpit sand had recently been replaced, resulting in the negligible lead levels present.

Some interior paint surfaces were found to contain up to 16 per cent Pb. This paint, however, was in relatively good condition. Surface wipes from along windowsills in two rooms contained high lead values of 1,020 µg Pb/m^2 and 5,310 µg Pb/m^2. These windows were below the eaves with deteriorating lead paint and as the windows were opened during the day, lead-bearing dust may have been blown in.

Even though most rooms contained ventilators and one room had poorly fitted "tongue and groove" ceiling panelling, the amount of dust entering the rooms through these pathways was low as shown by the low amounts of lead in the three-month dustfall accumulation. The baby room was in excellent condition and, being a more recent addition to the building and away from the eaves with deteriorating paint, contained very low lead levels.

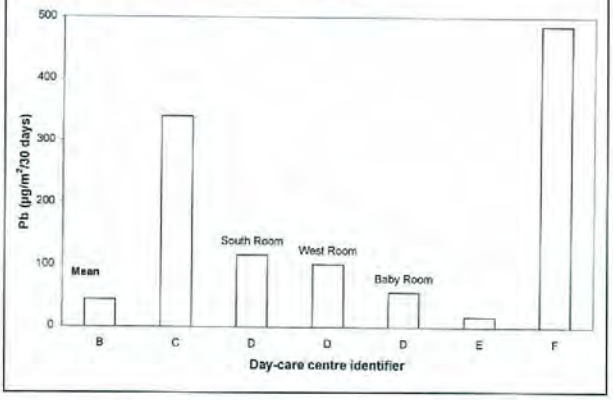
Day-Care Centre E was in an area of moderate traffic in an older suburb of Sydney. The day-care centre section of the building was relatively new and in good condition. The lead levels in all samples were low.

FIGURE 3



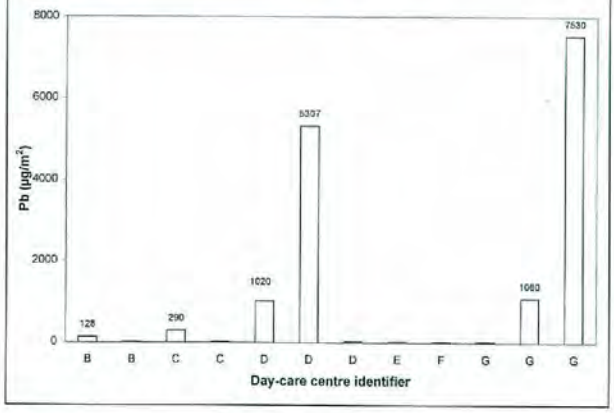
Lead concentrations in soil and dust from day-care centres in Sydney. A blank space indicates that no sample was available. The soil sample from Centre D containing 2,090 ppm Pb is not plotted. The action level for contaminated soils designated by the NSW EPA is shown. The vacuum cleaner dust results can be compared with a mean value of 2,255 w4,783 ppm Pb (median 1,000 ppm) found in 22 houses from inner Sydney[8].

FIGURE 4



Comparison of lead in dustfall accumulation (petri dust) representing airborne dust deposited over a designated time interval. The mean and standard deviation (90±60) for 164 samples from other Sydney houses is taken from Gulson et al.[9].

FIGURE 5



Lead concentrations in surface wipes from the day-care centres. Where no value is given, the result is <10 µg Pb/m². The level of clean-up after lead paint remediation recommended by the US Department of Housing and Urban Development is 1,076 µg/m².

Day-Care Centre F was in a high traffic area and this may be the explanation for the slightly elevated sandpit lead levels. The interior was in good condition and had no carpeted areas.

Day-Care Centre G was also in a high traffic area and this may be the explanation for the elevated lead levels in the playground soil and sandpit. Inside, there was a high, exposed, old timber ceiling. After high winds the staff noted that black dust could be seen on surfaces. With regular cleaning the lead levels are kept very low, as shown by the vacuum dust and surface wipe (cupboard) results. However, high lead levels can accumulate over time as evidenced from the wipes of surfaces not cleaned regularly and from the dustfall accumulation over an 80-day period in a petri dish (490 µg Pb/m² per 30 days).

DISCUSSION

Results from this pilot study were encouraging in that most measured levels were below any guideline level. Only one centre with deteriorating lead paint posed a hazard to the children, carers and the neighbourhood. Since the results of the study were provided to this centre, remedial actions have been implemented. However, there is a clear need for public health authorities, in conjunction with the Environment Protection Authority, to develop guidelines for interior sampling methods and clean-up levels.

The levels of lead in soil and dust measured in this pilot study of day-care centres varied considerably depending on factors which include location, and age and condition of the building. In general, centres housed in relatively new buildings have minimal problems unless they are near major traffic thoroughfares. When near such areas, exterior sites such as the sandpit and play areas may become contaminated over time. These problems have been minimised by regular replacement of the sand, covering of the sandpit when not in use, and installation of paving so daily hosing is possible.

In older buildings the potential hazards are paint and ceiling dust, typified by centres D and G respectively in this study. Remediation of leaded paint can be expensive, as can be ceiling dust from high, panelled ceilings commonly found in day-care centres housed in old church buildings.

On the positive side, regular cleaning by the carers of the day-care centres investigated in this pilot study has minimised the risk to the young children even in centres which are potentially a major hazard. Regular cleaning encompasses surface wiping with moist cloths and vacuuming of carpets and mats. Given the low levels of lead in all except one centre, it was not considered necessary to inconvenience the children, carers or parents with the second stage of this study, i.e. blood sampling. This does not mean the children have low blood lead levels, as they may live in a neighbourhood or dwelling where renovation and contamination from old leaded paint are ubiquitous.

This is the first published study that we know of specifically directed towards environmental sampling of day-care centres in Australia, so comparisons are not possible. With changes in Federal Government funding to day-care centres, extra attention to this area from public health authorities may be essential to ensure the health and safety of our infants.

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Lead in dust and soil from day-care centres

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ACKNOWLEDGMENTS

We thank: Macquarie University for financial support for this study under the Macquarie University Research Grants Scheme; Ms Ginie Udy of the Uniting Church Children's Services Forum for access to the day-care centres; the coordinators of the centres for their assistance; Professor Barry Batts for access to the ICP-AES; and Graeme Waller for the surface wipe digestion method.

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EDITORIAL NOTE

Lead is a ubiquitous contaminant of the urban environment. Children are at greatest risk of exposure and harm from absorption of environmental lead by virtue of their behaviour, their metabolism and the sensitivity of the developing nervous system to the toxic effects of lead. Lead in paint and lead additives in fuel are two sources of lead exposure in urban areas. This study confirms that lead is readily detected in the home and play environment of many children in NSW. A recent study showed that average blood lead levels in NSW preschool children were 5 to 7 µg/dL, with less than 25 per cent of inner-city children being above the goal for blood lead of 10 µg/dL[1]. This is an improvement since 1992[2]. Reductions in the proportion of cars using leaded fuel and in the the lead added to that fuel have reduced exposure. A study in Sydney showed that traffic flow near a child's home was a more important determinant of blood lead level than the traffic flow at the child's child-care centre[3]. These results do not detract from the need for efforts to reduce exposure to environmental lead, such as those now being coordinated by the Lead Reference Centre.

