



**Australian Government**  
**Department of Health and Ageing**  
**NICNAS**

Priority Existing Chemical  
Assessment Report No. 29

## Lead Compounds In Industrial Surface Coatings & Inks

---

September 2007

National Industrial Chemicals Notification and Assessment Scheme  
GPO Box 58, Sydney NSW 2001, Australia [www.nicnas.gov.au](http://www.nicnas.gov.au)

© Commonwealth of Australia 2007

ISBN 0-9803124-1-8

This work is copyright. Apart from any use as permitted under the Copyright Act 1968, no part may be reproduced by any process without prior written permission from the Commonwealth. Requests and inquiries concerning reproduction and rights should be addressed to the Commonwealth Copyright Administration, Attorney General's Department, Robert Garran Offices, National Circuit, Canberra ACT 2600 or posted at <http://www.ag.gov.au/cca>

# Preface

This assessment was carried out under the National Industrial Chemicals Notification and Assessment Scheme (NICNAS). This Scheme was established by the *Industrial Chemicals (Notification and Assessment) Act 1989* (the Act), which came into operation on 17 July 1990.

The principal aim of NICNAS is to aid in the protection of people at work, the public and the environment from the harmful effects of industrial chemicals.

NICNAS assessments are conducted in conjunction with the Australian Government Department of the Environment and Heritage, which carries out the environmental assessment.

NICNAS has two major programs: the assessment of the health and environmental effects of new industrial chemicals prior to importation or manufacture; and the other focusing on the assessment of chemicals already in use in Australia in response to specific concerns about their health and/or environmental effects.

Chemicals that have been assessed as new or existing chemicals may require a reassessment of the risk of the chemical under the secondary notification provisions of the Act.

This priority existing chemical assessment report has been prepared by the Director of NICNAS in accordance with the Act. Under the Act manufacturers/importers of priority existing chemicals are required to apply for assessment. Applicants for assessment are given a draft copy of the report and 28 days to advise the Director of any errors. Following the correction of any errors, the Director provides applicants and other interested parties with a copy of the draft assessment report for consideration. This is a period of public comment lasting for 28 days during which requests for variation of the report may be made. Where variations are requested the Director's decision concerning each request is made available to each respondent and to other interested parties (for a further period of 28 days). Notices in relation to public comment and decisions made appear in the *Commonwealth Chemical Gazette*.

In accordance with the Act, publication of this report revokes the declaration of these chemicals as priority existing chemicals; therefore manufacturers and importers wishing to introduce this chemical in the future need not apply for assessment. Manufacturers and importers need to be aware of their duty to provide any new information to NICNAS, as required under Section 64 of the Act.

For the purposes of Section 78(1) of the Act, copies of assessment reports for new and existing chemical assessments are freely available from the web ([www.nicnas.gov.au](http://www.nicnas.gov.au)). Hardcopies are available from NICNAS from the following address:

**GPO Box 58, Sydney, NSW 2001, AUSTRALIA**

**Tel: +61 (02) 8577 8800**

**Freecall: 1800 638 528**

**Fax: +61 (02) 8577 8888**

Other information about NICNAS (also available on request) includes:

- NICNAS Service Charter;
- Information sheets on NICNAS Registration;
- Information sheets on Priority Existing Chemical and New Chemicals assessment programs;
- Safety information sheets on chemicals that have been assessed as Priority Existing Chemicals;
- Details for the NICNAS Handbook for Notifiers; and
- Details for the *Commonwealth Chemical Gazette*.

More information on NICNAS can be found at the NICNAS web site:

<http://www.nicnas.gov.au>

Other information on the management of workplace chemicals can be found at the following web site:

<http://www.ascc.gov.au>

# Overview & Recommendations

## Overview

The lead compounds considered in this assessment were declared as Priority Existing Chemicals (PEC) for health risk assessment on 3 January 2006.

The toxicity of lead, particularly to young children, is well known. Use of lead compounds in domestic surface coatings (paints etc) has been eliminated in Australia. The declaration and assessment will identify the essential industrial uses of these compounds in industrial surface coatings and inks and allow regulatory action to be taken as appropriate. The Australian Paint Manufacturers' Federation Inc along with its industry members has embarked on a program to eliminate lead from those industrial surface coatings and inks where it continues to be used. The declaration and assessment is a part of the industry initiative.

The *Uniform Paint Standard* currently prohibits the sale of surface coatings with a lead level greater than 0.1% to consumers. Enforcement is through the states and territories. The planned phase out of lead compounds will affect industrial surface coating and ink products. Data collected in this assessment indicates that inks containing lead compounds are not sold into the consumer market.

## Uses

The lead compounds have a number of uses, such as mining, batteries, chemical manufacture, ceramic and glass manufacture, lubricants and colouring of plastics. This assessment is limited to their use in industrial surface coatings and inks. In these products, lead compounds are mainly used as pigments but a small proportion function as driers in finished formulations. The pigment compounds are used either singularly or in combination to obtain the desired colour and shade. As at 2005, 93% of imports for industrial surface coatings and inks were lead chromates, with lead sulfate and lead molybdate accounting for the bulk of the remainder.

## Exposure

Occupational exposure can occur from accidental spillage during import, transport and warehousing. During manufacture of industrial surface coatings and inks workers can be exposed to the chemicals from loading mixing vats, packing finished product and in the event of accidents. Use of the products can lead to dermal and inhalation exposure to dusts and aerosols. Exposure is likely to be particularly high during surface preparation by sanding, prior to use of the industrial surface coatings, where old coatings containing lead (e.g. automotive panels and bridge repair) are removed. Exposure during sanding is mainly through inhalation of lead containing dust while spray painting could result in high exposure to aerosols and mists. No atmospheric or biological monitoring data were available for occupational use of leaded surface coatings and inks, however EASE modelling predicted the following exposures:

1. Dermal exposure from spraying – 0.1 to 1 mg/cm<sup>2</sup>/day
2. Inhalation exposure from spraying – 0 to 0.1 ppm

### 3. Inhalation exposure to dusts – 2 to 5 ppm

Consumer exposure could arise from renovations of buildings where leaded paints were used or through inappropriate use of industrial coatings in a domestic situation. Hobbyists, such as car restorers or persons who undertake their own panel repairs on vehicles, could also be exposed to paints containing lead.

#### **Health effects**

Lead and inorganic lead compounds are known to have diverse effects on multiple body systems including neurological, gastrointestinal, reproductive and cardiovascular systems. The toxicity of lead compounds is related to the lead cation, however, in lead chromate compounds the toxicity of the chromate moiety is also important. Repeat dose exposure in animals shows widespread effects on the reproductive, neurological, renal and haemopoietic systems.

Inhaled inorganic lead deposits in the alveoli and is almost completely absorbed. In human adults, absorbed lead is stored in bone from which it can be mobilised. Lead can cross the placenta and be excreted in breast milk.

Genotoxicity results for lead are equivocal and no reliable conclusions can be drawn from the data on the genotoxic potential of lead. IARC considered there is inadequate evidence for the carcinogenicity of lead in humans.

Some studies have shown that lead causes a decrease in nerve conduction whilst other work did not find this relationship. However neurobehavioural effects have consistently been found to result from exposure causing lead blood levels of 40 µg/dl and above.

Exposure leading to lead blood levels above 70 µg/dL have resulted in renal tubular damage.

An increased risk of spontaneous abortion, perinatal death and low birth weight has been noted in epidemiological studies. There are no data on female fertility, however reduced fertility has been reported for men with a long duration of exposure to lead. Of most importance is the finding of impaired cognitive development in children exposed to lead during gestation.

#### **Risk**

The risk of adverse health effects associated with use of lead compounds in industrial surface coatings and inks varies according to the activity and resulting exposure.

The lead compounds are all imported and risk during import and transport is low except in the event of an accident. Formulation of surface coatings and inks appears well controlled based on information provided by medium to large companies. Reports to NICNAS indicated that the lead compounds in powder form are handled under local exhaust ventilation and workers wear personal protective equipment (PPE) hence risk of adverse effects is low. This risk is further mitigated when pastes are used as pigments for surface coatings rather than powders. However, only a small group of formulators provided information during the assessment. Risk is also considered to be low during maintenance of equipment.

Risk from use of inks is low and very little lead-based inks are currently in use.

The use of industrial surface coatings presents the greatest risk of adverse effects from exposure to lead. Surface preparation carries a high risk from dusts and application carries a low risk if brush painted and a high risk for spray painting. Appropriate engineering controls, PPE and regulatory requirements can mitigate the risk. However, the likelihood of use of effective control measures in small spray painting workshops is low.

A number of controls are already in place to mitigate exposure to, and health effects arising from, exposure to lead. However, as information on industry compliance with these controls is not available, it is not known whether these are effective.

## Recommendations

This section provides the recommendations arising from the priority existing chemical assessment of the declared inorganic lead compounds used in industrial surface coatings and inks. Recommendations are directed at regulatory bodies and users. The terms “surface coating” and “ink” have the meanings described in section 2 of this report.

