

Lead

by

Rebekah Jenkin

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New South Wales Parliamentary Library cataloguing-in-publication data

Jenkin, Rebekah

Lead / by Rebekah Jenkin

[Sydney, N.S.W.]: New South Wales Parliamentary Library, 1993. -

[viii, 22 p.; 30 cm.] (Current issues: background paper, ISSN 1320-4521 /

New South Wales Parliamentary Library; 1993/4)

At head of title: New South Wales Parliamentary Library

ISSN 1320-4521 ISBN 0724095608

- Lead--Health aspects (LCSH)
- Lead--Environmental aspects (LCSH)
- [1. LEAD-HEALTH-ASPECTS] (Parliamentary thesaurus)
- [2. LEAD-ENVIRONMENTAL-ASPECTS] (Parliamentary thesaurus)
- I. New South Wales. Parliamentary Library
- II. Title
- III. (Series) Current issues : background paper (New South Wales. Parliamentary Library) ; 1993/4

363.7384 (DDC20)

Published by the New South Wales Parliamentary Library.

Foreword

Topics for Background Papers are selected by the Parliamentary Library, or suggested by Members, but the views expressed remain those of the authors.

Suggestions for future topics are welcome.

Rob Brian Parliamentary Librarian November 1993

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Acknowledgments

I would like to thank my colleagues in the New South Wales Parliamentary Library, in particular Gareth Griffith, Sharon Rose, David Clune and Greig Tillotson, who all made helpful suggestions regarding both content and style. Philip Dixon was responsible for the presentation and production of the monograph.

I would also like to thank Elizabeth O'Brien of the Lead Group for supplying information and resources that would have otherwise been impossible to obtain.

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Appendix 1 Who Can Use Unleaded? Models of cars that can use unleaded petrol. From CHOICE October 1993.

Abbreviations

Australian Bureau of Agricultural and Resource Economics Australian and New Zealand Environment **ABARE**

ANZECC

and Conservation Council

EPA Environment Protection Authority

National Health and Medical Research Council NH&MRC

1 Introduction

Lead is an extremely versatile heavy metal which is used in a diverse range of products and applications. Use of lead dates back to ancient times when the Egyptians used lead in their plumbing. Later, many other peoples, including the Greeks and Romans, took advantage of the softness, low melting point and ease with which lead mixes with other materials to forge a variety of long lasting domestic appliances. Such was the benefit endowed by lead upon the lives of ancient humans.

Modern society has also benefited from the extensive use of lead but, armed with knowledge wrought from experience and learning, lead is no longer regarded as an innocuous useful metal. Instead twentieth century society is attempting to come to grips with the considerable and damaging repercussions of widespread and continuous lead exposure.

2 Background - Lead in Australia

Historically, like most modern societies, Australia has made extensive use of the adaptability and usefulness of lead in the preparation of a huge variety of products and processes. The paint, petrol, and car manufacturing industries are just a few examples of areas where lead has found an application. The overall use of lead in Australia has declined since 1985 due to the introduction of unleaded petrol and this decline is expected to continue.

However, domestic consumption of lead has increased slightly over the last decade from 68 700 tonnes/annum in 1980 to 74 310 tonnes/annum in 1990 (ABARE, 1992). Lead is used in batteries, cable sheathing, rolled and extruded metal products, for example in plumbing fittings, as an alloy and in solder, in ammunition and shot and as a petrol additive.

Australia is the largest mine producer of lead in the world and is a major exporter of lead concentrate, bullion and ore as well as refined lead. In 1990/91, Australian mines produced 579 000 tonnes of lead ore and 239 000 tonnes of refined lead. Only 56.5 tonnes of refined lead was used locally, the bulk of both refined lead and lead ore was exported (ABARE, 1992).

World consumption of lead is expected to rise approximately 3% per year over the next few years with Australian predicted earnings also expected to rise, perhaps by as much as 7% (to roughly \$421 million per annum in 1993). The mining industry employs approximately 9000 workers across all states except Victoria and indirectly contributes to the employment of thousands more. Thus lead is a major source of export earnings and

employment in Australia and this is anticipated to continue well into the next millennium.

Background levels of lead, that is, lead that occurs naturally in the environment, are usually extremely low. However, the versatility of lead and its wide usage within industry result in almost continuous exposure of most humans to lead. Lead is a persistent substance which is found in the air, water and soil. Lead endures in the atmosphere for extended periods. Thus, historical sources of lead may continue to be sources of exposure.

3 Sources of Lead

- (a) Air
- (b) Soil
- (c) Water
- (d) Other major sources of lead

Lead enters the ecosystem from a variety of sources. The major ones are atmospheric lead from motor vehicle emissions, lead from paint chips and used ammunition, lead from fertilizers and pesticides and lead from lead acid batteries and other industrial products.

(a) Air

Despite the introduction of unleaded petrol in 1985, 90% of the overall lead in air in urban Australia owes its origins to motor vehicle emissions. Approximately 550 tonnes of lead a year is emitted in Sydney. Studies (New Scientist, 1993) also show that although the most serious occurrences of lead contamination are found in large urban centres in the vicinity of major roads, lead pollution from petrol has affected even remote areas such as Northern Greenland and Cape Grim in Tasmania where lead levels have been shown to increase and decrease in direct correspondence with the increases and decreases in lead production in the industrialized world. Thus, even though rural areas are less likely to suffer the high level of air lead contamination from petrol, the contribution of lead in petrol to air lead levels is significant across all environments.

The distribution of lead in the air throughout the environment is largely dependent upon the size of the lead particles. In petrol containing lead in a concentration of 400mg lead/litre of petrol, about 40mg¹ of lead (10%) is retained by lubricating oil, 60mg (15%) is retained on the interior surfaces of the vehicle engine and exhaust system and 300mg (75%) is emitted into the air. Of the emitted lead (300mg), approximately 140mg (47%) is emitted as lead aerosol and the remainder (160mg or 53%) is emitted as large particles. Lead aerosol potentially may be widely dispersed whilst the larger size of other lead particles tends to result in localized deposition.

Other significant sources of lead in the air include secondary smelting and refining of lead, combustion of waste oil and incinerator emissions. In 1985, total lead emissions from municipal, domestic and industrial or commercial incinerators was estimated at 11.4 tonnes. Activities such as

All measurements quoted are in grams (g), milligrams (mg) or micrograms (μ g). 1g = 1/1000 kilogram, 1mg = 1/1000 gram and 1 μ g = 1/1000 milligrams. m³ = cubic metres.

sandblasting and paint removal from steel structures, for example bridges, can also contribute significantly to air lead levels. Smelters - copper and lead, power generators, iron, steel and Portland cement production tend to be the major source of lead emissions in rural and suburban locations.

Regulations on air lead levels target emissions and ambient air concentrations. Responsibility for monitoring and enforcing these controls falls predominantly on State Governments, although the NH&MRC (National Health and Medical Research Council) issues ambient air objectives and guidelines for air quality. The emission limit for heavy metals (combined and including lead) at ground level from a stationary source is 10mg/m^3 and the ambient air objective is less than $1.5 \mu \text{g/m}^3$ averaged over three continuous months and not more than once per year. Generally these limits are monitored by the State EPA, as happens in NSW, with audits of stacks and industry-operated monitors occurring randomly or in response to community complaints. There is no direct monitoring of vehicle emissions although this situation may change in NSW with the advent of new controls on motor vehicle emissions.

Monitoring and regulation of lead levels in petrol is presumed to largely result in an indirect control over vehicle emissions. Practically, however, this is not the case. This assumption fails to take into account the vast differences in emissions from new and old cars and also in cars subject to varying levels of engine maintenance. Thus, vehicle emissions of lead can vary from practically negligible levels in modern, well tuned and maintained cars running on unleaded petrol to levels well in excess of all guidelines in old 'bombs'.