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Measuring the amount of lead in indoor dust

► Continued from page 93

Thus, the petri dish method can be used as a measure of recontamination, allowing individuals and health authorities to assess the effectiveness of abatement strategies.

Although no single method has yet been established as a standard for measuring indoor lead exposure, the long-term dust-fall accumulation method has advantages: it has low cost, does not require a power source, causes minimal inconvenience to the householder, does not involve noise, can be measured over a specific period, and cannot be biased by the householder cleaning the house before a sample visit[4].

The disadvantages of the method are the potential for disturbance or contamination and the time delay to obtain a measurement. In our study only one dish was lost and there were no overt signs of contamination.

Further evaluation of the technique to quantify the direct relationship between lead loading and blood lead levels is under way in North Lake Macquarie and Broken Hill. Our pilot study has shown that the method provides a valuable indicator of comparative trends in lead exposure over time and between areas.

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Update on lead-related activities

► Continued from page 99

In June 1997, $4.6 million was received from the NSW Health Department, the NSW Environment Protection Authority and Pasminco Metals–Sulphide for ongoing remediation.

NORTHERN SYDNEY AREA PUBLIC HEALTH UNIT

Local Government Environmental Health Officers were invited to a regional workshop on roles and responsibilities for lead hazard management conducted by the Lead Reference Centre and the Northern Sydney Area Public Health Unit on 9 December 1997 at Macquarie Hospital, North Ryde. Representatives from local authorities within the area, NSW Environment Protection Authority, WorkCover and Environmental Health Officers from the NSAHS Public Health Unit attended.

Documents distributed included *Local Government Lead Management and Guidelines for Environmental Health Officers in managing cases of Elevated Blood Lead Levels*. The aim of of the workshop was to provide information about lead hazards and the powers available to deal with lead problems. The outcomes of the day were an awareness of the responsibility of local government in the approval of building applications, procedures to be recommended for building renovations and case investigation protocol.

LEAD AND ENVIRONMENTAL HEALTH IN BROKEN HILL

Bill Balding
Project Manager, Broken Hill Environmental Lead Centre (now at the NSW Environment Protection Authority)
Sean Reddan
Senior Scientific Officer, Broken Hill Environmental Lead Centre

The mining city of Broken Hill is in the far west of NSW. Since the early 1880s silver, lead and zinc have been extracted from what was one of the world's richest ore bodies of its type. Broken Hill has had a major role in developing the nation's prosperity.

However, development at Broken Hill was not always easy. Broken Hill was isolated. Water, building supplies and fuel to feed the developing industry were difficult to obtain. The demand for resources led to widespread environmental degradation, resulting in erosion and periodic dust storms that would darken a clear sky. Boom-time mentality and the lack of planning and environmental laws decades ago have left an environmental legacy that is still being dealt with.

Mining also took its toll on the residents of Broken Hill. Lead poisoning among the early miners and their families was common. This presented as clinical plumbism with anaemia, nephropathy and encephalopathy, and sometimes resulted in death. The incidence of lead poisoning was estimated to be as high as 2 per 100 miners in 1895. The effects of lead on health were not limited to the workforce. A committee convened by the NSW Government in 1892 heard that the high infant mortality rate in Broken Hill might have been attributable to lead poisoning.

Although lead exposure among miners is no longer a major health issue, lead toxicity in children has emerged as a major public health issue over the past decade. In 1991 the first comprehensive testing undertaken on children under five years of age revealed that more than 80 per cent had blood lead levels over the current guideline level of 10 µg/dL.

Current environmental exposure to lead appears to be related more to historical mining and mine management practice than current activities. Lead contamination of the broader community arises from sources including:

- naturally occurring surface ores;
- past smelting and mine waste management practices;
- entrapment of dust in linings of domestic buildings and the re-entrainment over time of this dust back into the living spaces;
- open-cut mining activities (undertaken in the centre of the city until the early 1990s);
- the handling and transportation of ore concentrates;
- dust from tailings dams and the contamination of open spaces;
- off-site use of mining by-products for private use, such as landfill and driveways; and
- carry-home occupational health problems, such as contaminated dust in clothing and in vehicles.

However, environmental health investigations and programs managed jointly by the NSW Health Department and the NSW Environment Protection Authority in consultation with Broken Hill Council, industry and the community have been very successful in reducing the impact of environmental lead contamination in Broken Hill.

In 1994 the NSW Government established the Broken Hill Environmental Lead Centre. The centre, which has brought together a team of nurses, technicians, educators and scientists, is implementing a comprehensive and integrated strategy aimed at reducing lead exposure to children and the whole community. The centre has developed a strategy with five elements:

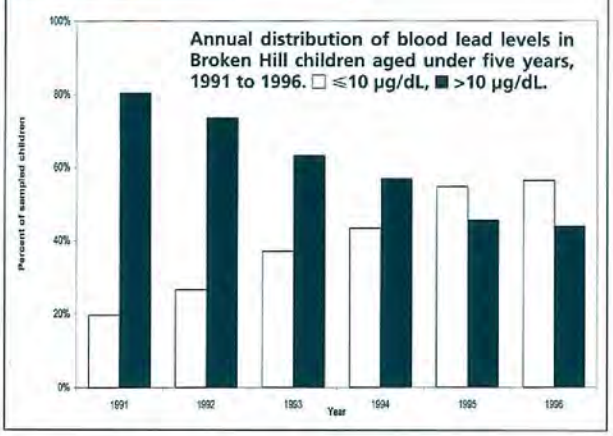
- education and awareness-raising;
- clinical monitoring;
- environmental testing of children's home environments (lead in paint, dust and soil);
- remediation of children's home environments (where appropriate); and
- the remediation of contaminated public land.

As part of this strategy, the centre has implemented a number of projects, including:

- monitoring of blood lead in newborn infants to help identify children at risk as early as possible;
- interactive preschool and school education programs;
- programs aimed at the Aboriginal community;
- early intervention programs, in which infants enter remediation programs before critical blood lead levels are attained;
- programs to support community groups to undertake lead-risk reduction "greening" programs (a greening program may include planting trees or lawn, removing contaminated soil from playgrounds, extracting contaminated dust from child-care centres, fencing off sensitive areas and controlling stormwater damage to rehabilitated areas); and
- monitoring and evaluation programs.

The centre's programs, with extensive site works undertaken by mining companies to control fugitive emissions, have been a major cause of the decline in average of the blood lead level in the population aged under five years in Broken Hill. The average blood lead level dropped from 18.4 µg/dL (0.88 µmol/L) in 1991 to 10.8 µg/dL (0.52 µmol/L) in 1996. At this time, the number of children exceeding the 10 µg/dL guideline for blood lead in Australians determined by the National Health and Medical Research Council was 44 per cent (Figure 6).

FIGURE 6



ACKNOWLEDGMENT

Staff of the Environmental Lead Centre, for their continued dedication in implementing this strategy.

LETTER TO THE EDITOR

BLOOD LEAD LEVELS OF CHILDREN, TRAFFIC, PAINT, AND SOCIAL DISADVANTAGE

In the Bulletin's previous issue focusing on lead (November 1995), Alperstein and Aldrich et al. discussed aspects of traffic related to environmental health and detailed the low average mean blood lead levels of children living near a motorway[1,2].