### **Proposed phase out of lead compounds in industrial surface coating and inks**

Having regard to the toxicity profile of lead, the associated risks to health, safety and the environment, and the availability of alternatives, the formal variation of the particulars in the Australian Inventory of Chemical Substances (AICS) in accordance with sections 13 and 13A of the *Industrial Chemicals (Notification and Assessment) Act 1989*, is recommended. The following conditions of use appropriately address the health, safety and environmental concerns detailed in this report.

#### **Effective 1 April 2008**

The declared lead compounds **MUST NOT BE**

- a. Imported or manufactured as a chemical entity for use in any ink or;
- b. Imported or manufactured as a component in any ink or;
- c. Imported or manufactured as a chemical entity for use in any industrial surface coating or imported or manufactured as a component of any industrial surface coating.

**EXCEPT FOR USE** in surface coating or in any components of surface coating for the following industrial applications:

- i. Auto refinish car collision repairs;
- ii. Commercial vehicle and component building;
- iii. Commercial vehicle refurbishing and repairs;
- iv. Aviation building (heavy, general and light); and
- v. Aviation refurbishing and repairs.

#### **Effective 1 January 2009**

The declared lead compounds **MUST NOT BE**

- a. Imported or manufactured as a chemical entity for use in any industrial surface coating;

- b. Imported or manufactured as a component of any industrial surface coating.

The proposed annotations will not apply where the declared lead compound is present at concentrations of  $\leq 0.1\%$ .

A notice of the proposed variation of the particulars in the AICS will be published in the *Chemical Gazette* and any statements received in accordance with section 13A of the *Industrial Chemicals (Notification and Assessment) Act 1989* will be considered.

Following the publication of this report, NICNAS will monitor lead compounds that are used in industrial surface coatings and inks or in components of industrial surface coatings and inks apart from those considered in this report. If necessary, a further variation to the particulars in the AICS may be proposed. As noted above, any such proposal will be notified in the *Chemical Gazette* and otherwise in accordance with section 13A of the *Industrial Chemicals (Notification and Assessment) Act 1989*.

## **Recommendations to Regulatory Bodies**

### *The Australian Safety and Compensation Council*

#### **Recommendation 1 –NOHSC National Model Regulations for the Control of Workplace Hazardous Substances.**

It is recommended that the ASCC consider including the declared lead compounds for use in industrial surface coatings and inks and mixtures containing lead compounds, used as or for use in industrial surface coatings and inks, in Schedule 2 (or its successor) of the NOHSC *National Model Regulations for the Control of Workplace Hazardous Substances* effective 1 January 2010.

Consistent with the NOHSC National Model Regulations employers should ensure that these substances are not used for the purpose specified in the schedule.

### *National Drugs and Poisons Schedule Committee*

#### **Recommendation 2 – Poison scheduling**

It is recommended that the National Drugs and Poisons Schedule Committee (NDPSC) consider:

- a) Including lead compounds for use in inks in Appendix C of the SUSDP.
- b) Reviewing the Uniform Paint Standard of the SUSDP in relation to the declared lead compounds for use in surface coatings.

## **Recommendation to Industry**

### **Recommendation 3– Dissemination of information**

Relevant industry associations, importers and manufacturers of the declared lead compounds used in industrial surface coatings and inks should disseminate information on conditions of use of these compounds down the supply chain to formulators and end users to raise awareness of the regulatory actions on these chemicals.

Given that no biological monitoring data were provided for this assessment by industry, it is recommended that industry disseminate information on the regulatory requirements for lead processes.



## **Recommendations to State and Territory Authorities**

### **Recommendation 4 – Compliance**

It is recommended that states and territories monitor compliance with existing regulatory requirements.

# Acronyms and Abbreviations

ACGIH	American Conference of Governmental Industrial Hygienists
ADG Code	Australian Dangerous Goods Code
AICS	Australian Inventory of Chemical Substances (NICNAS)
AS	Australian Standard
ASCC	Australian Safety and Compensation Council
ATSDR	Agency for Toxic Substances and Disease Registry (United States)
BUN	blood urea nitrogen
bw	bodyweight
CAS	Chemical Abstracts Service
conc.	concentration
d	day
dL	decilitre
EASE	estimation and assessment of substance exposure
EC	European Community, or European Commission
EU	European Union
h	hour
HSIS	Hazardous Substances Information System
id	intradermal

im	intramuscular
ip	intraperitoneal
iv	intravenous
IC(NA) Act	<i>Industrial Chemicals (Notification and Assessment) Act 1989</i> (Cwlth)
IUPAC	International Union of Pure and Applied Chemistry
LC50	median lethal concentration
LD50	median lethal dose
LEV	Local exhaust ventilation
LL50	lethal loading rate resulting in 50% mortality
LLR	lethal loading rate
LOAEL	lowest-observed-adverse-effect level
LOEL	lowest-observed-effect level
mg/kg bw/d	milligram per kilogram bodyweight per day
MOE	margin of exposure
MSDS	material safety data sheet
NICNAS	National Industrial Chemicals Notification and Assessment Scheme
NIOSH	National Institute of Occupational Safety and Health
NOEC	no-observed-effect concentration
NOAEL	no-observed-adverse-effect level
NOEL	no-observed-effect level
NOHSC	National Occupational Health and Safety Commission

OECD	Organisation for Economic Cooperation and Development
pKa	dissociation constant
ppb	parts per billion
PPE	personal protective equipment
ppm	parts per million
sc	subcutaneous
SCE	sister chromatid exchange
SUSDP	Standard for the Uniform Scheduling of Drugs and Poisons
TLV	Threshold Limit Value (ACGIH)
TSCA	Toxic Substances Control Act (US)
TWA	Time-weighted average (NOHSC)
v/v	volume per volume
w/v	weight per volume
w/w	weight per weight
WHO	World Health Organization

# Glossary

Adverse effect	Change in the morphology, physiology, growth, development, reproduction, or life span of an organism, system or (sub) population that results in an impairment of functional capacity, an impairment of the capacity to compensate for additional stress, or an increase in susceptibility to other influences.
Assessment	Evaluation of appraisal of an analysis of facts and the inference of possible consequences concerning a particular object or process.
Assessment endpoint	Quantitative/qualitative expression of a specific factor with which a risk may be associated as determined through an appropriate risk assessment.
Concentration	Amount of a material or agent dissolved or contained in unit quantity in a given medium or system.
Dose	Total amount of an agent administered to, taken up or absorbed by an organism, system or (sub) population.
Dose rate	Dose per unit time
Dose-related effect	Any effect to an organism, system or (sub) population as a result of the quantity of an agent administered to, taken up or absorbed by that organism, system or (sub) population.
Dose-response relationship	Relationship between the amount of an agent administered to, taken up or absorbed by an organism, system or (sub) population and the change developed in that organism, system or (sub) population in reaction to the agent. Related Terms: <i>Dose-Effect Relationship, Effect Assessment, Concentration-Effect Relationship.</i>
Effect	Change in the state or dynamics of an organism, system or (sub) population caused by the exposure to an agent.
Exposure	Concentration or amount of a particular agent that reaches a target organism, system or (sub) population in a specific frequency for a defined duration.
Exposure assessment	Evaluation of the exposure of an organism, system or (sub) population to an agent (and its derivatives). Exposure Assessment is the third step in the process of Risk Assessment.
Exposure period	The time of continuous contact between an agent and a target.
Exposure route	The way an agent enters a target after contact ( <i>e.g.</i> , by ingestion, inhalation, or dermal absorption).

Fate	Pattern of distribution of an agent, its derivatives or metabolites in an organism, system, compartment or (sub) population of concern as a result of transport, partitioning, transformation or degradation.
Hazard	Inherent property of an agent or situation having the potential to cause adverse effects when an organism, system or (sub) population is exposed to that agent.
Hazard assessment	A process designed to determine the possible adverse effects of an agent or situation to which an organism, system or (sub) population could be exposed. The process includes hazard identification and hazard characterization. The process focuses on the hazard in contrast to risk assessment where exposure assessment is a distinct additional step.
Hazard characterization	<p>The qualitative and, wherever possible, quantitative description of the inherent properties of an agent or situation having the potential to cause adverse effects. This should, where possible, include a dose-response assessment and its attendant uncertainties.</p> <p>Hazard Characterisation is the second stage in the process of Hazard Assessment, and the second step in Risk Assessment.</p> <p>Related terms: <i>Dose-Effect Relationship, Effect Assessment, Dose-Response Relationship, Concentration -Effect Relationship.</i></p>
Hazard identification	<p>The identification of the type and nature of adverse effects that an agent has inherent capacity to cause in an organism, system or (sub) population.</p> <p>Hazard identification is the first stage in hazard assessment and the first step in process of Risk Assessment</p>
Risk assessment	<p>A process intended to calculate or estimate the risk to a given target organism, system or (sub) population, including the identification of attendant uncertainties, following exposure to a particular agent, taking into account the inherent characteristics of the agent of concern as well as the characteristics of the specific target system.</p> <p>The Risk Assessment process includes four steps: hazard identification, hazard characterization (related term: dose-response assessment), exposure assessment, and risk characterization. It is the first component in a risk analysis process.</p>
Risk characterization	<p>The qualitative and, wherever possible, quantitative determination, including attendant uncertainties, of the probability of occurrence of known and potential adverse effects of an agent in a given organism, system or population, under defined exposure conditions.</p> <p>Risk Characterization is the fourth step in the Risk Assessment process.</p>
Risk management	Decision-making process involving considerations of political, social, economic, and technical factors with relevant risk assessment information relating to a hazard so as to develop, analyse, and compare regulatory and non-regulatory options and to select and implement appropriate regulatory response to that

hazard.