Reports in the media and confirmed in the NSW Government Lead Issues Paper (EPA, NSW Health, 1993) indicate that ambient air qualities are frequently exceeded along busy traffic routes and in heavily industrialized areas. However, the use of unleaded petrol has reduced lead emissions along major roads and this is expected to continue with the increased use of unleaded petrol and with further reductions in the concentration of lead in leaded petrol.

(b) Soil

Although some soils in Australia contain high, naturally occurring, background levels of lead, the major source of lead in soil is lead that has leaked or been deposited from other sources. Lead in air, again particularly from motor vehicle emissions, is a significant source of lead in soil in urban areas with lead from acute sources - sandblasting, smelters and paint removal - contributing greatly to localized lead hot spots.

Studies have shown significant differences in soil lead levels between urban, suburban and rural areas. Decreased volumes of traffic in rural areas result in decreased soil lead levels. Thus, with the exception of rural areas adjacent to smelters or mines, soil lead levels in the country tend to

be significantly lower than levels in the cities. Lower soil and air lead levels in country Australia occur largely because of the lack of traffic and heavy industry and might be even lower still except for the tendency for more motor vehicles to run on leaded petrol in country areas, no doubt due in part to the widespread and lengthy rural depression. Lower lead levels in the air and soil of rural areas of NSW might also result if the higher permitted levels of lead in petrol in rural areas were reduced in line with the permitted lead levels in urban areas.

Lead in the soil is extremely persistent and has been suggested to endure for up to 20 000 years. Plants grown in leaded soil take the metal up into their roots and fruits and leaves may also contain significant quantities of lead. Such lead in soil may leach into water supplies and thus be removed, but heavily leaded soils need to be totally replaced as even measures such as top soil dressing to cover the lead layer have proven ineffective in the long term.

Guidelines for the assessment and management of contaminated sites issued by the NH&MRC and ANZECC (Australian and New Zealand Environment and Conservation Council) identify lead levels of 300mg of lead/kg of soil as of concern (NH&MRC and ANZECC, 1992).

Lead levels in the soil around houses with lead base paint have been reported to reach 1000 - 2000 mg/kg. Soil adjacent to the Mulgrave Freeway in Melbourne prior to its opening contained only 60 mg/kg; six years later in 1980 when 37 000 vehicles per day used the freeway, soil lead levels were measured at 1250 mg/kg! Soil lead levels in Port Pirie have been measured in the range 500 - 100 000 mg/kg, averaging 3924 mg/kg, indicating just how much lead can accumulate in soil surrounding or adjacent to lead production or refinery sites.

The endurance of lead deposited in the soil necessitates consideration of past sites of high lead exposure as potential sources of lead contamination. However, identification of such sites has largely proceeded on an ad hoc basis throughout Australia and thus quantification of the exact number of contaminated sites where lead is a problem in NSW has yet to occur. Some EPA sources suggest that up to a quarter of contaminated industrial sites throughout Australia may have a lead problem; given that 7000 contaminated sites have been identified in NSW, the clean-up operation for as persistent and highly toxic a substance as lead will be expensive, time-consuming and difficult.

Regulation of contaminated land in NSW falls under the auspices of the EPA which has the power to prevent contamination by imposing conditional licences for industrial use and which also may seek a bond against the possibility of contamination, and issue "Clean-Up" notices on suspected contaminated sites. ANZECC guidelines for the cleaning up of contaminated sites are followed in most states of Australia. However, these guidelines are not as stringent as those required by many countries overseas. Also, as made clear by the ANZECC and NH&MRC 1992

guidelines, a coordinated approach to cleaning up contaminated sites is essential and is previously unlikely to have occurred.

(c) Water

Most lead in water is leached from lead contaminated soil or results from effluent containing high levels of lead released into waterways. Lead in drinking water, however, results mostly from corrosion of materials used in water supply and distribution (plumbing). In particular, lead solder was used extensively in plumbing in new homes during the seventies. Areas with soft or slightly acidic water are also more prone to higher lead levels in drinking water as lead is more soluble in waters containing less calcium and magnesium. Heated water also is likely to contain higher levels of lead as heating increases the solubility of lead and accelerates corrosion of lead solder or lead flux in copper pipes. Water left sitting in pipes overnight or due to infrequent use contains significantly higher concentrations of lead and thus taps should be run until the pipes are fully flushed before water is used for drinking or food preparation.

Although these sources of lead in drinking water are well recognized, there have been virtually no studies, either in Australia or overseas, systematically examining the contribution of these factors to the overall lead content of drinking water. In addition, although water sources are frequently monitored by environmental authorities, first flush water is seldom tested and monitoring programmes tend to concentrate on water distribution systems not on individual household plumbing.

Lead pipes are no longer used in water supply or plumbing throughout Australia, however, many older houses still contain original pipes which were quite likely to be lead. The phasing out of lead solder in plumbing fittings has also been much more recent and, to date, lead solder use in plumbing is not banned anywhere in Australia. Brass plumbing fittings have been found to contain up to 12 % lead and bronze fittings up to 15 %. The surface area of pipes and the number of joints also affect corrosion and influence lead levels; smaller, longer pipes with more joints are associated with higher water lead levels.

Another potential and, as yet largely unexplored, source of lead in drinking water is rainwater tanks. Studies examining the lead content of water in rainwater tanks are virtually non-existent and although the use of lead in tank construction is banned, lead solder is able to be used in sealing tanks. Voluntary phasing out of the use of lead solder has occurred but the absence of any regulatory body or guidelines raises the questions as to the extent and persistence of lead contamination of rainwater via lead solder use.

The NH&MRC recommends a maximum content of $50\mu g$ of lead/litre in drinking water. However, this is a legal standard only in Western Australia and is five times greater than the limit suggested in recently released new

draft guidelines issued by the NH&MRC. The draft guidelines correspond to the new WHO draft guidelines which also recommend a maximum lead content in drinking water of $10\mu g$ of lead/litre. Although welcome, the draft guidelines are still higher than those recommended by the US EPA and have yet to be enforced anywhere in Australia despite considerable evidence linking lead consumption to adverse effects in humans.

(d) Other major sources of lead

All other sources of lead are essentially secondary sources. Nonetheless, the potential of such secondary sources to contribute significantly to lead contamination is great and thus reduction of these sources needs to be considered in any lead abatement programme.

One of the most common and problematic secondary sources of lead is paint. Until the 1970s lead was widely used as a pigment, dryer or corrosion inhibitor in paint. It is estimated that there are over 3.5 million houses in Australia built prior to 1971 with lead based paint on either interior or exterior surfaces. It is also estimated that there are over 200 bridges in NSW that still are painted with toxic red lead primer. Paint presents a particular hazard because the integrity of painted surfaces tends to decline with time and weathering resulting in dust and paint chips containing relatively high concentrations of lead. Renovation and sandblasting therefore have the potential to generate large enduring deposits of lead, disposal of which may prove difficult.

Other potentially significant sources of lead exposure include cosmetics and hair dyes (assessment of the concentration of lead is these products has not been consistently completed, although there are now proposals for government regulations pertaining to the maximum concentration of lead in such products and to oblige manufacturers to label products with the lead content); plastics where lead additives have been commonly used for many years; lead shot or ammunition, the use of which has contributed markedly to the occurrence of lead poisoning leading to reproductive failure and death in wildlife, particularly migratory birds who tend to ingest lead shot that accumulates in the wetlands; lead glazes in handmade pottery and earthenware; lead in herbal and 'natural' remedies which are often imported from countries with less stringent restrictions on the amount of lead present in the ingredients of such products or in the utensils used for preparation²; crystal where lead is added to enhance lustre and density; liquors left to stand in crystal decanters or glasses can accumulate

There have been cases reported in the mainstream scientific literature of patients presenting with symptoms of lead toxicity which upon further investigation were found to be due either to ingestion of herbal remedies containing unaccountably high concentrations of lead or which were found to have been prepared using 'traditional' earthenware utensils manufactured from materials containing a high concentration of lead. During preparation of such remedies the lead from the utensils leached into the brew and was later ingested.

significantly increasing amounts of lead over a surprisingly short period of time³; lead present in the coatings of crayons and pencils, frequently sucked or chewed by children; lead used in yacht keels or as weights for fishing lines or curtains.