Coe et al. noted that "trade offs will be required between the level of risk and the economic resources that are available to manage risks"[3].

Problems may arise when community concern demands a public health response but the perception of risk does not have a scientific basis[1].

Blood lead levels are determined by intakes from sources such as lead in car exhausts and paint[4]. Bioavailability is important[5].

In 1986 unleaded petrol was marketed in Australia and subsequently the use of leaded petrol decreased. In 1995 the maximum allowable petrol lead became 0.2 g/L[6].

Epidemiological studies

Although experimental data have shown that exhausts may be a major source of lead, population data are less consistent. There have been national surveys in the United States, the United Kingdom and Australia[7-9]. Some medical findings have been difficult to interpret[10]. In recent years investigational and analytical techniques have improved. It may be possible to quantify any relationship between traffic density and the average blood lead levels of children.

In Culver City, California, there were no appreciable differences in the blood lead levels of those living or going to school at distances from 200 to 2,800 feet from a freeway used by 200,000 cars daily[11].

Woodstock, Cape Town, is predominantly a working-class area. The income of mixed-race persons is substantially lower than that of white persons, who have better homes. In a survey of blood lead levels, 13 per cent of mixed-race children, but no white children, had blood lead levels of 1.21 µmol/L (25 µg/dL) or more. The means, respectively, were 0.87 µmol/L and 0.58 µmol/L (18 µg/dL and 12 µg/dL). Those living near heavy traffic had levels 0.24 to 0.34 µmol/L (5 to 7 µg/dL) higher than those elsewhere. Daily traffic ranged from 240 to 20,000 cars. Lead in petrol was 0.836 g/L. Socially disadvantaged groups were at a higher risk of exposure and "indirect ingestion of lead-rich dust" contributed to blood lead levels[10].

A survey of primary-school children living near a motorway used by 15,000 cars daily near Newcastle, NSW, showed that lead burdens did not increase in the first year after the opening of the motorway in June 1993[2]. This may have been partly because of protective barriers. About half the vehicles were using unleaded petrol. In 1994, the mean blood lead level of 95 children was 0.30 µmol/L (6.16 µg/dL). Four per cent of the children had blood lead levels of 10 µg/dL or higher; 10 µg/dL or less is the goal for all Australians[12].

In a study of 718 pre-school children in the Central and Southern Sydney Areas, the geometric mean blood lead level was 0.34 µmol/L (7.0 µg/dL)[13]. The proportion with elevated levels did not "as a whole" exceed national guidelines. Nevertheless, Central Sydney required investigation of lead sources because, within a 10 km radius, 7 per cent of 316 children had levels of 15 µg/dL or higher.

The Australian National Survey report contains numerous statistical calculations of factors affecting blood lead levels: for example, age, education of parents and paint[9]. The mean blood lead level of 1,575 children was 0.28 µmol/L (5.72 µg/dL). The mean for those living within 25 m of a major road (more than 5,000 cars daily) was 0.29 µmol/L (6.0 µg/dL). The mean for persons in areas where there was no traffic count was 0.30 µmol/L (6.2 µg/dL). No statistically significant blood lead gradient associated with increase in traffic was found. However, there were gradients for factors such as the age of houses and pica. The blood lead levels of Aborigines and Torres Strait Islanders (0.36 µmol/L) exceeded those of other Australians. However, after adjustment for other variables, the difference was not significant. The national survey referred to factors such as traffic, low incomes, ill-health and paint. There was a strong association between paint and blood lead levels. A high mean value (0.4 µmol/L, 8.3 µg/dL) resulted from severely chalking paint. There were correlations between blood lead levels and cleanliness of homes and the frequency of pica.

Comments

Alperstein stressed the importance of surveying groups of pre-school children, including those at risk from paint or proximity to roads with heavy traffic (about 30,000 cars daily). Further debate is needed[1].

In 1993, the National Health and Medical Research Council revised its guidelines. For all children, the goal should be "below 10 µg/dL" (0.48 µmol/L)[12]. These guidelines should be used widely.

Reports have shown that the blood lead levels of non-white children usually exceed those of white children[9,10]. The reasons include sociodemographic and economic factors.

There are about 3.6 million pre-1971 houses that may have been painted with leaded paint[14]. Also, paint may recontaminate houses[15]. Therefore, such houses should be treated.

Donovan, in the Australian National Survey report, was not very critical about traffic and blood lead levels[9]. The effect of major roads was not statistically significant. There was no blood lead gradient associated with increasing traffic. Donovan stated that the mean level of 0.28 µmol/L (5.72 µg/dL) may have been contributed to by "children living in low income or older homes"[9]. Donovan[16] was aware of a 1996 Sydney study[13].

Importantly, Donovan stated that Australian children with high blood levels are "among the most disadvantaged. Action to alleviate this ... may in the long term be more beneficial than action directed specifically at exposure to lead"[9]. I agree.

Alan Bell
Formerly consultant to the Associated Octel Company Ltd, Melbourne

January 21, 1998

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Response from Dr Garth Alperstein, Central Sydney Area Health Service

Dr Bell makes two points:

- that lead-based paint as a source of lead exposure requires further attention
- that he believes, as does Dr Donovan, that alleviating social disadvantage may in the long term be more beneficial than action directed specifically at exposure to lead.

I do not believe anyone would disagree on either issue. Alleviating social disadvantage is a complex and difficult long-term issue and will positively affect more than just those problems associated with lead exposure. In the meantime, there are simple measures that can be implemented to reduce children's exposure to lead. How much effort, money and human resources are put into dealing with all health issues, including lead, are what governments and the community are constantly negotiating.

UPDATE ON LEAD-RELATED ACTIVITIES, FROM THE PUBLIC HEALTH UNITS

CENTRAL SYDNEY AREA PUBLIC HEALTH UNIT

Central Sydney Public Health Unit convenes quarterly meetings of the multidisciplinary Lead Advisory Committee. The committee has representation from the Central Sydney Public Health and Health Promotion Units, local Divisions of General Practice, the Lead Reference Centre, the Lead Advisory Service (NSW), the Department of Housing, and the paint industry, as well as the Central Sydney community paediatrician and a local council environmental health officer. The committee aims to reduce the effects on health of lead and the risks from lead within the Central Sydney Health Area by promoting and coordinating responses to lead within the Area, and liaising and collaborating with organisations outside the health sector. The committee also functions as a forum for information sharing about lead issues generally. In July 1997, a subcommittee was established to look at research issues. The Lead Research Sub-Committee aims to advise the Lead Advisory Committee and others about needs for research about lead, and to formulate research questions about lead issues.

A public health officer placed in the Central Sydney Public Health Unit undertook a literature review to examine evidence of the effectiveness of behavioural interventions aimed at health professionals, families and others to address lead issues.

The Central Sydney Area Public Health Unit developed a local protocol for the follow-up of notifications of people with elevated blood lead levels. This protocol was used to investigate cases of elevated blood lead levels in the Central Sydney Area before the NSW Health guideline for Environmental Health Officers became available. The protocol's public health unit operational summary, the letter to general practitioners about adult notifications, and the notification action checklist were used as examples in the NSW Health Department guideline.