Risk management comprises three elements: risk evaluation; emission and exposure control; risk monitoring.

Toxicity

Inherent property of an agent to cause an adverse biological effect.

Uptake (absorption)

The process by which an agent crosses an absorption barrier.

# Contents

PREFACE	III
OVERVIEW & RECOMMENDATIONS	V
ACRONYMS AND ABBREVIATIONS	X
GLOSSARY	XIII
CONTENTS	XVI
1. INTRODUCTION	1
1.1 Declaration	1
1.2 Objectives	2
1.3 Sources of information	2
1.4 Peer review	2
2. BACKGROUND	3
2.1 International perspective	5
2.1.1 United States of America	5
2.1.2 European Union	7
2.1.3 United Kingdom	7
2.1.4 Voluntary industry controls overseas	8
2.2 Australian perspective	8
2.3 Assessments by other national or international bodies	9
3. APPLICANTS	10
4. CHEMICAL IDENTITY, COMPOSITION AND PHYSICAL AND CHEMICAL PROPERTIES	12
5. USE, MANUFACTURE AND IMPORTATION	14
5.1 Manufacture and importation	14
5.2 Uses in Australia	17
5.3 Formulation of products	18
5.3.1 Industrial surface coatings	18
5.3.2 Inks	20
6. EXPOSURE	21



6.1	Occupational exposure	21
6.1.1	Importation and storage	21
6.1.2	Formulation of industrial paints	21
6.1.3	Formulation of powder coatings	23
6.1.4	Formulation of inks	23
6.1.5	Use of industrial surface coating products	23
6.1.6	Use of inks	26
6.1.7	Occupational monitoring and exposure	27
6.1.8	Biological monitoring	27
6.2	Consumer exposure	30
6.2.1	Biological monitoring of the general population	30
7.	KINETICS AND METABOLISM	34
7.1	Absorption	34
7.1.1	Inhalation	35
7.1.2	Dermal	35
7.1.3	Oral	35
7.2	Distribution	35
7.3	Metabolism	36
7.4	Excretion	36
8.	HEALTH EFFECTS	37
8.1	Health effects of lead	38
8.1.1	Acute toxicity	38
8.1.2	Irritation	39
8.1.3	Sensitisation	39
8.1.4	Repeat dose toxicity	39
8.1.5	Mutagenicity	56
8.1.6	Carcinogenicity	61
8.1.7	Fertility	63
8.1.8	Developmental toxicity	65
8.2	Health effects of specific lead compounds	67
8.2.1	Lead monoxide	67
8.2.2	Lead sulfate	68
8.2.3	Lead molybdate	68
8.2.4	Lead octanoate	68
8.2.5	Lead 2-ethylhexanoate	68
8.2.6	Lead oxide	69
8.2.7	Lead nitrate	69
8.2.8	Lead naphthenate	71

8.2.9	Lead peroxide	71
8.2.10	Lead carbonate (white lead)	71
8.2.11	Lead chrome 1244	72
8.2.12	Lead chromate	72
8.2.13	Lead chromate oxide	73
8.2.14	Lead chromate molybdate sulfate red	73
8.2.15	Lead sulfo-chromate	74
9.	RISK CHARACTERISATION	75
9.1	Occupational health risk	75
9.1.1	Occupational exposure	75
9.1.2	Critical health effects	76
9.1.3	Risk estimate(s)	76
9.2	Public health risk	78
10.	RISK MANAGEMENT	79
10.1	Assessment of current control measures	79
10.1.1	Elimination/substitution	79
10.2	Regulatory controls in Australia for industrial uses	81
10.2.1	National framework	81
10.2.2	State and Territory workplace legislation around Australia	87
10.3	Australian Standards for industrial settings	90
10.4	Voluntary industry controls for industrial settings	91
10.3	Regulatory controls in Australia for consumer goods	92
10.3.1	Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP)	92
10.3.2	Australian Standards for consumer settings	94
10.3.3	Consumer legislation around Australia	95
11.	DISCUSSION AND CONCLUSIONS	99
11.1	Importation and use	99
11.2	Health hazards	99
11.3	Occupational health and risk	100
APPENDIX A CHEMICAL IDENTITY, COMPOSITION AND PHYSICAL AND CHEMICAL PROPERTIES		101
A1.1	Lead monoxide	101
A1.2	Lead chromate	102
A1.3	Lead sulfate	103
A1.5	Lead sulfo-chromate	104

A1.6	Lead chromate molybdate sulfate red	105
A1.7	Lead chromate oxide	106
A1.8	Lead octanoate	107
A1.9	Lead 2-ethylhexanoate	107
A1.10	Lead oxide	108
A1.11	Lead nitrate	109
A1.12	Lead naphthenate	109
A1.13	Lead oxide	110
A1.14	Lead carbonate	111
A1.15	Lead chrome 1244	112
REFERENCES		113



# 1. Introduction

## 1.1 Declaration

The lead compounds listed below were declared Priority Existing Chemicals (PEC) for health risk assessment under the *Industrial Chemicals (Notification and Assessment) Act 1989* (the Act) on 3 January 2006.

Chemical Name	CAS Number
Lead monoxide	1317-36-8
Lead chromate	7758-97-6
Lead sulfate	7446-14-2
Lead molybdate	10190-55-3
Lead sulfo-chromate	1344-37-2
Lead chromate molybdate sulfate red	12656-85-8
Lead chromate oxide	18454-12-1
Lead octanoate	7319-86-0
Lead 2-ethylhexanoate	301-08-6
Lead oxide	1314-41-6
Lead nitrate	10099-74-8
Lead naphthenate	61790-14-5
Lead peroxide	1309-60-0
Lead carbonate (white lead)	1319-46-6
Lead chrome 1244	unknown

The lead compounds used in industrial surface coatings and inks were identified through a joint Australian Paint Manufacturers' Federation Inc (APMF) and NICNAS survey of the surface coatings and printing inks industries. The chemicals were declared as PECs because of the toxicity of lead particularly to young children. Use of lead compounds in domestic surface coatings (paints etc) has been eliminated in Australia. The declaration and assessment will identify the essential uses of these compounds in industrial surface coatings and inks and allow regulatory action to be taken as appropriate. The Australian Paint Manufacturers Federation Inc along with its industry members has embarked on a program to eliminate lead from those industrial surface coatings and inks where it continues to be used. The declaration and assessment is intended to provide the basis for a regulatory framework to supplement the industry initiative.

The assessment is limited to the use of lead compounds in industrial surface coatings and inks (see Chapter 2 for a discussion of the terms surface coatings and inks as used in this report).

## 1.2 Objectives

The objectives of this assessment are to:

- identify the extent and use of the declared lead compounds in industrial surface coatings and inks;
- characterise the human health hazards of lead;
- determine the current and potential occupational and public exposure to lead from the use of leaded industrial surface coatings and inks;
- determine the risk of adverse effects to workers and the public resulting from exposure to lead compounds in industrial surface coatings and inks; and
- make recommendations for minimizing occupational and public health risks, and appropriate hazard communication measures, where applicable.

## 1.3 Sources of information

In accordance with the Act, those who wish to manufacture and/or import any of the declared lead compounds for use in industrial surface coatings and inks were required to apply for assessment and supply relevant data. Applications were received from importers of the declared lead compounds and relevant products containing declared lead compounds as well as local formulators of industrial surface coatings and inks.

The hazards of lead are well documented, and, to enhance efficiency and avoid duplication of effort, this assessment utilises international reviews from reputable organisations. These reports were used mainly for the health hazard section and include reviews by the World Health Organisation, the International Agency for Research on Cancer and the United States Agency for Toxic Substances and Disease Registry (draft report only).

Information on use in Australia and potential exposure were provided by applicants and notifiers to this assessment.

## 1.4 Peer review

During all stages of preparation, the report has been subject to internal peer review by NICNAS.

## 2. Background

Lead occurs naturally in the earth's crust, in soils, in seawater, ground water and surface water, in the atmosphere and in plants.

Lead is found in a variety of minerals the most important of which are galena (PbS), cerrusite (PbCO<sub>3</sub>) and anglesite (PbSO<sub>4</sub>). Galena is by far the most important source of primary lead, occurring mostly in deposits also associated with other minerals, particularly those containing zinc.