Although, individually, such sources of lead tend to be minor, collectively, and particularly in urban, heavily industrialized areas or in areas adjacent or in the midst of major traffic routes, even small exposures can provide the proverbial straw and push an individual's exposure over recommended levels.

Lead in food is largely the result of the absorption of lead from soil or water contaminated by lead during plant growth or due to the contamination of food during its preparation, either commercial or domestic. Maximum permitted concentrations for lead in food vary between 0.2mg/kg of food to 2.5mg/kg. Food additives currently may contain up to 10mg/kg. New regulations under consideration propose limiting the lead content in beverages to 0.2mg/kg, in infant foods to 0.3mg/kg and in all other foods to 0.5mg/kg. Such regulations would certainly simplify matters and would represent a considerable advance on the current regulatory situation both in terms of reducing the overall level of lead permitted in food and also by making the regulations much more uniform.

Lead solder on cans was previously thought to be a significant contributor to lead contamination of food. The use of lead solder on food cans has been banned in many countries (not in Australia) and welding used to seal cans instead. Acidic foods such as tomatoes or pineapple are particularly prone to contamination from lead solder as lead is corroded more easily in an acidic medium. Proposals put forward by the NH&MRC suggest that the use of lead soldered tin cans should be banned both in relation to locally produced cans and imported ones. Many companies have voluntarily switched to welded cans already and it should be noted that the use of lead soldered baby food cans has been prohibited for some time.

Before leaving a discussion of sources of lead, it is important to emphasize that the effects of lead exposure are cumulative and enduring. Thus it is not sufficient to merely reduce or remove identified sources of exposure over a number of years and expect a corresponding reduction in lead levels as lead is ubiquitously distributed throughout the twentieth century environment and many potential sources of lead contamination have yet to be fully investigated. For example, the extent of use of lead solder by home handypersons remains uninvestigated as does the use of lead in the

One study showed that port (with an initial lead concentration of around 90µg/l) left standing in a crystal decanter had around fifty times more lead (about 5000µg/l) after four months. Another study showed that the concentration of lead in wine poured into a crystal wine glass doubled over one hour and tripled over four hours. Leaching of lead from crystal ware can be avoided, however, by soaking the crystal in vinegar for 24 hours and then discarding the solution and washing thoroughly.

preparation of cosmetics and hair dyes. Any lead abatement strategy needs to consider the minor, apparently unlikely, sources of lead as well as the major well documented ones.

4 Effects of Lead

- (a) Effects of lead on intelligence
- (b) Effects of lead on the cardiovascular system
- (c) Effects of lead on foetal growth and maturation
- (d) Metabolic effects of lead

Lead is a well established mutagen⁴ and teratogen⁵ and is known to have no beneficial physiological effects in humans. To date, there is no substantial or definitive evidence suggesting that lead acts as a carcinogen⁶. Many human and animal organs and systems, including the kidney, heart, blood and central nervous system (brain and nerves) are highly susceptible to the toxic effects of lead.

The effects of lead are often insidious and go undetected because although there are measurable changes in the biochemistry of the blood, there is no apparent pathology until extreme exposure and lead poisoning occurs. Despite the plethora of well-designed studies examining the effects of lead on a range of human and animal physiological systems, the lowest concentration of lead that will result in clinically measurable effects has yet to be clarified.

Lead is absorbed mainly from the gut although some lead in air can be absorbed via inhalation through the lungs. Women are more susceptible to the adverse effects of lead than men but male children tend to have higher blood lead levels than female children, in part possibly because of their increased tendency to eat soil and dirt, suck objects and play outside. Children absorb lead three to four times more efficiently than adults⁷ and are also much more likely to indulge in pica (picking up and eating soil and dust) and hand to mouth behaviour than adults. Children, particularly infants and toddlers, are therefore much more vulnerable to lead poisoning.

A mutagen is any substance which is known to cause or promote the occurrence of sudden and random changes in the genetic material of cells. Such changes may give rise to defects as apparent as Down's Syndrome, others cause changes which have no apparent visible physiologic result. Mutagens can affect either (or both) reproductive and non-reproductive cells. Where reproductive cells are affected, the change is likely to be passed on to the next generation.

A teratogen is any environmental factor known to act on the foetus to cause a congenital abnormality. Common examples include x-rays and the measles (rubella) virus.

A carcinogen is a cancer causing agent.

Lead absorption in children has been estimated to be of the order of up to 50 % of ingested lead compared to just 10 to 15 % in adults.

Studies have identified the primary source of lead absorption in children as ingestion of lead from dust, soil, paint, drinking water, parental occupations, air and food. Although, individually these sources may be minor, the cumulative effect is often significant.

Lead accumulates in body tissues, in particular in bone. It has been estimated that up to 95% of the body burden of lead may be stored in the bones and teeth and even if blood lead levels are reduced, significant body stores of lead may therefore remain and prolong exposure to toxic side effects.

The toxic effects of high concentrations of lead have been known for hundreds, if not thousands of years. Australia has been at the forefront of research examining the effects of lead on health and behaviour; the study by Turner and Gibson in Queensland in 1904 was the first in the world to demonstrate an association between lead based paint and poisoning in children. Epidemiological data collected in studies, such as the one completed in Port Pirie (Baghurst et al, 1992), is of as high a standard as data collected elsewhere.

Unfortunately, even with the availability of a number of good quality studies, uncertainties as to the significance and strength of findings are still a problem. In part, difficulties are inherent as the effects examined are often small, particularly in relation to concentrations of lead well below those conventionally defined as toxic, but, also because of the problem of accounting for possible confounding factors which make the interpretation of research even more problematic.

Despite these difficulties, a number of consistent and well verified effects of lead on various aspects of human physiological functioning and human behaviour have been identified. The most important of these are discussed in the following paragraphs.

(a) Effects of lead on intelligence

Lead adversely affects the central nervous system. High blood lead levels in children have been correlated with behavioural abnormalities, learning problems and hyperactivity. The most profound associations between high blood lead levels and children's intelligence are observed in children aged between six months to two years. Children with higher blood lead levels have lower IQ scores and these effects are observed in children with blood lead levels in the range $10 - 25\mu g/dl$. In children aged four or greater, every $10\mu g/dl$ increase in blood lead is associated with a 1-3 point decrease in IQ.

In Australia, the ramifications of using the $10\mu g/dl$ level as the level of

concern are immense⁸; half of all Australian children aged less than four may have blood lead levels higher than $10\mu g/dl$ and half a million Australian children are estimated to have absorbed lead in levels capable of causing brain damage. Approximately 100 000 children nationwide are estimated to have blood lead levels greater than $15\mu g/dl$ and a further 300 to 600 000 are estimated to have blood lead levels in the range $10 - 15\mu g/dl$.

At blood lead levels above $40\mu g/dl$, medical treatment is required as otherwise permanent severe brain damage and even death may result. The effects of lead exposure at even low levels are likely to persist and there is reasonable evidence to suggest that deficits in IQ and/or behavioural alterations present in pre-school children will endure through to the teenage years.

The effects of lead on intelligence are not limited to children. Studies examining the verbal reasoning skills of lead workers have shown that workers with a blood lead average of $25\mu g/dl$ show a significant impairment of these skills when compared to lead workers with an average blood lead level of $13\mu g/dl$.