HUNTER AREA PUBLIC HEALTH UNIT

In the North Lake Macquarie area, the Hunter Area Public Health Unit has been undertaking programs and research on lead issues:

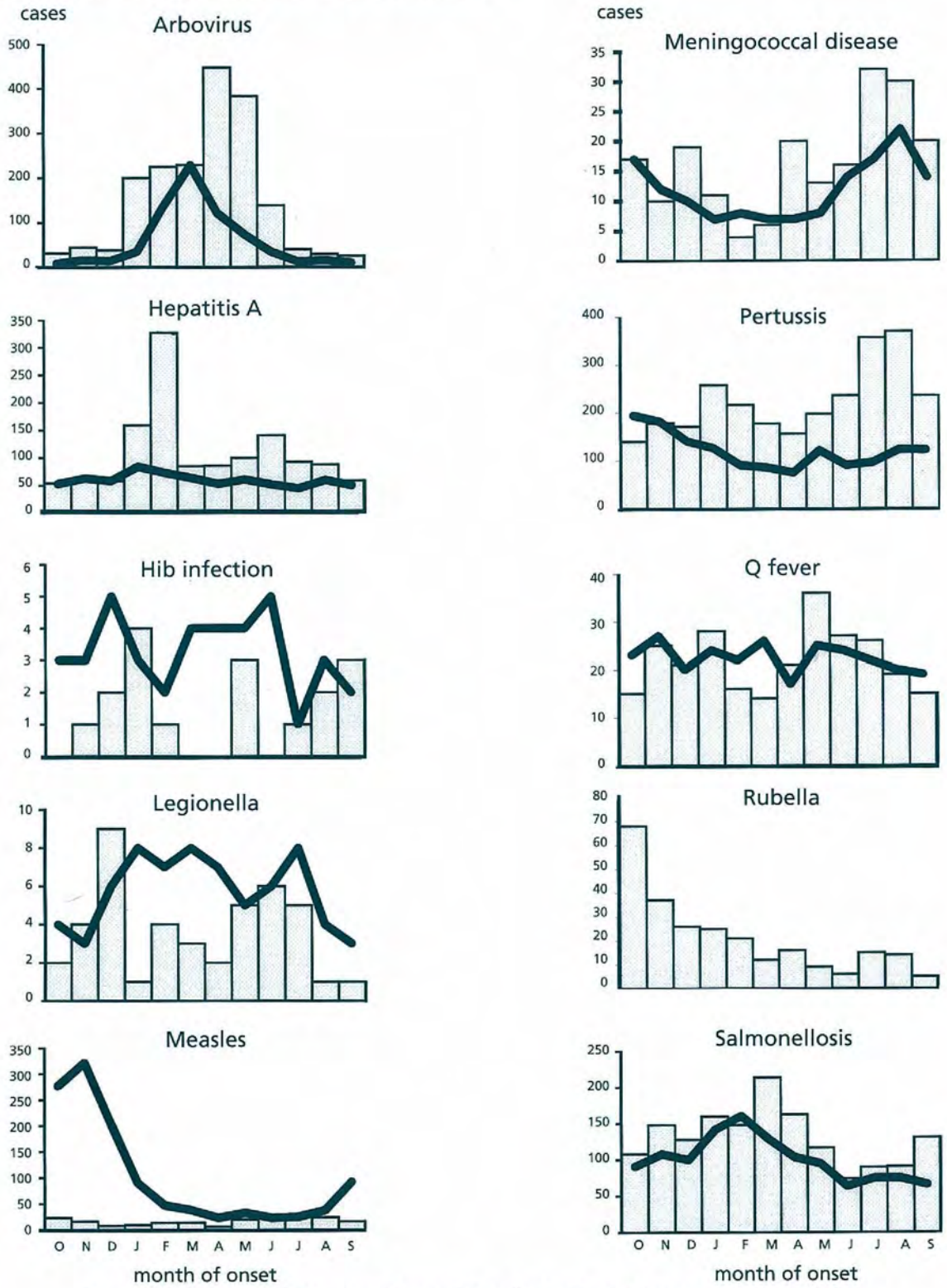
- analysis of blood lead levels of children in the suburbs of North Lake Macquarie, and relevant comparisons
- ongoing evaluation of children's blood lead levels after household remediation
- a knowledge, attitudes and practices survey of parents whose homes have been remediated.

The North Lake Macquarie Remediation Centre was established at the end of 1995 with funding of $300,000 from the NSW Government Environmental Trust, the NSW Health Department, Pasminco Metals–Sulphide and Lake Macquarie City Council. In 1996, the Centre started remediation of houses around the Pasminco Cockle Creek smelter, where children had high blood lead levels.

Continued on page 96 ▶

FIGURE 7

REPORTS OF SELECTED INFECTIOUS DISEASES, NSW, 12 MONTHS TO SEPTEMBER 1997, BY MONTH OF ONSET (WITH HISTORICAL COMPARISON)



Because of data collation problems, historical rubella figures are unavailable.

Oct - Sep 97 / Mean Oct 93 - Sep 96

INFECTIOUS DISEASES, NSW – NOVEMBER 1997

TRENDS

Reports of disease to September indicate that cases of **meningococcal disease** are falling after a peak in July and August (Figure B). To the end of October 165 cases were reported in the State, the same as for all of 1996 (Table B).

Most concerning has been the sustained epidemic of **pertussis** throughout the State. To the end of October 2,528 cases had been reported in NSW – more than double the number reported in all of 1996 (1,171) (Table A).

PERTUSSIS

Of the more than 2,500 pertussis cases reported so far in 1997, 56 per cent were females; 14 per cent were aged under 5 years, 24 per cent were aged 5-9 years and 62 per cent were aged 10 years or over. Most cases were reported by laboratories on the basis of a serological test result. Only one-quarter of cases aged under five years were reported to be up-to-date on their pertussis immunisations. Compared with cases reported in previous years, cases reported in 1997 tended to be a little older (42 per cent versus 37 per cent of cases reported between 1992 and 1996 were children aged 5-14 years). Only 10 per cent of cases were reported to have an identified exposure to an earlier case.

Because many people with the illness never consult a doctor and many diagnosed cases are not notified by doctors, the number of notified cases of pertussis is likely to underestimate substantially the true burden of the disease in the community. Assuming notified cases are representative of all people affected in the epidemic, analysis indicates that multiple strategies are needed to stop the pertussis epidemic.

Immunisation will prevent cases in preschool and many primary school children, but is less effective in preventing cases in, and transmission from, older cases. Therefore, early **case finding** to allow treatment and prophylaxis of close contacts is essential to reduce transmission of pertussis in the wider community.

On November 27 the NSW Health Department convened an expert panel (including paediatricians and public health and immunisation experts), to review the epidemiology of the outbreak, and to make recommendations for its control. The panel endorsed the Department's plan to provide more information to general practitioners about boosting immunisation rates, and early case detection, treatment and notification. The panel also recommended reviewing the immunisation strategy for NSW.