The definitions of terms such as paint, varnish, lacquer are not uniform throughout the world and literature sources differ in the distinction they make between these products. The term "paint" is defined in the Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP, 2006) as:

*".....including any substance used or intended to be used for application as a colouring or protective coating to any surface but does not include graphic material"*

The Macquarie Dictionary defines "paint" as:

*"noun 1. a substance composed of solid colouring matter intimately mixed with a liquid vehicle or medium, and applied as a coating.*

*2. the dried surface pigment. [art]*

*3. the solid colouring matter alone; a pigment [art] ....."*

And defines "ink" as:

*"noun 1. a fluid or viscous substance used for writing or printing."*

And defines "coating" as:

*"noun 1. layer of any substance spread over a surface....."*(Macquarie, 2005)

In Europe the following definitions have been used

*" a paint is a pigmented material that, when applied as a liquid to a surface, forms, after a time, a dry adherent film;*

*a lacquer is a coating which dries by evaporation of the solvent – a thin-bodied, quick drying, coating material which form a hard protective film;*

*a varnish is a transparent coating material based essentially on resins and/or drying oil and solvent"* (EUBEES, 2004)

To encompass the coating applications under review, this report will use the term surface coatings, which are defined as including:

*Paints, which without limiting the ordinary meaning are defined as a product in liquid form which, when applied to a surface, forms a dry film having protective, decorative or other specific technical properties, and includes lacquers and varnishes but excludes gelcoats and vitreous coatings (see below).*

*Powder coatings, which are defined as a solid thermosetting coating, generally mixtures of resins, pigments, curing agents and additives, ground into fine particles, applied as a fine powder, adhered to the surface by heating to a temperature above the melting point of the binder, forming a continuous film.*

But excluding:

*Vitreous coatings, defined as an inorganic coating applied to a metallic, ceramic or glass substrate which has been fired at elevated temperatures, to form a glassy finish which is fused to the substrate.*

*Gel Coates, defined as a thin outer layer of resin, sometimes with pigment, applied to a reinforced plastic moulding.*

For the purpose of this report, inks are defined as:

*A non-vitreous, liquid or viscous substance used for writing or printing.*

Surface coatings are used for a number of reasons including:

- protection of the coated substrate from corrosion, UV degradation,
- providing decorative, aesthetic and other surface effects.

Paints and varnishes are composed of 5 main categories of ingredients, viz binder, solvent, pigment, colourants, fillers and additives (EUBEES, 2004).

Industrial thermosetting powder coatings are generally mixtures of resins, pigments, curing agents and additives, ground into fine particles (CEPE, 2005).

A surface coating builds a continuous, adhesive layer (known as a film) on a surface. Drying of a surface coating is called film formation and can be achieved by a number of mechanisms:

- evaporation of solvents from solutions or dispersions;
- solidification of melted substances;
- chemical reactions.

The first two instances involve physical drying and no chemical reaction between the compounds takes place. After evaporation of the solvents (water or organic solvents), the binder, which consists of high molecular weight compounds, forms the film. The third instance involves chemical reaction between components, such as polymerisation.



Powder coatings do not contain a solvent. After application of the powder coating to the substrate, the coating is bonded to the surface by heating in an oven to a temperature above the melting point of the binder (CEPE, 2005).

## **2.1 International perspective**

International regulatory regimes provide useful examples of restrictions and prohibitions on the use of lead compounds.

The first European and international legal recognition of the toxicity of lead paints was introduced in 1921 by the International Labour Organisation (ILO), concerning the implementation of the *ILO Convention 13 - Convention Concerning the Use of White Lead in Painting*. This convention came into full force in 1923 although not ratified by all countries.

The following discussion relates to overseas regulation of lead compounds in surface coatings and inks both for consumer and industrial use and does not include other uses of lead compounds such as motor fuels.

### **2.1.1 United States of America**

The US has a strict regulatory regime in place controlling and prohibiting the use of lead-based paints and surface coatings. Residential, public and consumer uses of lead-based paints are banned, and industrial use requires strict warning labels. The US has also instituted legislation requiring full disclosure of the presence of lead-based paint on the sale and purchase of land, plus regulations relating to the renovation and repair of homes and the disposal of lead paint debris. This is complimented by training programs for contractors and of information pamphlets and guidelines for the community and industry.

Under the US *Federal Hazardous Substances Act* section 1261(f)(1), any household products that expose children to hazardous quantities of lead under reasonably foreseeable conditions of handling or use are ‘hazardous substances.’ This general provision includes lead-based paints.

#### **Restrictions and bans on the sale of lead-containing paint**

The primary piece of US federal legislation placing restrictions on the use of lead-based paint is the *Ban of Lead-Containing Paint and Products Bearing Lead-Containing Paint* [Part 1303] from the *Consumer Product Safety Act* (Code of Federal Regulation, Title 16, Volume 2). This instrument bans the commercial sale of paints and surface coatings to consumers that contain lead in excess of 0.06 percent of the total non-volatile content of the paint or the weight of the dried paint film. These paints are referred to as ‘lead-containing paints’ and are listed as banned hazardous products under section 8 and section 9 of the *Consumer Product Safety Act*. The regulation also bans two categories of consumer products; toys intended for use by children that contain or bear ‘lead-containing paint’ and furniture articles for consumers that bear ‘lead-containing paint’. This ban applies not only to products sold directly to consumers, but also products that are not sold to consumers but that could be used by the public (e.g. playground equipment). Hence, the ban applies to paints used in residences,

schools, hospitals, parks, playgrounds and public buildings. The ban applies to all products manufactured after February 27, 1978.

### **Exemptions: with warnings**

The Regulation establishes categories of 'lead-containing paints' that are exempt from the above ban, but that still require labels warning users of the danger of lead and directing that the products not be applied to toys, children's articles, furniture, exterior surfaces to residences, etc. The products that are so exempt are:

1. Agricultural and industrial equipment refinish coatings;
2. Industrial building and equipment maintenance coatings;
3. Graphic art coatings;
4. Touchup coatings for agricultural, lawn and garden equipment; and
5. Coatings for use on radio-controlled model aircraft.

### **Exemptions: without warnings**

The Regulation stipulates that there are 3 product types exempt from the ban and exempt from labeling requirements. These are:

1. Mirrors which are part of furniture articles;
2. Artists' paints and related materials; and
3. Metal furniture articles bearing factory-applied lead coatings.

The effect of the above regulation is that lead-based paints and coatings are now banned (subject to the above exemptions) for residential use and in places and equipment used by the public. Industrial uses remain permissible provided there is a warning label.

### **Renovation and remodelling**

Section 402(c)(3) of *Toxic Substances Control Act* directs the EPA to formulate guidelines to ensure that individuals engaged in renovation and remodelling activities that create lead-based paint hazards are properly trained; that training programs are accredited; and that contractors engaged in such activities are certified. The US EPA has proposed new rules that introduce lead training, certification and safe work practice requirements for contractors involved in renovation, repair and painting activities in pre-1978 houses. Certified and appropriately trained contractors must also provide lead information to residents before they commence renovation of a pre-1978 home.

This information is part of the Pre-Renovation Education (PRE) program and includes the giving of pamphlets and oral information to homeowners.

### **Disposal of lead paint waste**

The regulation entitled *Management and Disposal of Lead-based Paint Debris* (Federal Register 63 (243)) places duties on individuals, contractors and firms for

the identification, management, storage and safe disposal of lead-based paint debris.

### 2.1.2 European Union

The European Parliament has issued a directive relating to dangerous substances and preparations (76/769/EEC). This directive deals specifically with lead-based paints. The 8<sup>th</sup> amendment to this directive proposed a complete ban on the use of lead carbonate and sulphate pigments (white lead) in paints. However, in September 1989 an exception to this rule was inserted. Lead carbonates and sulphates may be used in paints for the restoration and maintenance of works of art and historic buildings. In all other respects the directive implements ILO Convention 13.

EEC directives are binding on all countries within the European Union in relation to the handling and packaging of lead-based paint products.

The Directive Concerning the Approximation of the Laws, Regulations and Administrative Provisions of the Member States Relating to the Classification, Packaging and Labelling of Dangerous Substances (1999/45/EC) enacts general provisions relating to the labelling and packaging of dangerous substances. Annex V contains specific provisions relating to lead-based paint. Any paint or varnish which contains lead in quantities exceeding 0.15% of the total weight must display warning labels stating; "contains lead: should not be used on surfaces liable to be chewed or sucked by children".

'White lead' paints are effectively prohibited in the European Union. All other lead-based paints and varnishes must satisfy strict safety, packaging, transportation and labelling requirements.

### 2.1.3 United Kingdom

The UK has banned the general sale of lead paint that consists of lead carbonate and sulphate (sic) pigments (white lead). This has been instituted through the *Environmental Protection (Controls on Injurious Substances) Regulations 1992 (Statutory Instrument 1992/31)*. The Act stipulates in Section 3;

*"...no person shall-*

*supply by way of sale for any purpose, or*

*use in connection with any trade or business in manufacturing process,*

*lead carbonate or lead sulphate which is intended for use as paint, or any substance so intended of which lead carbonate or lead sulphate forms a constituent."*

However, the Act makes a notable exception to this rule for the restoration or maintenance of historic and heritage buildings. The exception is only in very strict terms involving a completed declaration. The paint cannot be supplied less than three weeks after a declaration has been given to the relevant authority. Other than this exception, lead-based paints containing lead sulphate or carbonate

cannot be supplied in the UK for any purpose, whether it be residential, commercial or industrial.