It is extremely difficult to accurately quantify the lower limit for observing any effects of lead exposure and this is the primary reason for controversy and dispute concerning recommended safe levels of lead as measured by blood lead issued by bodies such as the NH&MRC. Also it seems clear that blood lead levels are only an indication, albeit a good one, of lead exposure and the true effects of exposure can only really be gauged by taking into account various other factors such as socio-economic status and diet.

Much of the controversy concerning studies of the effects of lead is related to the greater apparent susceptibility of indigent, urban children to the toxic effects of lead. Various studies have demonstrated that socio-demographic factors such as substandard housing, low socio-economic status, large family size, low household cleanliness, infrequent handwashing, unemployment, inadequate parental supervision and poor parent-child interaction are related to blood and tooth lead level. The

Estimated percentage of Australian children aged 0 to 4 years with blood lead levels greater than 10, 15, 20 and 25µg/dl

Australia	44.6	15.4	5.5	2.1
	97.5	80.4	52.6	29.1
Rural During renovation	5.5	0.3	0.02	0.00
Other urban	23	2.9	0.4	0.04
Major urban	60	19	4.8	1.1
	>10	>15	> 20	> 25

Data from the Newcastle Environmental Toxicology Research Unit, 1993.

quality of the care-giving environment has been shown to play a major role in early lead exposure in disadvantaged populations with the extent of childhood lead exposure determined to a significant extent by the quality of environment and standard of care experienced by the child.

It should be clearly emphasized, however, that these findings do NOT mean that children with high blood lead levels are neglected or that it is entirely the fault of parents that children are exposed to lead. It is obviously necessary to consider the care-giving environment when evaluating the effects of lead exposure on children but it is not sufficient to merely attribute the occurrence of blood lead levels of concern to poor parenting or difficult socio-demographic conditions and then do nothing. The same studies that show increased susceptibility of poor urban and disadvantaged populations to lead exposure also list factors such as the presence of petrol stations, major or minor highways, four-laned roads and industrial sites within six blocks as factors significantly correlated with blood lead levels.

The only viable conclusion therefore is that, although the care-giving environment may contribute to lead ingestion by children, largely through poor cleanliness, supervision and hygiene, the lead is in environments initially because of external factors beyond parents' control. It is also interesting to note at this point, that the strong age-association with blood lead levels which peaks at around two years is probably as much to do with environmental and behavioural patterns as decreased exposure. Families tend to move to less industrialized areas as children age and become more mobile and children indulge in less hand-to-mouth behaviour after age two. As with many problems evident in twentieth century living, it is the poor and disadvantaged peoples who suffer most from the adverse effects of lead, but no one, no matter how high their socio-economic status, is exempt from risk.

Other effects of lead on the central nervous system include the slowing of the transmission of nerve impulses, interference with the actions of various neurotransmitters⁹ and the development of encephalopathy¹⁰.

(b) Effects of lead on the cardiovascular system

Lead exposure and high blood lead levels have been shown to be associated with an increased incidence of adult heart attacks and strokes.

Neurotransmitters are naturally occurring substances such as adrenaline which regulate the activities of all the organ systems around the body by their actions on receptors on cells. Some neurotransmitters are excitatory, some inhibitory. Interference with their function crucially affects the capacity of the body to maintain normality.

Encephalopathy is irritation of the brain not caused by an injury or localized infection. Symptoms include high fevers and convulsions. Death may result particularly in children whose developing brains are more susceptible to permanent damage.

Men are particularly susceptible as they are most at risk from heart disease and also the group least likely to be aware of their blood lead levels and the relationship of those levels to an increased risk of suffering a heart attack. Increased blood lead levels have also been associated with a small increase in blood pressure. It has been estimated that reducing blood lead levels below $10\mu g/dl$ may save around \$4.6 billion dollars currently spent remediating the effects of childhood brain damage and adult heart attacks and strokes.

(c) Effects of lead on foetal growth and maturation

As mentioned earlier, lead has been shown to be both a mutagen and a teratogen. In addition, there is some evidence to suggest that blood lead levels greater than $15\mu g/dl$ are associated with premature births and inadequate foetal growth. Most authorities recommend that pregnant women avoid exposure to lead, and lead abatement programmes need to target women involved in a flurry of home renovation to accommodate the new baby as renovation of older houses often generates dust and paint chips containing significant amounts of lead.

(d) Metabolic effects of lead

Lead severely affects the formation and life span of red blood cells. Lead alters the formation of haem, the protein in red blood cells which binds to oxygen and transports it around the body. In children up to one year of age, blood lead levels of 20 - $39\mu g/dl$ have been shown to cause anaemia. Blood lead levels greater than $20\mu g/dl$ have also been shown to result in a depletion of Vitamin D. Although these affects are usually only observed at the higher end of blood lead levels, the tendency of many women and children to be iron deficient or to have an iron deficient diet may accentuate the occurrence of lead induced anaemia to a clinically obvious level. Iron inhibits the uptake of lead by red blood cells and thus iron in the diet acts as a protective factor against lead poisoning. Children are roughly twice as susceptible to the anaemia inducing effects of lead than adults and thus nutrition is an important factor mediating the effects of lead upon child development.

Lead also appears to interfere with drug metabolism so that some drugs will have extended half-lives¹¹.

The half-life of a drug is amount of time (usually hours but perhaps days) it takes the body to get rid of half the initial dose of drug taken.

5 Screening programmmes for detecting blood lead levels

One of the repercussions of wider advertising of the effects of lead on children and the endemic contamination of urban and industrial environments by lead has been a call for wide-scale screening of individuals, particularly children, to measure blood lead levels and identify children at risk. Unfortunately whilst such an idea may sound sensible, the logistics of implementing it take on nightmarish proportions and there is considerable doubt in the mainstream scientific literature as to whether such a programme would be worthwhile let alone cost effective. In practical terms, governments have limited monies to spend on the control and reduction of lead exposure. Wide scale blood lead screening is costly and may contribute more to hysteria than to knowledge of the real extent of the lead problem.

Overseas experience with blood lead screening programmes, particularly in the USA and the UK, suggests that it is effective and useful in communities where the risk of high lead exposure is considerable and where other resources are available to counsel families and carers on the meaning of any such tests. Simply identifying children with high blood lead levels is not enough. If parents do not have the resources or means to do something about those levels then considerable anxiety and confusion will result. In addition, even if parents are aware of the blood lead levels of their children, this does not increase the removal of lead from the environment. Arguably, awareness of the problem of lead is large enough now to assume that most people are aware of the risks of exposing their children to lead and what really is required are strategies to reduce the lead in the environment and practical information for parents and carers on how to avoid lead.

Screening programmes may be useful initially in places of high lead contamination such as Broken Hill, but on a wider scale, their use is likely to cost more money and anxiety than is worthwhile. It should be emphasised, however, that parents who do seek blood tests from their local GP should not be brushed off or dismissed as neurotic. GPs need to be educated as to the significance and extent of the lead problem throughout Australia and pathology labs need to upgrade their procedures to provide reliable and sensitive blood lead tests.

6 Ways to reduce lead

- (a) Lead in petrol
- (b) Lead in paint
- (c) Removing lead from soil or contaminated sites
- (d) Removing lead from other sources

Despite considerable disagreement between interest groups concerning the exact policies and programmes that should be implemented as part of a lead abatement scheme, there is general consensus on some key issues.

(a) Lead in petrol

It is imperative that Australia as a whole, and NSW in particular, implement effective reduction and ultimate removal of lead from petrol. Despite the introduction of unleaded petrol in 1985, and regulations requiring all new cars to run on unleaded petrol in 1986, Australia lags badly behind much of the industrialized world in reducing lead in petrol. There is also considerable variation across Australia; Victoria currently has the lowest permitted level of lead in petrol - 0.3g/l - whilst country NSW and Queensland have the highest levels - 0.84g/l. There is no apparent reason for the continuation of different levels between urban and rural centres, especially since many oil companies have voluntarily reduced the amount of lead in petrol supplied to rural centres.