The key messages for health care providers in stopping this epidemic are:

- Review every child's immunisation status, regardless of why he or she presents for care.
- Opportunistically immunise children who are not up-to-date.
- Think of pertussis in any patient with a coughing illness (especially those with bouts of coughing followed by an inspiratory whoop or vomiting, or a cough that lasts more than two weeks).
- Notify suspected cases to your Public Health Unit.
- Confirm the diagnosis using either a nasal swab or paired IgA serology.
- Treat cases (within three weeks of onset) and their household contacts (within five weeks of case's onset, regardless of immunisation status) with erythromycin (40-50 mg/kg (maximum 1 g) per day in four doses for 10 days).
- Tell cases not to cough on others, especially small children who are at increased risk of severe disease, and to stay away from preschool, school or work until they have received five days of treatment.

Continued on page 102 ▶

PUBLIC HEALTH EDITORIAL STAFF

The editor of the *NSW Public Health Bulletin* is Dr Michael Frommer, Director, Centre for 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, described in the *New England Journal of Medicine* 1997; 336:309-315.

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) 9391 9232.

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Back issues can be obtained from the Better Health Centre, Locked Mail Bag 961, North Sydney 2059. Telephone: (02) 9954 1193, Facsimile (02) 9955 5196.

TABLE 2

INFECTIOUS DISEASE NOTIFICATIONS FOR NSW RECEIVED IN OCTOBER 1997 BY AREA HEALTH SERVICES

Condition	Area Health Service																	Period	
	CSA	NSA	WSA	WEN	SWS	CCA	HUN	ILL	SES	NRA	MNC	NEA	MAC	MWA	FWA	GMA	SA	Total for Oct**	Total to date**
Blood-borne and sexually transmitted																			
AIDS	4	2	2	–	–	–	1	2	15	–	–	–	1	–	–	1	–	28	219
HIV infection*	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	211
Hepatitis B – acute viral*	–	–	1	–	–	–	1	–	1	1	–	–	–	–	–	–	–	4	49
Hepatitis B – other*	60	47	33	2	1	1	4	8	45	2	1	1	2	1	1	2	2	213	3,149
Hepatitis C – acute viral*	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	10
Hepatitis C – other*	67	55	36	5	–	14	39	35	130	35	19	16	5	39	5	14	30	545	7,055
Hepatitis D – unspecified*	–	–	–	–	–	–	–	–	1	–	–	–	–	–	–	–	–	1	9
Hepatitis E	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	8
Hepatitis, acute viral (NOS)	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1
Gonorrhoea*	8	4	1	–	1	–	1	–	15	1	3	1	–	–	–	–	–	35	534
Syphilis	6	–	1	–	–	1	–	–	11	–	2	1	2	2	1	–	–	27	455
Vector-borne																			
Arboviral infection*	2	3	–	–	–	–	3	1	–	2	7	1	3	–	–	1	1	24	1,783
Malaria*	1	1	–	1	–	1	–	–	3	–	1	–	–	1	–	1	–	10	141
Zoonoses																			
Brucellosis*	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	3
Leptospirosis*	–	–	–	–	–	–	2	–	–	1	1	–	–	–	–	1	–	5	27
Q fever*	–	–	–	–	–	–	–	–	1	1	3	4	2	–	–	7	–	18	225
Respiratory/other																			
Legionnaires' disease	–	–	–	–	–	–	–	–	1	–	–	–	–	–	–	–	–	1	36
Meningococcal (invasive) infection	–	2	2	–	–	1	–	4	1	1	–	–	–	2	–	–	1	14	165
Leprosy	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1
Mycobacterial tuberculosis	2	6	3	–	–	–	–	2	1	–	–	–	–	–	–	2	–	16	317
Mycobacteria other than TB	3	12	1	–	–	1	2	–	3	–	1	–	–	–	–	1	3	28	313
Vaccine-preventable																			
Adverse event after immunisation	1	–	2	1	–	–	–	–	1	–	1	3	–	–	–	3	–	12	59
H.influenzae B (invasive) infection	–	–	–	–	–	–	1	–	1	–	–	–	–	–	–	–	–	2	15
Measles	–	1	4	2	–	–	2	1	–	–	–	11	5	2	–	–	2	30	183
Mumps*	1	–	–	–	–	–	–	–	–	1	–	–	–	–	–	–	–	2	25
Pertussis	41	43	56	14	–	9	57	18	34	35	10	12	–	5	–	4	10	348	2,528
Rubella*	–	–	1	2	–	–	–	–	–	–	–	–	–	–	–	–	1	4	132
Tetanus	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	3
Faecal-oral																			
Cholera*	–	–	–	–	–	–	–	–	–	–	2	–	–	–	–	–	–	2	4
Foodborne illness (NOS)	–	–	–	–	–	1	–	–	1	–	–	–	–	–	–	–	2	4	108
Gastroenteritis (instit)	26	–	1	–	–	–	–	–	–	–	–	–	–	–	–	–	–	27	691
Hepatitis A	8	3	6	6	–	4	17	2	17	10	4	4	3	–	2	–	1	88	1,243
Listeriosis*	–	–	–	–	–	–	1	–	–	–	–	–	–	–	–	–	–	1	19
Salmonellosis (NOS)*	8	21	16	20	–	4	13	5	20	7	9	6	1	10	4	5	2	151	1,360
Typhoid and paratyphoid*	–	–	1	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1	19

* lab-confirmed cases only
** includes cases with unknown postcode

INFECTIOUS DISEASES, NSW – DECEMBER 1997

TRENDS

The outbreak of **pertussis** in NSW has reached **epidemic** proportions, with 666 cases being reported in November alone. The Chief Health Officer has written to all doctors in the State alerting them to the epidemic, and urging them to:

- consider a diagnosis of pertussis in people with coughing illnesses,
- treat cases of pertussis and their household contacts with erythromycin,
- notify their Public Health Units of suspected cases; and
- vaccinate all children against pertussis, following NHMRC recommendations.

Health care providers should routinely check the vaccination status of all their patients and opportunistically update children's immunisations if they are due. Routine childhood immunisations are free in NSW. In addition, the NSW Health Department has provided funding for acellular pertussis vaccine, available as DTPa (a vaccine containing acellular pertussis vaccine with tetanus and diphtheria vaccines). Immunisation providers can now obtain DTPa **free** of charge for **children due for vaccination at 18 months and 4-5 years of age**, as well as for **children who have had previous moderate reactions to triple-antigen vaccination**. PHUs will provide further advice on the use of this and other vaccines and the control of pertussis to health care providers and parents.

After an earlier rise in the number of cases, notifications of **meningococcal disease** further declined through October (Figure A) and November (Table A), with a total of 204 notifications for the year to the end of November.

Laboratory notifications of **salmonellosis** rose above historical expectations in October, in part because of an increase in *Salmonella typhimurium* phage type 64 (40 cases identified for October). While preliminary investigations have revealed no common source of this infection, further investigations are under way.

OUTBREAK OF GASTROENTERITIS LINKED TO EATING PIPIS

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On December 16, 1997 the Food and Nutrition Unit received reports from South Western Sydney, Central Sydney and South Eastern Sydney Public Health Units of five separate groups of people who had been ill after consuming pipis. On December 16 the Food and Nutrition Unit sent an e-mail to all PHUs seeking further reports of gastroenteritis related to pipi consumption. Over the next 13 days details of 56 people with pipi-related illness were received from Central Sydney, South Western Sydney, South Eastern Sydney, Northern Sydney and Northern Rivers. In addition, anecdotal reports of a further 46 people, for whom individual details were not available, were reported.