The *Environmental Protection (Duty of Care) Regulation 1992* places duties on professional painters and contractors to dispose of lead wastes in a responsible way so as to ensure that there is no harmful deposit or escape of the waste from their control. This is a general regulation that applies to all wastes.

No other legislative instruments appear to place any direct restriction or prohibition on the use of lead-based paints or coatings in the UK. Other legislation relating to lead in the UK concerns Occupational Health and Safety issues. European Union directives also apply to the UK concerning the labelling, packaging and transportation of lead-based paints (as discussed below).

#### **2.1.4 Voluntary industry controls overseas**

A number of overseas countries have surface coating and ink industry representative bodies similar to Australia's APMF. The International Paint and Printing Ink Council (IPPIC) is comprised of paint and printing ink associations representing Australia, Brazil, Canada, Europe, Japan, Mexico and the United States.

##### **Coatings Care<sup>®</sup>**

The IPPIC has developed a product stewardship program known as Coatings Care<sup>®</sup>. This is a global program created by the surface coatings industry designed to assist companies manage their health, safety, and environmental responsibilities. Its aim is to protect the environment and worker and community health and safety. The industries in Brazil, Canada, France, Japan, Mexico, Netherlands, Peru, Phillipines, the United Kingdom, the United States of America and Australia have adopted the program.

Coatings Care<sup>®</sup> is not specifically aimed at leaded paints but is a general industry program.

#### **2.2 Australian perspective**

A joint survey by NICNAS and the Australian Paint Manufacturers' Federation Inc (APMF) found that the 15 lead compounds declared for assessment are currently used, or have in the past been used as pigments or driers in industrial surface coatings and inks. Information received from industry indicated that no lead-based compounds used in surface coatings and inks are manufactured in Australia.

A number of regulatory initiatives are in place to control the exposure of workers, the public and the environment to lead. Paint containing more than 0.1% lead in its non-volatile component is prohibited for sale for domestic use under Appendix I (Uniform Paint Standard) of the Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP). Workplace controls are in place throughout Australia to limit atmospheric levels of lead and also mandate maximum blood levels of lead for both male and female workers. These are based on the National Standard for the Control of Inorganic Lead at Work (NOHSC, 1994). Certain lead chromate

compounds specifically are listed in the Hazardous Substances Information System (HSIS) database and the remaining inorganic lead compounds are captured under a general class classification.

### **2.3 Assessments by other national or international bodies**

International reviews of the health and/or environmental effects and risk assessments for lead and lead compounds have been carried out by the International Agency for Research on Cancer, (IARC 1972, 1980, 1987); the WHO's International Program on Chemical Safety (IPCS 1977, 1989) and the US Agency for Toxic Substances and Disease Registry (ATSDR draft September 2005).

### 3. Applicants

Following the declaration of lead compounds used in industrial surface coatings and inks as priority existing chemicals, 23 companies applied for assessment. Companies supplied information on the properties, import quantities and uses of the chemical. In accordance with the *Industrial Chemicals (Notification and Assessment) Act 1989*, NICNAS provided the applicants with a draft copy of the report for comments during the corrections/variation phase of the assessment. The applicants were:

Akzo Nobel Car Refinishers  
Unit 1, 269 Williamstown Road  
PORT MELBOURNE VIC 3207

Ameron (NZ) Limited  
1-5 Fourth Avenue  
BURLEIGH HEADS QLD 4220

BASF Australia Limited  
500 Princes Highway  
NOBLE PARK VIC 3174

Ciba Specialty Chemicals Pty Limited  
235 Settlement Road  
THOMASTOWN VIC 3074

Clariant (Australia) Pty Limited  
675 Warrigal Road  
CHADSTONE VIC 3148

Colorlinx Australia Pty Limited  
2 Verey Crt  
DANDENONG VIC 3175

Degussa Coatings and Colorants Pty  
Limited  
30 Commercial Drive  
DANDENONG SOUTH VIC 3175

DIC International (Australia) Pty  
Limited  
30 – 32 Kilkenny Court  
DANDENONG SOUTH VIC 3175

The Duha Group Pty Limited  
137 – 139 McEwan Rd  
WEST HEIDELBERG VIC 3081

DuPont Australia Limited  
49 – 53 Newton Road  
WETHERILL PARK NSW 2164

Ferro Corporation (Australia) Pty  
Limited  
105 – 115 Cochranes Road  
MOORABBIN VIC 3189

Flores Nominees  
74 Bayfield Road East  
BAYSWATER VIC 3153

Fujifilm Sericol Australia Pty Limited  
4 Coronation Avenue  
KINGS PARK NSW 2148

Hempel Australia Pty Limited  
12 Fitzgerald Road  
LAVERTON NORTH VIC 3026

Multichem Pty Limited  
Suite 5, 400 High Street  
KEW VIC 3101

Nuplex Industries (Australia) Pty  
Limited  
8 Abbott Road  
SEVEN HILLS NSW 2147

PPG Industries Australia Pty Limited  
McNaughton Road  
CLAYTON VIC 3168

Protec Pty Limited  
97 – 105 Bedford Street  
GILLMAN SA 5013

Quantum Chemicals  
70 Quantum Close, Quantum Industrial  
Park  
DANDENONG SOUTH VIC 3175

Redox Pty Limited  
2 Swettenham Road  
MINTO NSW 2566

Signet Pty Limited  
56 Ingleston Road  
WAKERLEY QLD 4153

Tradechem Pty Limited  
5 Farleigh Street  
ASHFIELD NSW 2131

Wynn's Australia Pty Limited  
Unit C5 – C7, 391 Park Road  
REGENTS PARK NSW 2143

## 4. Chemical Identity, Composition and Physical and Chemical Properties

Lead is a naturally occurring element normally existing in three oxidation states, Pb(0), Pb(II) and Pb(IV). In the environment lead generally exists in the Pb(II) form. Inorganic Pb(IV) compounds do not occur in nature but are formed under extreme oxidative conditions. Some organolead (II) compounds occur in nature but generally organolead compounds exist as Pb(IV). Rarely does metallic lead, Pb(0) exist in the environment. Lead metal has high resistance to corrosion and a low melting point.

Lead is a member of Group 14 of the Periodic Table. It is a main group element with four electrons in its outer, or valence, shell. Some inorganic lead compounds can occur in two or more crystalline forms having different properties such as colour and solubilities. In addition, complex mixed salts, such as white lead,  $2\text{PbCO}_3 \cdot \text{Pb(OH)}_2$ , are readily formed.

Exposure of a clean lead surface to dry air does not result in any chemical reaction, however in moist air the lead surface becomes coated with a film of lead monoxide, PbO. This compound can combine with carbon dioxide from the atmosphere to produce a lead carbonate.

The inorganic compounds of tetravalent lead are relatively unstable; e.g., in the presence of water they hydrolyze to give lead dioxide.

Except for leaded zinc oxide, for which high grade lead ore is used, all lead-containing compounds are produced using the metallic lead extracted after refining lead ore. Most lead compounds are prepared directly or indirectly from lead monoxide, commonly known as litharge, by one or more of three methods:

1. reaction between a slurry of litharge, or a similar lead compound such as the hydroxide or carbonate, and the desired acid, or solution thereof in the case of an organic acid, or soluble salt of that acid;
2. reaction between the solution of a lead salt and a solution of the desired acid, or soluble salt of the acid. These reactions are facilitated by the fact that the desired lead compound usually is relatively insoluble, thus forming as a precipitate; and
3. fusion or calcination of litharge and the desired oxide. (Kirk Othmer, 2006).

Details of chemical names, Chemical Abstract Service (CAS) Number, synonyms, molecular formulae and molecular weight for each of the declared lead compounds are given in Appendix A.



The physical and chemical properties for the chemicals are also detailed in Appendix A. Unless otherwise specified, these properties are provided for the pure chemicals.

# 5. Use, Manufacture and Importation

## 5.1 Manufacture and importation

No company reported manufacturing lead compounds in Australia for use in surface coatings and inks. Lead compounds for industrial purposes are imported as either raw chemical for use in formulations or as part of pigment products.

The compounds are imported for a number of different uses and are not restricted to industrial surface coatings and inks. This assessment focuses on industrial surface coatings and inks and data on introduced quantities does not include quantities imported for uses other than industrial surface coatings and inks. Uses of the compounds for other than industrial surface coatings and inks however is shown where this information was provided by applicants and notifiers.

Import of lead compounds in powder form is in 25 kg multi-layered paper bags, tinting bases and pastes are imported in 4 L or 20 L drums and powder coatings are imported in boxes of 20 kg or 2 kg.

Pigments, powder coats and bases/pastes contain either one lead salt or a mixture of up to three different lead salts to achieve the desired colour. The lead salt(s) comprise up to 100% in pigments. It should be noted that the concentration of lead (as metal) in any pigment, powder coat or base/paste will be less than the concentration of the lead compound.