In the Lead Issues Paper¹² a target concentration of 0.15g/l in leaded petrol is proposed. Whilst this target is obviously valuable, the real problem is the continued use of cars running on leaded fuel. Approximately 60 % of the petrol sold in NSW contains lead and this is largely because of the much slower than anticipated turnover of motor vehicles. When unleaded petrol was introduced in 1985, it was expected that there would be a constant and reasonably quick phasing out of leaded petrol cars. However, due to a number of factors, including the recession, this has not happened and the average age of cars in Australia is of the order of 9-10 years. Thus only 45 % of the total car fleet runs on unleaded petrol and there are very few incentives for cash strapped Australians to buy new unleaded petrol cars. A number of proposals have been made to tackle this problem.

Firstly, there is wide support for a differential pricing structure between unleaded and leaded petrol. Unfortunately, whilst most people will

NSW Government Lead Issues Paper is a joint paper prepared by the NSW EPA and NSW Health. It was published in March 1993 and is intended to form the backbone of the Government's strategy for tackling the lead problem in NSW.,

acknowledge the sense of such a strategy and allow that the Federal government budget proposal along these lines was reasonable, there are real problems accommodating the tendency for an unfair burden of such a manouveure to fall on the shoulders of those who can least afford it. Pensioners, lower income earners and younger people are more likely to own older cars and thus to be buying leaded petrol. These groups therefore are likely to suffer disproportionately.

Secondly, older car owners could be offered subsidies or incentives to purchase newer model cars. Simultaneously the price of new cars could be made more affordable by reductions in tariffs. However, this would have serious economic and policy implications.

Thirdly, owners of cars manufactured pre-1986 could be informed of the possibility of running their cars on unleaded fuel. Of these, the latter suggestion would seem to be the most viable in the short and medium term. Over 30% of cars manufactured before 1986 are able to run on unleaded petrol without any modifications and a further 30% of cars would require only minor modifications. (For a list of models and cars see Appendix 1). Despite considerable money spent on a campaign to educate drivers of this fact, most car owners are unaware of the possibility of running their pre-1986 cars on unleaded fuel. Governments need to address this anomaly and encourage drivers to use unleaded petrol. It should be emphasized, however, that this is really only a stop-gap measure as the ultimate aim is to remove unleaded petrol from the marketplace.

(b) Lead in paint

The use of lead as a pigment in domestic paint has already been banned. The problem therefore is restricted to lead in paint in houses built prior to 1970 and to lead in industrial paints. Most NSW councils have banned or phased out the use of red lead primers already. However there are significant numbers of structures, particularly bridges which still have lead based paint and these need to be cleaned and repainted in lead free paint. The real problem therefore lies in alerting the public and local governments to the need to take special precautions when removing paint from surfaces and when disposing of paint chips and dust. An education programme has been mooted as one way to inform would-be home renovators of the dangers involved in removing lead paint, but this would not address the practical problems.

Home renovators are often renovating because they are unable to afford to have professional assistance and they are also often unable to afford alternate accommodation whilst they renovate. Disposal of paint chips and dust is also often ad hoc as local councils provide no specialised collection services and paint residue is disposed of in hired skips or at the local dump. Home renovators therefore not only need information about the potential dangers involved in renovating, but also need to be supplied with practical alternatives for rubbish disposal.

(c) Removing lead from soil or contaminated sites

Aside from lead in petrol, one of the biggest problems to be overcome in any lead abatement plan is that of removing lead from contaminated sites, be they large industrial sites or small domestic ones. Chemical treatment of soil to remove lead is expensive, largely because of the labour involved in removing the soil, treating it and returning it. Often replacement of the soil with clean lead free soil is a more viable alternative. Unfortunately, this solution causes further problems as the lead still needs to be disposed of or made safe.

On a domestic level, it is often impractical and too expensive for households to remove all lead contaminated soil from home gardens and yards and instead top soil dressing with clean soil is tried. This method is not overly effective as the top soil ultimately disappears and the lead returns once more to the top layer. Sowing lawn over contaminated areas is more effective but both options tend to hide the problem rather than remove it. Wholesale removal of contaminated soil is really the only completely safe means of creating lead free yards and gardens, but it is costly and difficult.

Large contaminated sites such as Boolaroo and Broken Hill need to be addressed in programmes tailor made for the towns, their people and the resources available. Such programmes would have to be coordinated at a state government level if they are to prove effective and that of course requires money and resources that may be difficult to find in the short-term. Taking a long term perspective, evidence from many sources suggests that tackling the lead problems in those areas is not only cost effective but will avoid the problem of burgeoning costs from a growing lead problem.

(d) Removing lead from other sources

Most of the other sources of lead could be regulated more easily if there were Australia-wide uniform limits on the amount of lead in various products and if manufacturers and importers were forced to list lead as an ingredient or component of products and warn consumers accordingly. Measures such as completely banning the use of lead solder in all applications and reducing the amount of lead in plumbing fittings would go a long way towards reducing lead contamination of water supply and distribution systems.

In addition, further work needs to be completed in identifying other potential sources of lead contamination such as lead solder in rainwater tanks. Research efforts should target these small but significant sources of lead. Highly contaminated water streams and supplies may need to be chemically cleaned or isolated. Implementation of the new guidelines for lead in food proposed by the NH&MRC and bringing regulatory authorities into line across the nation would also facilitate the removal of lead from

the environment. The extent of lead contamination in products such as cosmetics and pottery also needs to be investigated as otherwise consumers will in all innocence continue to expose themselves to lead.

Overall, the aim of lead abatement programmes is to reduce the number of Australians, particularly children, who are most susceptible to harm from lead, exposed to levels of lead which push them over the recommended blood lead level.

In June this year, the NH&MRC, after considerable debate, reduced the recommended blood lead level from $25\mu g/dl$ to $10\mu g/dl$. Although a reduction had been expected, the recommendation was lower than many groups expected and this has had several repercussions. Firstly, the number of children with blood lead levels of concern increased dramatically and, in association with media attention, concern was felt as to the significance of this increase on demand for resources to combat the problem. Many parents who had felt comfortable that their child was not exposed to undue amounts of lead were forced to think again and many organizations and individuals have been confronted with a lead exposure problem where previously apparently none existed.

This is not to say that the NH&MRC erred in any way in recommending a low blood lead level as one of concern, nor can it really be suggested that the level recommended is too low; evidence from studies completed both here and overseas has shown significant and substantial detrimental effects associated with blood lead levels above $10 - 15\mu g/dl$ and the NH&MRC would have been negligent not to adjust Australian recommended blood lead levels accordingly. What does need to be considered, however, is that, virtually overnight, there was an explosion in the number of children at risk from lead, and the resources required to combat that explosion and eradicate lead from the environment cannot be produced, or abatement programmes implemented in the same instance.

All governments in Australia need to join in tackling the lead problem. Devising a concerted uniform policy at all levels of government for lead abatement is essential because lead is endemic in our communities and protection of our children will only result from united and considered efforts.

7 NSW Lead Taskforce

As a result of the NSW government's lead issue paper, a taskforce on lead has been set up. The priorities and aims of this taskforce are amply described in the Issues Paper, but it is of interest to note that in contrast to many similar situations, the taskforce has made progress. Not only are reports expected from the eight working parties (lead in air, paint, soil and dust, water and waste water, petrol and Broken Hill) early next year, but the Government is currently considering proposals from both the Broken Hill and petrol working parties.

8 Conclusion

In general, there is sufficient good quality research data to show that lead has substantial and consistent adverse effects on a range of body systems and functions and that action is required to reduce lead exposure.