Pipis

Pipis (*Plebidonax deltoides*) are bivalve molluscs that live in the intertidal zone of sand on gently sloping ocean beaches, a few centimetres below the surface. In NSW pipis are collected in commercial quantities from the beaches around Ballina, South West Rocks, Forster–Tuncurry and Stockton. Most commercial supplies in NSW come from Ballina, which has an annual collection of 300,000 kilograms. After harvesting, the sand is removed from pipi shells by placing them in salt water for several hours.

Epidemiological investigation

For this investigation, a case was defined as a person reporting gastrointestinal symptoms of either vomiting or diarrhoea after ingestion of pipis from December 5, 1997. Between December 12 and 16, 56 cases were reported, including 29 females and 27 males. The mean age of cases was 33.9 years (range 9-71 years). Age was unknown for 8 cases. Cases were reported from PHUs in South Western Sydney (18), Northern Sydney (13), Central Sydney (11), Northern Rivers (7) and South Eastern Sydney (7).

Reported symptoms included diarrhoea (56 cases, 100 per cent), abdominal cramping (50 cases, 89 per cent), nausea (27 cases, 48 per cent), vomiting (14 cases, 25 per cent), headache (12 cases, 21 per cent) and fever (8 cases, 14 per cent).

Based on the reported time between pipi consumption and illness (reported for 40 cases), the mean incubation period was 16.4 hours (range 2-49 hours). The mean duration of symptoms (reported for 28 cases) was 49 hours (range 12-84 hours).

Stool samples from three cases were negative for bacteria and (as preliminary results indicate) negative for viruses.

Nine of the cases were among a party of 27 who ate a meal at a restaurant in south-western Sydney. Of the 15 people who completed questionnaires administered by PHU staff, 13 reported eating pipis. Nine of the 13 people (69 per cent) who ate pipis at the dinner were ill, compared with no illness in the two people who did not eat pipis at the dinner (P=0.14).

Environmental investigation

The implicated pipis were traced to a supplier on the NSW north coast. Samples of pipis were tested by the Division of Analytical Laboratories. Standard bacterial plate counts for raw pipis were under 10^5 for all samples, the limit set by Standard D of the Australian Food Standards Code. The level of *E. coli* was above the limit set in Standard D in some raw and cooked samples. Some raw samples, but not all, had levels of *Vibrio parahaemolyticus* under 10^4. Levels of *V. parahaemolyticus* over 10^5 are associated with illness[1]. *Salmonella typhimurium* and *S. sophia* were present in two raw samples and *S. sophia* was present in one raw sample. No enteroviruses or Norwalk viruses were found in any of the pipi samples.

Two frozen samples of implicated pipis collected from cases were tested at the Institute of Environmental Science and Research in Wellington, New Zealand, for the presence of marine biotoxins, specifically diarrhoetic shellfish poisons. Diarrhoetic shellfish poisoning is caused by a group of polyether toxins produced by microalgae of the *Dinophysis* species[2]. Bivalve shellfish, such as pipis, accumulate the toxin through filter-feeding on microalgae. Results obtained on December 31, 1997 showed that initial ELISA tests for the presence of two specific diarrhoetic shellfish poisons were negative, but a mouse bioassay was positive,

indicating the presence of a toxin. These results do not rule out diarrhoetic shellfish poisoning as the cause of the illness. Further testing will be undertaken to identify the specific toxin present.

Intervention

On December 17, 1997 the NSW Department of Health issued a media release warning the public to cook pipis well before eating them. When subsequent cases reported incubation periods as short as 2 hours and consuming pipis that had been cooked for up to 30 minutes, concern that a heat-stable toxin may be responsible for the illness led the Department to issue a second warning, on December 19, that pipis from Northern NSW should not be eaten. The Sydney Fish Markets voluntarily suspended sale of pipis on December 17 and the Ballina Fish Co-operative voluntarily recalled all pipis from sale on December 18. On December 19 the Fisheries Department closed the beaches between the Richmond and Evans rivers on the far north coast to harvesting of bivalve molluscs at least until January 23, 1998.

Pipis implicated in all reported cases are being traced to their source to determine whether the handling processes are implicated as a cause of the contamination. The presence of salmonella in some pipi samples is being investigated. Further testing of pipis is required to determine the specific toxin present and whether the toxin is the cause of the presenting illness.

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1. Benenson AS (editor). *Control of communicable diseases manual*. 16th ed. Washington: American Public Health Association, 1995.
2. Hallegraeff GM. A review of harmful algal blooms and their apparent global increase. *Phycologia* 1993; 32(2):79-99.

AVIAN INFLUENZA A (H5N1) IN HUMANS, HONG KONG, 1997

A strain of influenza virus that previously was known to infect only birds has been associated with infection and illness in humans in Hong Kong. The first known human case of influenza type A (H5N1) occurred in a 3-year-old child who died from respiratory failure in May 1997. In Hong Kong the virus initially was identified as influenza type A but the subtype could not be determined using standard reagents. By August the virus was identified as influenza A (H5N1). The possibility of laboratory contamination was excluded. Since this initial case was identified six additional Hong Kong cases have been confirmed and two possible cases were identified to mid-December.

Confirmed cases

Patient 1: On May 9, 1997 a previously healthy 3-year-old boy developed fever, sore throat and cough. His illness progressed and on May 18 he was admitted to the paediatric intensive care unit. On May 21 the child died from acute respiratory distress secondary to viral pneumonia. Influenza A (H5N1) virus was isolated from a tracheal aspirate collected on May 19. The child may have been exposed to ill chickens before he became ill.

Patient 2: On November 6 a 2-year-old boy with a congenital heart disease developed high fever, cough and sore throat and was admitted to hospital the next day for presumed pneumonia. He had an uneventful recovery and was discharged from the hospital on November 9. A nasopharyngeal swab yielded influenza A (H5N1) virus.

Patient 3: On November 20 a previously healthy 13-year-old girl developed fever, sore throat and cough. She was admitted to hospital on November 26 because of pneumonia. On November 27 she was transferred to the ICU and placed on mechanical ventilation. As of December 17 she remained in hospital. Influenza A (H5N1) virus was isolated from a tracheal aspirate.

Patient 4: On November 24 a previously healthy 54-year-old man developed fever and cough and on November 29 he was admitted to hospital because of pneumonia. His condition deteriorated and he died on December 5. A bronchoalveolar lavage specimen yielded influenza A (H5N1) virus.

Patient 5: On December 4 a 24-year-old woman developed fever, sore throat, cough and dizziness. Her symptoms worsened and she was admitted to hospital on December 7. Her condition deteriorated and on December 9 she was transferred to the intensive care unit and placed on mechanical ventilation. Influenza A (H5N1) was isolated from a tracheal aspirate.

Patient 6: On December 7 a 5-year-old girl developed fever, rhinitis, cough, sore throat and vomiting. As of December 17 she remained in hospital in satisfactory and stable condition. A nasopharyngeal aspirate yielded influenza A (H5N1).