Import quantities of the declared lead compounds for use in industrial surface coatings and inks during calendar years 2003 – 2005 are shown in Table 5.1. Tables 5.2 and 5.3 show quantities imported during the same time period, broken down into amounts used in automotive paints and amounts used in inks, respectively. This information was provided by applicants in response to the declaration notice.

Primary activities of firms in the paint/surface coatings industry are manufacture or supply of (IBIS world, 2005):

- Caulking compound
- Filler or putty
- Lacquer
- Architectural & decorative paints, enamels & clears, industrial paints, enamels & clears (except bituminous)
- Paint or varnish remover, prepared,
- Paint tinting colour, prepared,
- Primer or undercoat, paint,

- Rubbing compound
- Stain
- Wood stain (packed for sale)

**Table 5.1 - Quantities of lead compounds introduced into Australia for industrial surface coatings and inks, 2003 to 2005 (tonnes)**

<b>Chemical Name</b>	<b>CAS Number</b>	<b>2003</b>	<b>2004</b>	<b>2005</b>
Lead monoxide	1317-36-8	Nil	Nil	Nil
Lead chromate	7758-97-6	32	175	168
Lead sulfate	7446-14-2	2	15	15
Lead molybdate	10190-55-3	2	4	7
Lead sulfo-chromate	1344-37-2	60	90	76
Lead chromate molybdate sulfate red	12656-85-8	29	30	50
Lead chromate oxide	18454-12-1	Nil	Nil	Nil
Lead octanoate	7319-86-0	Nil	Nil	Nil
Lead 2-ethylhexanoate	301-08-6	1	0.02	Nil
Lead oxide	1314-41-6	Nil	Nil	Nil
Lead nitrate	10099-74-8	Nil	Nil	Nil
Lead naphthenate	61790-14-5	Nil	Nil	Nil
Lead peroxide	1309-60-0	Nil	Nil	Nil
Lead carbonate (white lead)	1319-46-6	Nil	Nil	Nil
Lead chrome 1244	unknown	Nil	Nil	Nil
<b>Total Lead Salts (Tonnes)</b>		<b>126</b>	<b>314</b>	<b>316</b>

**Table 5.2 - Quantities of lead compounds introduced into Australia for automotive coatings 2003 to 2005 (tonnes)**

<b>Chemical Name</b>	<b>CAS Number</b>	<b>2003</b>	<b>2004</b>	<b>2005</b>
Lead monoxide	1317-36-8	Nil	Nil	Nil
Lead chromate	7758-97-6	0.2	0.3	0.1
Lead sulfate	7446-14-2	Nil	Nil	Nil
Lead molybdate	10190-55-3	Nil	Nil	Nil
Lead sulfo-chromate	1344-37-2	23	19	137
Lead chromate molybdate sulfate red	12656-85-8	24	24	22
Lead chromate oxide	18454-12-1	Nil	Nil	Nil
Lead octanoate	7319-86-0	Nil	Nil	Nil
Lead 2-ethylhexanoate	301-08-6	Nil	Nil	Nil
Lead oxide	1314-41-6	Nil	Nil	Nil
Lead nitrate	10099-74-8	Nil	Nil	Nil
Lead naphthenate	61790-14-5	Nil	Nil	Nil
Lead peroxide	1309-60-0	Nil	Nil	Nil
Lead carbonate (white lead)	1319-46-6	Nil	Nil	Nil
Lead chrome 1244	unknown	Nil	Nil	Nil
<b>Total Lead Salts (Tonnes)</b>		<b>47</b>	<b>43</b>	<b>159</b>

**Table 5.3 - Quantities of lead compounds introduced into Australia for inks 2003 to 2005 (tonnes)**

<b>Chemical Name</b>	<b>CAS Number</b>	<b>2003</b>	<b>2004</b>	<b>2005</b>
Lead monoxide	1317-36-8	Nil	Nil	Nil
Lead chromate	7758-97-6	2	2	5
Lead sulfate	7446-14-2	2	1	1
Lead molybdate	10190-55-3	Nil	Nil	Nil
Lead sulfo-chromate	1344-37-2	3	3	4
Lead chromate molybdate sulfate red	12656-85-8	3	2	2
Lead chromate oxide	18454-12-1	Nil	Nil	Nil
Lead octanoate	7319-86-0	Nil	Nil	Nil
Lead 2-ethylhexanoate	301-08-6	Nil	Nil	Nil
Lead oxide	1314-41-6	Nil	Nil	Nil
Lead nitrate	10099-74-8	Nil	Nil	Nil
Lead naphthenate	61790-14-5	Nil	Nil	Nil
Lead peroxide	1309-60-0	Nil	Nil	Nil
Lead carbonate (white lead)	1319-46-6	Nil	Nil	Nil
Lead chrome 1244	unknown	Nil	Nil	Nil
<b>Total Lead Salts (Tonnes)</b>		<b>10</b>	<b>8</b>	<b>12</b>

## 5.2 Uses in Australia

Though this assessment focuses on lead compounds in industrial surface coatings and inks, all reported uses of the declared lead compounds in Australia are listed below and the reported uses for the individual compounds are given in Table 5.4. The list is not exhaustive given that applicants were required to report compounds used in industrial surface coatings and inks only. Some persons providing data did however detail other uses but it is likely this information does not cover all uses of the declared lead compounds in Australia.

### Industrial surface coatings

- Metals
- Steel
- Road marking
- Automotive
- Aerospace
- Drum reconditioning

- Colour sample swatches

#### Inks

- Flexographic inks

#### Other uses

- Marine batteries
- Mining; Gold mining (used to stop copper reacting with sodium cyanide)
- Pigment for plastics (Masterbatch)
- Chemical manufacture
- Powder coatings
- Glass fluxes
- Gear lubricant oil additive

It is known that manufacture of motor vehicle batteries involves the use of lead compounds however no respondents to the declaration indicated use of any of the declared lead compounds for this purpose.

Table 5.5 shows the proportion of all imported declared lead salts used in automotive coatings, inks and other unspecified industrial purposes.

The reasons for the observed trend in imports for use in automotive and other industrial surface coatings is not known.

### **5.3 Formulation of products**

A general description of the processes used in formulating industrial surface coatings and inks are provided below. Detailed descriptions can be found in the Exposure section of this report.

#### **5.3.1 Industrial surface coatings**

Formulation processes differ depending on the form used, i.e. powders or base/paste. Concentrations of lead in formulated industrial surface coatings could be up to 50% w/w.

##### **Powder**

The lead compound in powder form is mixed into a wet slurry and the slurry is then milled to reduce particle size. Some formulators grind the slurry in a fully enclosed mill, from which it is transferred to a tank where it is mixed at high speed with resins and other ingredients in a partly enclosed process to make the final paint product.

**Table 5.4 - Reported uses of declared lead compounds introduced into Australia 2003 to 2005**

Chemical Name	CAS Number	Use
Lead monoxide	1317-36-8	Glass, ceramics, mining
Lead chromate	7758-97-6	Paints, inks, plastics
Lead sulfate	7446-14-2	Paints, inks, plastics
Lead molybdate	10190-55-3	plastics
Lead sulfo-chromate	1344-37-2	Paints, ceramics, powder coatings, inks, plastics
Lead chromate molybdate sulfate red	12656-85-8	Paints, ceramics, powder coatings, inks, plastics
Lead chromate oxide	18454-12-1	No data
Lead octanoate	7319-86-0	Paints
Lead 2-ethylhexanoate	301-08-6	Paints
Lead oxide	1314-41-6	Glass, ceramics, marine batteries
Lead nitrate	10099-74-8	Mining and mineral extraction, chemical manufacture
Lead naphthenate	61790-14-5	Lubricant additive
Lead peroxide	1309-60-0	No data
Lead carbonate (white lead)	1319-46-6	No data
Lead chrome 1244	unknown	No data

**Table 5.5 - Percentage (%) of total import of declared lead compounds for different uses 2003 to 2005**

Use	2003 (%)	2004 (%)	2005 (%)
Automotive Coatings	38	14	51
Inks	8	3	4
Other uses *	54	83	45

\* calculated as 100% – (% automotive + % inks)

Other formulators transfer the slurry to a partly enclosed mill which is under negative pressure exhaust ventilation and then mix the final product under exhaust ventilation. Others use an open system to grind the slurry with exhaust ventilation over the mill, then transfer and mix the ingredients again under local exhaust ventilation. The finished product is packed and stored for distribution to customers.

### **Base/paste**

Users of the tinting base or paste, mix the product in a fully automated enclosed process. Drums of paste are set up on the production line, open drums containing premixed ingredients move along the line and measured amounts of paste are added to achieve the final colour. The drums are re-sealed and agitated to mix the ingredients, then sent for distribution.

### **5.3.2 Inks**

Only one company reported formulation of lead-based inks, using a process similar to that used for industrial surface coating. Lead pigment powder is mixed into a slurry, the slurry is milled, mixed with other ingredients and packed.



# 6. Exposure

## 6.1 Occupational exposure

Occupational exposure to lead may occur during transport, storage or use of the chemical or formulated products containing lead compounds. Lead compounds are available either as a powder or paste form, or in finished products (solutions or powder coats) containing varying concentrations of lead. Lead compounds for use in industrial surface coatings and inks are not manufactured in Australia.