Of particular concern is the effect of lead on the cognitive development of children, and most efforts at reducing human lead exposure are driven by an understandable desire to protect our children.

There are still difficulties in quantifying the extent and full nature of the effects of lead on intelligence and behaviour.

The importance of securing funding for continuing Australian studies examining these effects cannot be overestimated. There is certainly enough evidence now to warrant action on lead abatement, but further information is needed if lead abatement programmes are to target the most needy areas in the most cost and time effective way.

Overseas data largely corresponds to Australian data, but significantly different climatic and socio-demographic situations necessitate that Australian studies continue in conjunction with lead abatement programmes.

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WHO CAN USE UNLEADED?

ARS MADE from 1986 on run on unleaded petrol only. If you have a car currently running on leaded petrol you may be able to use unleaded petrol in it without modifications. The following list comes from the Federal Chamber of Automotive Industries. Some of these models can only use unleaded petrol if they get a regular dose of leaded petrol as well, or have minor adjustments made — contact the dis-tributor to find out

Make/m	odel	Models which can use unleaded petrol			
AUDI					
80 Fox		1977 onwards			
100SE		1978/1979			
BMW The followi	ng vehic	les can use <i>piemium</i> unleaded petrol			
2002 Sep		t 1975-March 1976*			
318i Sept 1		982-Sept 1985*			
320		nder Sept 1975-Aug 1977			
320		nder (UK spec.) Sept 1977-Aug 1982°			
320i		nder Oct 1975-March 1978			
323i	Jan 19	978-Sept 1985*			
520i	April 1	1981-Sept 1985*			
528i	Aug 1	977-Sept 1985*			
*Leaded pe	trol sho	uld be used at every fifth fill.			
DAIHATSU					
Charade	G.	10, G11 Series			
Delta Truck	s V6	60,V67, V68 Series 1978 onwards			
Handivan	L6	O Series 1982 onwards			
Hi-Jet		50,\$65, \$70, \$75 Series 1978 onwards, '6 Series 1984 onwards			
Rocky	F8	0, F85 Series 1984 onwards			
FORD					
laser/Mete	or an	June 1984 onwards (except Laser sport and Meteor with high performance engine)			
Telstar, TX	5 Ju	ne 1983 onwards			
Econovan 1	L Ma	arch 1984 onwards			
Spectron 1	L M	arch 1984 onwards			
Courier 2 L	Ju	ne 1983 onwards			
HOLDEN		•			
Astra	LE	Series 1.5L			
Drover	QE	Series 1.3L (manual)			
Gemini		T,TC,TD,TE,TF,TG Series 1.6L			
Jackaroo		3S Series 2L (manual)			
Rodeo		Series 1.6, 2L (manual)			
Shuttle		FR Series 1.8L (manual)			
HONDA -		ded petrol every third fill			
Accord, Ac					
Prelude		1980 onwards			
petrol, Adju	ng vehic strnent ns shou	R les can run on <i>premium</i> unleaded of Ignition timing and/or engine Id be carried out by Jaguar franchised			
XJS HE		V12 (5.3) 1975–1981, V12 HE (5.3) 1981–1989			
		6 Culinder (4.2) 1068-1086			

6 Cylinder (4.2) 1968–1986, V12 (5.3) 1975–1981, V12 HE (5.3) 1981–1989

XJS, Sovereign and Daimler

	MAZDA 323	MAZDA 323 1983 onwards (except 'SS' series)			PORSCHE For all these models. Porsche recommends using one			
323wagon 1983 onwards				except oo series;			er every three tankfuls of	
626 1983 onwards							The minimum fuel octane e pertinent owner's manual is	
	929			except EGI fuel injection)	always the dec	isive point a	s to whether or not unleaded	
	929wagon	•		except fuel injection)	petrol (91-93 RON) can be used.			
	82000	1982 on		except thet injection/	911T, 911E		1972-73	
	E1800/E2000	1984 onv			9115, 911		1972-77	
	RX-2,RX-3,		Maius		91 1Carrera 2.7		1972-75	
	RX-4, RX-5	All			91 (Carrera 3.0		1976-77	
	RX-7	1979 onv	vards		911 SC 3.0		1978-79	
	MERCEDES-BI				928		1978-79	
	The following i	information	applies to Australian-		ROVER			
	specification v	ehicles only	. Owne	ers of vehicles not z Australia would need to	3500 SDI	All		
	check their veh				Quintet	All		
I	380 SE Saloon		0 001117		Land Rover	8.13:1 Co	mpression ratio engines only	
	380 SEL Saloo		_	September 1981	Range Rover	8.13:1 Co	mpression ratio englnes only	
	380SEC Coupe			onwards	SUBARU			
	380SL Roadst				1800 Sedan &	S wagon	(EA81) 1980-84,	
i	230E Saloon		_	February 1984	1000 Sedan &	S. Wayun	Including MV Brumby-1985	
I	230TE Station wagon			onwards	Sherpa 700		(EK42) 1982-85	
			aloon		1800 "L" Serie	s	(EA82) with carburettor 198	
	280 SE (W 116 and 126) Saloon 280E (W123) Saloon		MIUUII	Before August 1982 (W116 from May	1800		(EA82) XT vortex, turbo, fue	
	280CE (W123)			1978)	*Use premiun	unleaded pe	etroł.	
Į	280 TE (W 123	Station W	agon		SUZUKI			
I				trol operation provided	Carry	ST90 Seri	es 1980 onwards	
l	leaded petrol u		ry tiltth		Hatch	540 cc an	d 800 cc	
l	380 SE Saloon		_	Before September 1981	Slerra	1.0L, 1.3	L	
I	380SEL Saloor		_		Swift	1.0 L		
I	380SL Roadsto	er		D (1 4004	TOYOTA			
Į	230E Saloon		_	Before January 1984	Cellca (21R-C	en gine)	1981-1983, RA60	
I	230TE Station				Corolla (3K-C	ona)	1974-1978, KE 30,	
i	280SE (W126)		_	September 1982		••	KE 35, KE 36	
Į	280E (W123) S			onwards	Corolla Uftbac	k (3K-C eng)	1977-1978, KE 50	
į	280CE (W123)				Corolla (4K-C	ena)	1978-1980, KE 55, KE 38;	
i	280 TE (W123	121	agon	All			1981~1984, KE 70 1985 Refer owner's manua	
ļ	250 (W123) Sa MITSUBISHI (I		All		Corolla (2A-C eng) Corolla (4A-C eng)		for details	
I	Scorpion		_	, GK, GL, 2.6L				
I	Colt			3, RC, 1.4L, 1.6L	T18 Liftback (3T-C eng)		1979-1983, TE 72	
ı	Cordia				Corona 2 Litre (21R-C eng) Corona MK-2 (4M eng)		1981-1983, RT 132, RT 13	
I	Lancer	1983-84,		LC, 1.2L, 1.4L, 1.6L		-1-	1976-1977, MX 13, MX23 1977-1980, MX 32, MX 36	
I				A, GB, GC, GD, 1.3L,	Cressida (4M		1980-1983, MX 62	
I	Galant	1.4L, 1.5L		A, do, do, do, 1.5E,	Cressida (5M-E eng*)		1977-1980, MS 83, MS 85	
ı	1.300 Express			SC, SD, 1.6L, 1.8L	Crown (4M eng) Crown (5M-E eng*)		1980-1983, MS 111, MS 1	
l	L200 Express			B, MC, MD, 1.6L	*Up to engine			
ı	Pajero	1983-85,			Op to engine	110. 3141-3423	YH 51/61/71, June 1984	
ı	· i			.85L, 2.6L;	Hiace (3Y eng)	onwards	
I	Sigma	1979-84,	GE, GH	I, GJ, GK, 2.6L; 1980	Liteace (4K, 4l	(-C ena)	KM20	
l				iJ, 1.6L; 1984 GK, 1.6L	Coaster Bus (2		RB20, 1982 onwards	
I	NISSAN (inclu	dın g Datsur	_		19 101/	-	YN57/60/65 series, October	
I	Pulsar van		E13		Hilux (3Y eng)		1984 onwards	
I	Pulsar sedan	_	E15		Hilux (1Y, 1Y-	C eng)	YN55 series	
I	Pulsar hatch		E13, E15		Tarago (3Y eng) 2 litre		YR21	
ı	Pulsar ET hatchback EXA				Land Cruiser		FJ62/70/73/75, 1985	
I	Prairie		E15				onwards	
I	Gazelle		CA20E		Land Cruiser New Model		RJ70, 1985 onwards	
J	Skyline		L24E		(22R eng)			
J	300C, 300ZX		VG30	DE .	Dyna		YU60	
I	Half tonne ute		A12		Landcruiser (2F Engine) SP		FJ40/60 manufactured specifically for sale in:	
J	Vanette		A15				NSW after 1977;	
J	Uivan		220 0				other states after 1979	
J	720		L18,		TRIUMPH	T-		
I	Patrol		L28 only		Stag	July 1976	onwards	
1	Cahetar		720 722		All			