Patient 7: On December 12 a 2-year-old boy developed fever and was admitted to hospital in good condition. The child is a cousin of patient 6, who frequently visited him and his family at their home. A culture from the child was positive for influenza A (H5N1) virus.

Possible cases

On November 24 a previously healthy 37-year-old man was hospitalised because of pneumonia; the onset of the illness was on November 17. He recovered and was discharged. Preliminary results of serologic tests suggest infection with influenza A (H5N1); results of a neutralisation assay (which is required to confirm infection) are pending. The other possible case is the 3-year-old sister of patient 7 and cousin of patient 6. She lived in the same apartment as patient 7 and had the onset of fever on December 13 and was admitted to hospital in good condition. Preliminary laboratory results were positive for influenza A (H5N1) virus; confirmation of these results by virus isolation is pending.

Ongoing investigation

The primary objectives of the ongoing investigation are to detect and investigate new cases and to identify potential sources, including whether and to what extent infection is being transmitted from person to person, birds to humans, or both. Blood specimens for measurement of antibody against influenza A (H5N1) and information concerning respiratory illness, exposure to birds, the type and degree of exposure to cases, and other relevant information are being collected from people who had contact with cases and from control groups that did not have contact with cases.

Patients 1 to 6 lived in different parts of Hong Kong, had no contact with each other and had no apparent common exposures. Patients 6 and 7 and the 3-year-old girl who is a possible case have all had contact with each other and common exposures. Influenza A (H5N1) viruses isolated from these patients are being fully characterised both

antigenically and genetically by the United States Centers for Disease Control and Prevention.

Surveillance for influenza has been intensified in Hong Kong and Guangdong Province, China, following the identification of the first human case, mostly through outpatient facilities and hospitals. Surveillance among poultry in Hong Kong indicates continued circulation of A (H5N1) viruses since March, when outbreaks on poultry farms were first detected.

Comment

The cases described in this report represent the first documented human infections with avian influenza A (H5N1) virus. One of the most important aspects of the investigation is to determine the source of infection and mode of transmission. This effort is complicated by the high prevalence of exposure to live poultry among residents of Hong Kong.

Although the spectrum of illness caused by human influenza virus infection can range from asymptomatic to fatal, most human influenza infections cause acute febrile respiratory illnesses that resolve without complications. Many of the cases of human infection with type A (H5N1) identified in Hong Kong have been unusually severe. However, because influenza surveillance in Hong Kong has been conducted primarily in hospitals, milder cases may not have been recognised, and the severity of infections identified to date may not be representative of the spectrum of illness caused by A (H5N1) infection in humans.

Infection with this influenza strain, which is new to humans, prompts consideration about whether this virus has the potential to spread globally and cause a pandemic. For an influenza pandemic to occur, a novel human influenza strain against which all or most of the human population has no antibody must be capable of sustained person-to-person transmission, causing widespread illness[1]. As of December 17 acute respiratory illness among the population of Hong Kong apparently had not increased.

Although the potential for widespread transmission of this strain is unknown, as a precautionary measure laboratory studies have been initiated to identify a candidate A (H5N1) vaccine strain. There are no plans for commercial vaccine production. Two antiviral drugs, amantadine and rimantadine, inhibit replication of virtually all naturally occurring human and animal strains of influenza type A and therefore can be useful for prophylaxis and treatment of influenza A infections[2-4]. Influenza A viruses resistant to amantadine and rimantadine can emerge during treatment, but drug-resistant influenza viruses have only rarely been isolated from specimens collected as part of routine influenza surveillance[5,6]. Influenza A (H5N1) isolates from Hong Kong that have been tested are sensitive to amantadine and rimantadine.

People considering travel to Hong Kong should consider that:

- the number of clinical cases of influenza A (H5N1) identified to date is small, despite the intensive surveillance that has been conducted among the 6.5 million residents of Hong Kong; and
- there has been no detected increase in the incidence of acute respiratory illness among residents of Hong Kong. However, the risk for infection to people living in, or visiting, Hong Kong cannot be determined with certainty, and the risk may change over time. Although no human influenza A (H5N1) infections have been identified outside Hong Kong, worldwide surveillance for influenza is critical to monitoring the circulation of various influenza strains. Human influenza types A (H3N2), A (H1N1) and B continue to circulate worldwide[7,8].

Avian influenza (H7N4), Tamworth

The avian influenza virus in Hong Kong is an entirely different strain from the one that affected chicken and other bird flocks in the Tamworth area in November and December 1997. An increased mortality rate (up to 96 per cent) was noted on a Tamworth chicken farm in mid-November. Viral studies indicated the cause of that outbreak was avian influenza A (H7N4). This virus has not been shown to affect people or mammals or the quality of poultry meat or eggs, but is important to the agriculture industry because of its potential economic effect. NSW Agriculture reports that this is the fifth outbreak of avian influenza in Australia in the past 20 years. This and all previous outbreaks were successfully eradicated. The cause of such outbreaks is hypothesised to be contamination of drinking water by wild birds (especially water fowl).

Implications of Hong Kong avian influenza for NSW

This report has raised concern about the possible emergence of a new strain of influenza. New strains of influenza were responsible for devastating epidemics in 1918-19 (more than 20 million deaths), 1957 (70,000 deaths in the United States alone), and 1968-69 (33,000 US deaths). However, it is quite possible that such avian strains may occasionally infect humans (who act as a dead-end host), but be identified only because of the intense surveillance for emerging influenza strains that has been going on in Hong Kong and southern China as part of a World Health Organisation influenza pandemic early-warning system.

The NSW Health Department is working closely with the Commonwealth and other State and Territory Health Departments to develop enhanced influenza surveillance and control measures. A group of local public health, laboratory, primary care and infectious disease experts will be convened early in 1998 to provide advice as events unfold.

Adapted from: CDC. Isolation of Avian Influenza A (H5N1) Viruses from humans – Hong Kong, May-December 1997. *MMWR* 1997; 46:1204-7.