The main routes of occupational exposure to lead are inhalation and dermal. Lead chromate, lead sulfate, lead molybdate, lead sulfo-chromate, lead chromate molybdate sulfate red and lead 2-ethylhexanoate (Table 5.1) are imported for use in surface coatings and inks. Therefore some workers exposed to these compounds are also likely to be exposed to chromates during use of these pigments and products containing them.

### 6.1.1 Importation and storage

Lead compounds are imported either as a powder form or in a premixed concentrated tinting base or tinting paste.

Importers either on-sell the chemicals directly to customers or formulate products which are then marketed. Storage of imported compounds prior to sale to customers is in warehouses and the chemicals are normally transferred in their original packaging.

The chemicals or finished products are imported in a container by sea. Commonly, the whole container is transported to the warehouse where it is unpacked. The packaging of the chemical or product are not opened prior to distribution to customers. If the container is a mixed delivery then it is unpacked at the wharf, the lead product is loaded onto trucks and distributed as above. Use of PPE is not anticipated in these processes.

Three importers use a contract dangerous goods store remote from their own site. Lead compounds are sent to customers from the contractor site as required. It is reported that up to 25 workers could be potentially exposed to lead over the three storage sites. However this is likely only in the event of an accident.

Importers who on-sell and use onsite storage also do not unpack finished products but dispatch it directly to customers. Up to 8 workers may be potentially exposed in the event of an accident

### 6.1.2 Formulation of industrial paints

It is estimated that there are approximately 60 formulators involved in formulation of lead-based industrial surface coatings. Limited information on current industry practices was obtained from approximately 11% of formulators.

It is therefore likely that the information provided below is not entirely representative of the industry sector.

### **Use of powders**

Typically the whole bag of powder is not used for a single batch, though where possible formulators prefer to use full bags. The bag is cut open using a knife under local exhaust ventilation. The powder is scooped out either directly into a vessel which contains liquid dispersants or is weighed into a smaller bag which is sealed, then transferred to another production area where a worker cuts open the bag and empties it into a vessel containing liquid dispersants. The powder is then mixed into a wet slurry. Some formulators then mill the slurry in a fully enclosed mill, transfer the milled slurry to a storage tank where it is mixed at high speed in a partly enclosed process with resins and other ingredients to make the final paint product.

Other formulators transfer the slurry to a partly enclosed mill which is under negative pressure exhaust ventilation. The milled slurry is transferred in an open system to another mixing vessel, mixed, then packed. Others use an open system to grind the slurry, with exhaust ventilation over the grinding machine, then mix the ingredients, again under local exhaust ventilation, followed by packing into containers. Other formulators mill at the same time the slurry is mixed. The milled slurry is pumped to another mixing vessel where it is added to other ingredients, mixed then packed. In each case the mixing vessels are closed by a lid after the ingredients are added.

In all cases, finished product is packed and stored for distribution to customers. Up to 35 workers are involved in the formulation process Australia wide, with only up to 3 workers at any one site. Potential routes of exposure in formulation would be via inhalation and from dermal contact. Applicants reported that workers generally wear glasses, gloves, overalls and masks.

Empty bags and disposable overalls are placed in plastic bags which are sealed and sent for disposal, or placed into bins and disposed of as hazardous waste or disposed to landfill.

### **Use of pastes**

The paste containing the lead pigment, is supplied in 4 L drums and the base with pre-mixed ingredients in 20 L drums. The drums are opened manually and placed into an automated partly enclosed machine. The paste is added to the drum (20L) containing a base of pre-mixed ingredients. The 20 L drum is re-sealed and the ingredients are mixed. Up to 2 workers are involved in this activity. Because there is no powdered pigment used no local exhaust ventilation is provided in the mixing area however workers wear protective overalls, gloves and glasses. The empty paste containers are placed in a rubbish skip containing other hazardous waste then disposed of as hazardous waste.

Exposure is potentially via the dermal route.

## **Maintenance and cleaning**

Personnel cleaning production lines and servicing manufacturing equipment, are likely to be exposed to residues. Exposure is potentially by inhalation and skin contact. No data were provided on engineering controls or PPE worn by maintenance personnel.

### **6.1.3 Formulation of powder coatings**

Lead-based powder coats are imported as the finished product and no local formulation occurs.

### **6.1.4 Formulation of inks**

Data were received from three formulators of inks. Two ceased using lead compounds for inks in 2005. The third formulator manufactures lead-based inks as a very small part of its product range and is phasing out the leaded inks.

Formulation is a batch process with typically 2 – 4 batches per year. Four workers are potentially exposed to the chemicals during each batch process. Batch manufacture involves four stages. In the first stage an operator cuts open the bag of powder with a knife and manually takes out the required amount using a scoop. The lead compound is added to a vat with other liquid ingredients and mixed into a slurry, in an open system under local exhaust ventilation. Possible routes of exposure are dermal and inhalation and the worker involved wears goggles, dust mask, disposable overalls and gloves. In the second stage the mixed contents are transferred in an open system to a milling area where the powder is wetted and ground again in an open system under local exhaust ventilation. The worker involved at this stage does not wear a dust mask as no dust is generated. In the third stage the milled powder is transferred to another open mixing vat with other ingredients and mixed into the final product by one worker. The final product is then packed in 1 kg containers. The packing system is open and one worker is involved.

Typically the full contents of the bag containing the lead compound is not used at one time and the top of the opened bag is folded over and the bag kept in the initial mixing area until needed again. The empty bag is disposed of in a dump bin. Liquid wastes including solvents used in cleaning operations are disposed of as hazardous waste.

## **Maintenance and cleaning**

Personnel cleaning production lines and servicing manufacturing equipment, are likely to be exposed to residues. Exposure is potentially by inhalation and skin contact. No data were provided on engineering controls or PPE worn by maintenance personnel.

### **6.1.5 Use of industrial surface coating products**

Industrial surface coatings are generally applied in industrial situations by brushing, spraying or powder coating.

## **Brushing**

In this process the coating is applied with a brush to the surface of the object to be coated and can lead to dermal exposure. No data was available to indicate the extent to which brushing is used for application of industrial surface coatings.

## **Powder coating**

Powder coating is employed for substrates that can withstand heating, typically metals. No data were received indicating the extent to which powder coats are used, however lead containing products are used for coating sheet metal equipment such as cabinets and small metal equipment.

Application is by manual or automated systems where the powder coat is sprayed by air pressure onto the substrate. The powder particles are charged electrostatically using a high voltage, low amperage system so that the particles adhere to the substrate. After spraying the equipment is moved to an oven and heated to a temperature greater than the melting point of the binder (CEPE, 2005). Dermal and inhalation exposure to lead containing dusts is possible during this process. No data were provided on controls or PPE use by workers applying powder coatings.

## **Spray painting**

There are a number of different spraying techniques in use:

- Air spray systems, including low volume/high pressure (LVHP).
- High volume/low pressure (HVLP), and low pressure/low volume coating applications (LPLV).
- Airless and air-assisted airless spray coating systems.
- Electrostatic spray systems.

Spraying can lead to dermal, oral and inhalation exposure to aerosols and dusts, as a result of overspray and dried residues.

Inhaled particles less than 1  $\mu\text{m}$  in diameter can be deposited in the alveolar region of the lungs. Particles of diameter greater than 2.5  $\mu\text{m}$  are deposited in the nasopharyngeal and tracheobronchial regions from where mucocillary transport can move them into the oesophagus from where the particles are swallowed (ATSDR, 2005).

LaPuma and Rhodes (2002), investigating particle size from chromate containing primers found sizes ranging from 0.7 to 34.1  $\mu\text{m}$ . In a study by Kalman and co-workers, around 2% of particles generated from spraying lead-based paint were found to be greater than 10  $\mu\text{m}$  in diameter. Particle size in overspray was found to be between 2.9 to 9.7  $\mu\text{m}$  for high solid content paints (D'Arcy and Chen, 1990) and 5.9  $\mu\text{m}$  (Rania et al, 2004) from spraying chromate based paint. Mean aerosol diameter was 8.2  $\mu\text{m}$  with gun atomisation pressure of 6 psi, decreasing to 7.0  $\mu\text{m}$  under 10 psi pressure (Rania et al, 2004).

No data on exposure mitigation methods used in general spray painting were provided.

Spray painting can be used in any application but is probably the only method of paint application in the automotive repair industry.

Table 5.5 shows that 51% of the 2005 import of lead-based pigments was used in automotive refinishing. Since this industry is the largest single user of lead-based industrial surface coatings and all important exposure scenarios are represented in this industry, this assessment will consider automotive refinishing as representative of possible worker exposure to lead compounds in industrial surface coatings. Users of lead-based paints in this industry are also subject to exposure to chromates, as Table 5.2 shows, the only lead compounds used in automotive paints are the chromates.

Lead-based paints are also used in “two pack” systems where isocyanates are used as a hardener. No data were provided on which lead-based products were used as “two pack” systems but advice from industry indicates that up to 80% of lead-based paints, when used for automotive vehicle panel repair, would be applied with the addition of an isocyanate hardener.