1981-1984, KE 70	petrol (91-93 F		s to whether or not unleaded used.			
91 1Carrera 2.7 1972–75 911 Carrera 3.0 1976–77 911 SC 3.0 1978–79 928 1978–79 929 1800 1800 920 1800 1800 930 1800 1800 944 1982–85 945 1800 1800 946 1800 1800 947 1980 1800 947 1980 1800 947 1980 1800 947 1980 1800 947 1980 1800 947 1980 1800 947 1980 1800 947 1980 1800 947 1980 1800 947 1980 1800 947 1980 1800 947 1800 1800 947 1800 1800 947 1800 1800 947 1800 1800 947 1800 1800 947 1800 1800 947 1800 1800 948 1800 1800 948 1800 1800 949 1800 1800 940 1			1972-73			
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911 SC 3.0 1978-79 928 1978-1978, KE 50 1978-1983, RT 132, RT 1979-1983, RT 132, RT 1979-1983, RT 132, RT 1979-1983, RT 132, RT 1979-1983, MS 31, MS 80 1977-1980, MS 83, MS 80 1978-1980, MS 83, MS 80 1977-1980, MS 83, MS 80						
1978-79 1978-79 1978-79 1978-79 1978-79 1978-79 1978-79 1978-79 1978-79 1978-79 1978-79 1978-79 1978-79 1978-79 1978-79 1978-79 1978-798 1978-1980						
ROVER 3500 SDI						
3500 SDI			1978-79			
Quintet		All				
Land Rover 8.13:1 Compression ratio engines on Range Rover 8.13:1 Compression ratio engines on SUBARU						
Range Rover 8.13:1 Compression ratio engines on SUBARU 1800 Sedan & S. wagon (EA81) 1980-84, Including MV Brumby-198 1800			moression ratio engines onl			
SUBARU 1800 Sedan & S. wagon (EA81) 1980—84, Including MV Brumby—1981						
Including MV Brumby-198 Sherpa 700						
	1800 Sedan &	S wannn				
1800 "L" Series (EA82) with carburettor 19		O. Wagon				
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Hatch	SUZUKI					
Sierra	Carry	ST90 Ser	ies 1980 onwards			
Switt	Hatch	540 cc ar	d 800 cc			
TOYOTA Cellca (21R-C engine) 1981–1983, RA60 Corolla (3K-C eng) 1974–1978, KE 30, KE 35, KE 36 Corolla (4K-C eng) 1977–1978, KE 50 Corolla (4K-C eng) 1978–1980, KE 55, KE 31 1981–1984, KE 70 Corolla (2A-C eng) 1985 Refer owner's manufor details T18 Liftback (3T-C eng) 1979–1983, TE 72 Corona 2 Litre (21R-C eng) 1981–1983, RT 132, RT Corona MK-2 (4M eng) 1976–1977, MX 13, MX2 CressIda (4M eng) 1977–1980, MX 32, MX 32, MX 32, CressIda (4M eng) 1977–1980, MX 32, MX 32, CressIda (5M-E eng*) 1980–1983, MX 62 Crown (4M eng) 1977–1980, MS 83, MS 83, MS 83, MS 84 Crown (5M-E eng*) 1980–1983, MS 111, MS *Up to engine no. 5M-3429547 Hiace (3Y eng) YH 51/61/71, June 1984 onwards Liteace (4K, 4K-C eng) KM20 Coaster Bus (22R eng) RB20, 1982 onwards Hilux (1Y, 1Y-C eng) YN55 series Tarago (3Y eng) 2 litre YR21 Land Cruiser New Model (22R eng) RJ70, 1985 onwards Land Cruiser New Model (22R eng) Py060 Py0a YU60 FJ40/60 manufactured specifically for sale in: NSW after 1977; other states after 1979 TRIUMPH Stag July 1976 onwards Kombi CV engine, 1978–79 Microbus CV engine, 1978–79 Microbus CV engine, 1982-83 Transporter DH watercooled			L			
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Corolla (4K-C eng) 1978–1980, KE 55, KE 31 1981–1984, KE 70 Corolla (4A-C eng) 1985 Refer owner's mant for details 118 Liftback (3T-C eng) 1979–1983, TE 72 Corona 2 Litre (2IR-C eng) 1976–1977, MX 13, MX2 CressIda (4M eng) 1977–1980, MX 32, MX 32, MX 32 CressIda (5M-E eng*) 1980–1983, MX 62 Crown (4M eng) 1977–1980, MS 83, MS 83 Crown (5M-E eng*) 1980–1983, MS 111, MS "Up to engine no. 5M-3429547 Hiace (3Y eng) VH 51/61/71, June 1984 onwards Liteace (4K, 4K-C eng) KM20 Coaster Bus (22R eng) RB20, 1982 onwards Hilux (3Y eng) 1985 series Tarago (3Y eng) 2 litre VN57/60/65 series, Octof 1984 onwards Hilux (1Y, 1Y-C eng) YN55 series Tarago (3Y eng) 2 litre Land Cruiser Land Cruiser New Model (22R eng) Pyna YU60 FJ40/60 manufactured specifically for sale in: NSW after 1977; other states after 1979 TRIUMPH Stag July 1976 onwards Kombi CV engine, 1978–79 Microbus CV engine, 1978–79 Microbus CV engine, 1982-83 Transporter DH watercooled	Corolla i Itthaci	(3K-Cena)				
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CressIda (4M eng) 1977–1980, MX 32, MX 32 CressIda (5M-E eng*) 1980–1983, MX 62 Crown (4M eng) 1977–1980, MS 83, MS 8 Crown (5M-E eng*) 1980–1983, MS 111, MS *Up to engine no. 5M-3429547 Hiace (3Y eng) YH 51/61/71, June 1984 onwards Liteace (4K, 4K-C eng) KM20 Coaster Bus (22R eng) RB20, 1982 onwards Hilux (3Y eng) YN57/60/65 series, Octor 1984 onwards Hilux (1Y, 1Y-C eng) YN55 series Tarago (3Y eng) 2 litre YR21 Land Cruiser FJ62/70/73/75, 1985 onwards Land Cruiser New Model (22R eng) RJ70, 1985 onwards Land Cruiser New Model (22R eng) YU60 FJ40/60 manufactured specifically for sale in: NSW after 1977; other states after 1979 TRIUMPH Stag July 1976 onwards VOLKSWAGEN All VOLKSWAGEN CV engine, 1978-79 Microbus CV engine, 1982-83 Transporter DH watercooled						
CressIda (5M-E eng*) 1980-1983, MX 62 Crown (4M eng) 1977-1980, MS 83, MS 8 Crown (5M-E eng*) 1980-1983, MS 111, MS *Up to engine no. 5M-3429547 YH 51/61/71, June 1984 onwards Liteace (3Y eng) YH 51/61/71, June 1984 onwards Liteace (4K, 4K-C eng) KM20 Coaster Bus (22R eng) RB20, 1982 onwards Hilux (3Y eng) YN57/60/65 series, Octol 1984 onwards Hilux (1Y, 1Y-C eng) YN55 series Tarago (3Y eng) 2 litre YR21 Land Cruiser FJ62/70/73/75, 1985 onwards Land Cruiser New Model (22R eng) RJ70, 1985 onwards Dyna YU60 FJ40/60 manufactured specifically for sale in: NSW after 1977; other states after 1979 TRIUMPH Stag July 1976 onwards Stag July 1976 onwards All VOLKSWAGEN Golf 1977 onwards Kombi CV engine, 1978-79 Microbus CV engine, 1982-83 Transporter DH watercooled						