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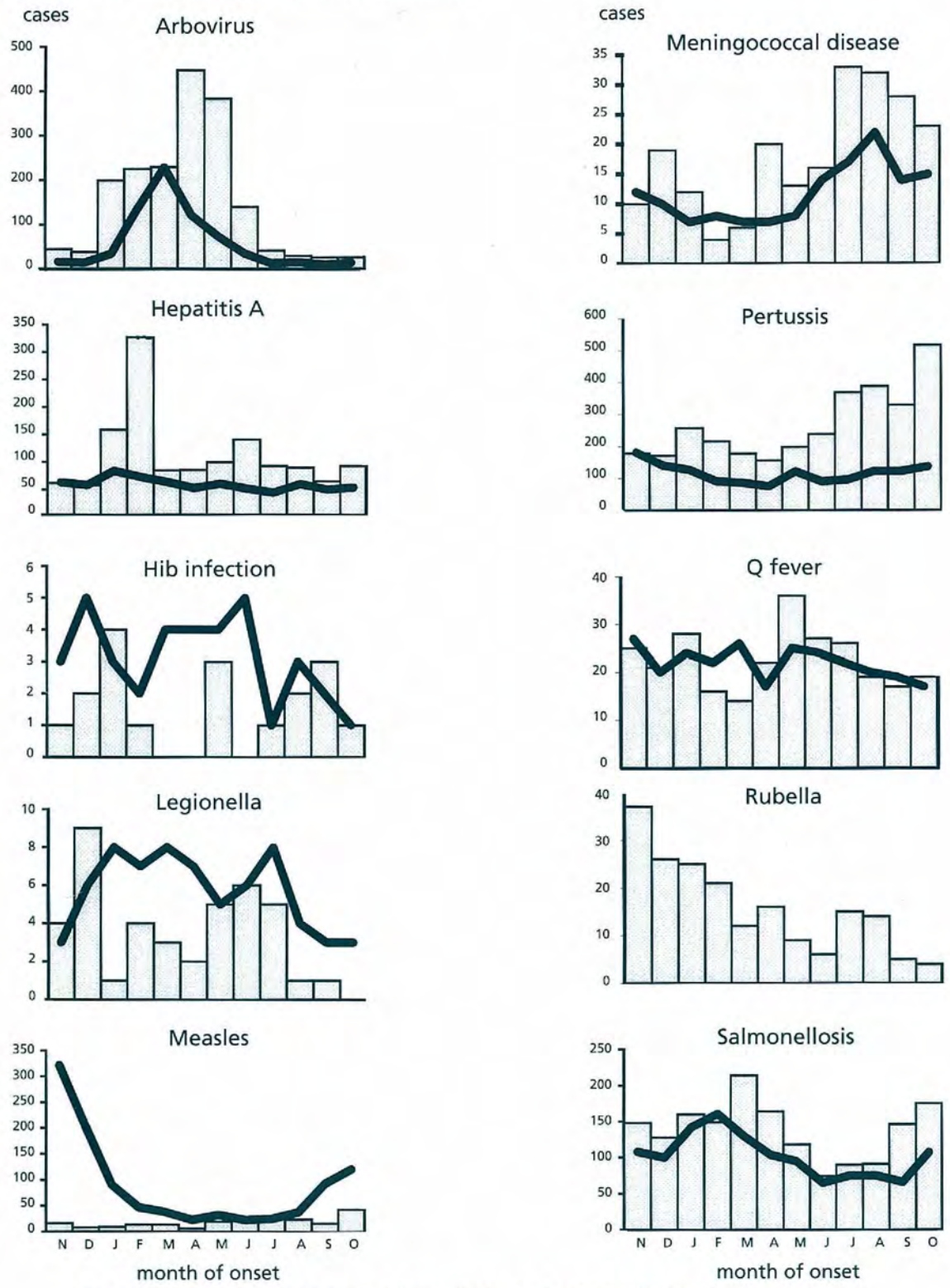
FIGURE 8

REPORTS OF SELECTED INFECTIOUS DISEASES, NSW, 12 MONTHS TO OCTOBER 1997, BY MONTH OF ONSET (WITH HISTORICAL COMPARISON)



Because of data collation problems, historical rubella figures are unavailable.

Nov 96 - Oct 97 / Mean Nov 93 - Oct 96

TABLE 3

INFECTIOUS DISEASE NOTIFICATIONS FOR NSW RECEIVED IN NOVEMBER 1997 BY AREA HEALTH SERVICES

Condition	Area Health Service																	Period	
	CSA	NSA	WSA	WEN	SWS	CCA	HUN	ILL	SES	NRA	MNC	NEA	MAC	MWA	FWA	GMA	SA	Total for Nov**	Total to date**
Blood-borne and sexually transmitted																			
AIDS	10	1	–	–	2	–	1	–	25	–	–	1	–	–	–	–	–	40	259
HIV infection*	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	211
Hepatitis B – acute viral*	–	–	1	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1	50
Hepatitis B – other*	54	43	6	1	26	7	3	6	32	1	1	1	2	6	4	3	1	197	3,588
Hepatitis C – acute viral*	–	–	–	–	–	–	–	–	–	–	–	–	1	–	1	–	–	2	12
Hepatitis C – other*	58	55	55	5	34	30	38	41	80	18	12	13	8	31	4	19	27	528	7,865
Hepatitis D – unspecified*	–	–	1	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1	10
Hepatitis E	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	8
Hepatitis, acute viral (NOS)	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1
Gonorrhoea*	8	3	–	–	1	–	3	1	20	–	–	1	–	–	–	–	1	38	582
Syphilis	5	–	1	–	3	–	–	–	13	–	1	3	–	–	5	–	–	31	512
Vector-borne																			
Arboviral infection*	–	3	–	1	–	2	–	–	1	4	3	–	–	2	3	2	1	23	1,805
Malaria*	1	2	–	–	–	1	–	–	–	–	–	–	–	–	–	1	1	6	154
Zoonoses																			
Brucellosis*	1	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1	4
Leptospirosis*	–	1	1	–	–	–	–	–	–	–	–	–	–	–	–	–	–	2	29
Q fever*	–	–	1	–	–	–	2	–	–	–	1	1	2	4	3	6	–	20	246
Respiratory/other																			
Legionnaires' disease	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	36
Meningococcal (invasive) infection	–	1	2	3	2	3	1	–	1	1	1	1	1	–	–	1	1	19	204
Leprosy	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1
Mycobacterial tuberculosis	3	2	3	–	8	–	3	1	3	–	1	–	–	–	–	–	–	24	367
Mycobacteria other than TB	10	9	–	–	–	2	4	–	4	–	1	–	1	–	–	1	1	34	364
Vaccine-preventable																			
Adverse event after immunisation	–	–	–	–	–	–	–	–	1	–	–	8	–	–	–	4	1	14	74
H.influenzae B (invasive) infection	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1	1	17
Measles	2	5	6	3	1	–	14	1	1	1	2	2	3	–	–	1	3	45	230
Mumps*	–	–	1	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1	26
Pertussis	56	77	95	49	64	8	96	33	86	40	12	23	–	2	–	6	19	666	3,282
Rubella*	–	–	–	2	–	–	–	–	–	–	–	–	1	–	–	–	–	3	135
Tetanus	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	3
Faecal-oral																			
Cholera*	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	4
Foodborne illness (NOS)	4	4	–	–	–	–	–	–	2	–	–	–	–	–	1	–	6	17	128
Gastroenteritis (instit)	16	–	5	–	12	–	103	–	1	–	–	–	–	–	1	–	–	138	867
Hepatitis A	10	4	8	1	7	3	7	2	5	6	4	4	2	–	1	–	2	65	1,329
Listeriosis*	–	1	1	–	–	–	–	–	–	–	–	–	–	–	–	–	–	2	21
Salmonellosis (NOS)*	14	19	10	13	6	6	6	4	9	7	5	7	5	10	1	7	2	131	1,528
Typhoid and paratyphoid*	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	19

* lab-confirmed cases only
** includes cases with unknown postcode

Abbreviations used in this Bulletin:
CSA Central Sydney Health Area, SES South 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, NRA Northern Rivers Health Area, MNC Mid North Coast Health Area, NEA New England Health Area, MAC Macquarie Health Area, MWA Mid West Health Area, FWA Far West Health Area, GMA Greater Murray Health Area, SA Southern Health Area, 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.