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Crown (5M-E eng*) 1980-1983, MS 111, MS *Up to engine no. 5M-3429547 Hiace (3Y eng) YH 51/61/71, June 1984 onwards Liteace (4K, 4K-C eng) KM20 Coaster Bus (22R eng) RB20, 1982 onwards Hilux (3Y eng) YN57/60/65 series, Octof 1984 onwards Hilux (1Y, 1Y-C eng) YN55 series Tarago (3Y eng) 2 litre YR21 Land Cruiser FJ62/70/73/75, 1985 onwards Land Cruiser New Model (22R eng) RJ70, 1985 onwards Dyna YU60 FJ40/60 manufactured specifically for sale in: NSW after 1977; other states after 1979 TRIUMPH Stag July 1976 onwards VOLKSWAGEN All VOLKSWAGEN CV engine, 1978-79 Microbus CV engine, 1978-79 Microbus CV engine, 1982-83 Transporter DH watercooled		_				
*Up to engine no. 5M-3429547 Hiace (3Y eng)		-				
Hiace (3Y eng)	•					
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Coaster Bus (22R eng) RB20, 1982 onwards Hillux (3Y eng) YN57/60/65 series, Octof 1984 onwards Hillux (1Y, 1Y-C eng) YN55 series Tarago (3Y eng) 2 litre YR21 Land Cruiser FJ62/70/73/75, 1985 onwards Land Cruiser New Model (22R eng) RJ70, 1985 onwards Dyna YU60 Landcruiser (2F Engine) FJ40/60 manufactured specifically for sale in: NSW after 1977; other states after 1979 TRIUMPH Stag July 1976 onwards Stag July 1976 onwards Kombi CV engine, 1978-79 Microbus CV engine, 1982-83 Transporter DH watercooled	Hiace (3Y eng)					
Hillux (3Y eng)						
Hillux (37 eng) 1984 onwards	Coaster Bus (2	2R eng)				
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Land Cruiser Land Cruiser New Model (22R eng) Dyna YUG0 Landcruiser (2F Engine) TRIUMPH Stag July 1976 onwards All VOLKSWAGEN Golf 1977 onwards Kombi CV engine, 1978-79 Microbus Microbus Microbus FJ62/70/73/75, 1985 RJ70, 1985 onwards FJ40/60 manufactured specifically for sale in: NSW after 1977; other states after 1979 TRIUMPH Stag July 1976 onwards CV engine, 1978-79 Microbus CV engine, 1982-83 Transporter DH watercooled						
Land Cruiser New Model (22R eng) RJ70, 1985 onwards)				
YU60 FJ40/60 manufactured specifically for sale in: NSW after 1977; other states after 1979 TRIUMPH Stag						
Dyna YU60 Landcruiser (2F Engine) FJ40/60 manufactured specifically for sale in: NSW after 1977; other states after 1979 TRIUMPH Stag July 1976 onwards All VOLKSWAGEN Golf 1977 onwards Kombi CV engine, 1978-79 Microbus CV engine, 1982-83 Transporter DH watercooled	Land Cruiser	lew Model	RJ70, 1985 onwards			
Landcruiser (2F Engine) FJ40/60 manufactured specifically for sale in: NSW after 1977; other states after 1979 TRIUMPH Stag July 1976 onwards All VOLKSWAGEN Golf 1977 onwards Kombi CV engine, 1978-79 Microbus CV engine, 1982-83 Transporter DH watercooled	Land Cruiser N					
Specifically for sale in: NSW after 1977; other states after 1979 TRIUMPH	Land Cruiser N (22R eng)					
TRIUMPH Stag July 1976 onwards All VOLKSWAGEN Golf 1977 onwards Kombi CV engine, 1978-79 Microbus CV engine, 1982-83 Transporter DH watercooled	Land Cruiser N (22R eng)					
TRIUMPH Stag July 1976 onwards All VOLKSWAGEN Golf 1977 onwards Kombi CV engine, 1978-79 Microbus CV engine, 1982-83 Transporter DH watercooled	Land Cruiser N (22R eng) Dyna	ee	FJ40/60 manufactured			
Stag	Land Cruiser N (22R eng) Dyna	F Engine)	FJ40/60 manufactured specifically for sale in: NSW after 1977;			
All VOLKSWAGEN Golf 1977 onwards Kombi CV engine, 1978-79 Microbus CV engine, 1982-83 Transporter DH watercooled	Land Cruiser N (22R eng) Dyna	F Engine)	FJ40/60 manufactured specifically for sale in: NSW after 1977;			
VOLKSWAGEN Golf 1977 onwards Kombi CV engine, 1978-79 Microbus CV engine, 1982-83 Transporter DH watercooled	Land Cruiser N (22R eng) Dyna Landcruiser (2 TRIUMPH		FJ40/60 manufactured specifically for sale in: NSW after 1977; other states after 1979			
Golf 1977 onwards Kombi CV engine, 1978-79 Microbus CV engine, 1982-83 Transporter DH watercooled	Land Cruiser N (22R eng) Dyna Landcruiser (2 TRIUMPH	July 1976	FJ40/60 manufactured specifically for sale in: NSW after 1977; other states after 1979			
Kombi CV engine, 1978-79 Microbus CV engine, 1982-83 Transporter DH watercooled	Land Cruiser N (22R eng) Dyna Landcruiser (2 TRIUMPH Stag	July 1976	FJ40/60 manufactured specifically for sale in: NSW after 1977; other states after 1979			
Microbus CV engine, 1982-83 Transporter DH watercooled	Land Cruiser N (22R eng) Dyna Landcruiser (2 TRIUMPH Stag	July 1976	FJ40/60 manufactured specifically for sale in: NSW after 1977; other states after 1979 onwards			
Transporter DH watercooled	Land Cruiser N (22R eng) Dyna Landcruiser (2 TRIUMPH Stag VOLKSWAGEN Golf	July 1976 All 1977 onw	FJ40/60 manufactured specifically for sale in: NSW after 1977; other states after 1979 onwards			
	Land Cruiser N (22R eng) Dyna Landcruiser (2 TRIUMPH Stag VOLKSWAGEN Golf Kombi	July 1976 All 1977 onw CV engine	FJ40/60 manufactured specifically for sale in: NSW after 1977; other states after 1979 onwards			
Passat 1977 onwards	Land Cruiser N (22R eng) Dyna Landcruiser (2 TRIUMPH Stag VOLKSWAGEN Golf Kombi Microbus	July 1976 All 1977 onw CV engine CV engine	FJ40/60 manufactured specifically for sale in: NSW after 1977; other states after 1979 onwards onwards			

Z20, Z22

L20B

L28 E

1.16

A12

A12

A14, C20, A12

1983 L20B (no EAI) series 1

Cabstar

Bluebird

280C, 280ZX

200B

Stanza

120Y

Sunny

Sunny van