### **Automotive refinishing**

It is estimated there are between 4000 and 6000 motor vehicle panel repair shops in Australia (APMF, 2006). Data compiled by Insurance Statistics Australia (ISA) indicates that around 13% of motor vehicle insurance policies are claimed on as a result of a collision (ISA, 2006) (Table 6.1). ISA collects data from insurers that have about 70% market share based on number of registered vehicles.

**Table 6.1 - Claims frequency for comprehensively insured motor vehicles in Australia involved in collisions or from other causes**

<b>Quarter</b>	<b>Claim Frequency % *</b>	<b>Ave Claim Size (\$)</b>
June 2003	13.19	2261
Sept 2003	13.08	2216
Dec 2003	14.22	2429
Mar 2004	13.72	2271
June 2004	13.16	2226
Sept 2004	13.55	2229
Dec 2004	13.90	2279
Mar 2005	14.00	2490
June 2005	13.55	2288
Sept 2005	12.87	2412
Dec 2005	13.43	2564
Mar 2006	13.31	2415
Jun 2006	12.82	2429

\* Claim frequency – 100 (number of claims reported/number of policies in force)

No data on monitoring, numbers of workers exposed or duration of exposure were available for workers in this industry. A number of stages make up automotive panel repair.

An initial stage prior to painting is surface preparation where the damaged body panel is stripped of old paint. This is done either by chemical means or by a sanding machine, or a combination of both. No data were provided to indicate the extent to which a particular stripping technique is used. There is potential for high exposures, both by inhalation and dermal routes, during use of sanding machines for surface preparation. The potential for exposure also exists when chemical means are used for paint stripping as this is followed by manual removal of paint flakes.

A study of vehicle repair technicians in the United States undertaking vehicle preparation prior to painting, found blood lead levels of above 30 µg/dL, compared with blood levels of lead in workers not engaged in preparatory work, of 2.8 µg/dL (Enander et al, 2004). The authors note that the sanding dust adhered to workers hands and was available for ingestion from handling of food and hand to mouth contact. Exposure in this situation could therefore be dermal, particularly for chemical stripping and oral and inhalation of dusts during mechanical stripping. In one study of body repair facilities and vocational training establishments in the United States, methylene chloride was used as a chemical stripper by 17% of respondents as an adjunct to mechanical stripping (Enander et al, 2002). In the same study the authors found that Pb, As, Cr, Mn and Ni were present in sanding dust.

Paints are mixed prior to application and mixing can result in dermal exposure.

Spray application potentially provides the greatest opportunity for inhalation exposure from both aerosols and dusts of dried paint, but this depends on particle size. Particles less than 1 µm in diameter are almost completely absorbed through the lung but particles exceeding 1 µm in diameter are orally ingested and absorption is far less than via the lung. Dermal exposure is also possible. However, spray booths and work practices limit exposure at this stage.

Following application the paint surface is hand sanded using a wet sanding technique which can lead to dermal exposure to wet sludges containing surface coating materials. Inhalation exposure is unlikely in this situation.

The APMF *Code of Practice for the Automotive Refinishing Industry* (APMF, 2005) details ways to avoid exposure to the hazardous components of paints, ranging from good housekeeping and PPE when storing and mixing paints, to the use of PPE and specialised fully enclosed spray booths during paint application.

#### **6.1.6 Use of inks**

Lead-based inks used in commercial printing processes are not available for consumer use. However, items printed with these inks are used as products or as packaging for products, which are used by consumers.

In the printing process, liquid inks are poured manually into an ink pump, which feeds the product to a printing press for application to the item to be printed. After

addition of the ink, the process is fully automated and can be either open or fully enclosed. The press is cleaned of ink residues at the end of each day using a solvent and the waste recycled for re-use. NICNAS visited one printing company and apart from the use of gloves when filling and cleaning the ink pump and printing press, no other PPE was routinely worn.

Potential routes of exposure would be dermal as a result of spills and from the cleaning process.

### **6.1.7 Occupational monitoring and exposure**

Atmospheric and biological monitoring are required under the *National Standard for the Control of Inorganic Lead at Work [NOHSC:1012 (1994)]*. However, no companies provided atmospheric or biological monitoring data, hence exposure was estimated using EASE assuming local exhaust ventilation is available during the painting operation. To estimate inhalation exposure the model requires a vapour pressure. No data on vapour pressures on the various lead salts were available. An upper limit for vapour pressure was estimated using the vapour pressure of metallic lead of 1.77 mmHg at 1000°C (ATSDR, 2005). A value of  $10^{-9}$  kpa for room temperature vapour pressure was used for the model as an estimate of the vapour pressure of all the declared lead compounds.

EASE predicted exposures were:

1. Dermal exposure from spraying – 0.1 to 1 mg/cm<sup>2</sup>/day
2. Inhalation exposure from spraying – 0 to 0.1 ppm
3. Inhalation exposure to dusts – 2 to 5 ppm

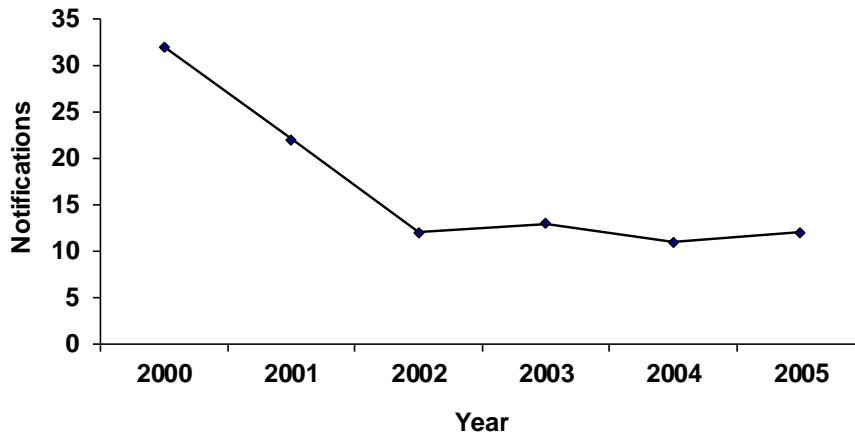
### **6.1.8 Biological monitoring**

Biological monitoring data over all occupational groups were available from Queensland Health and New South Wales Health.

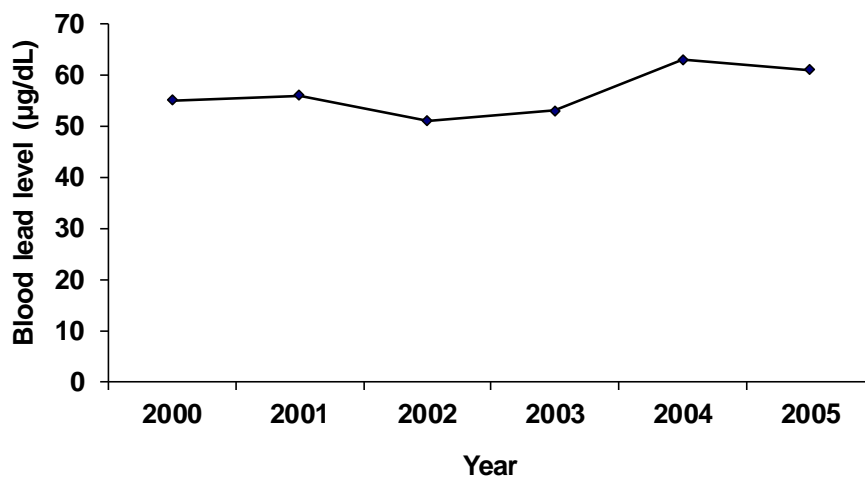
Under the *Public Health Act (2005)* (Qld) lead exposure is a notifiable condition. For the purposes of determining whether a blood lead level is notifiable, a distinction is made under the *Public Health Act (2005)* (Qld) between occupational and non-occupational exposure, the cut-off for the former being 2.41 µMol/L (~ 50 µg/dL) or greater. Data on notifiable conditions in Queensland is compiled by Queensland Health and published on their website at <http://www.health.qld.gov.au/phs/documents/ehu/31749.pdf> - (QLD, 2005).

The following is taken from the published document on the internet site, and reproduced here with permission.

**Figure 6.1 – Total notifications of elevated blood lead levels over all occupational groups in Queensland during the years 2000 to 2005 (Source: Queensland Health)**



**Figure 6.2 – Median blood lead levels over all occupational groups in Queensland during the years 2000 to 2005 (Source Queensland Health)**



The data in Figures 6.1 and 6.2 are for all occupational groups over the age range 20 to 41 years, however workers involved in manufacture and use of surface coatings and inks are not specifically listed.



Table 6.2 shows the total number of notifications for industries where paint is removed from surfaces, for workers in the age range of 20 to 41 years. Though painters, for example are not given as a specific occupational group it is likely that some workers involved in paint removal also applied paint to the cleaned surface. No data from workers in the automotive repair industry were available.

**Table 6.2 - Total notifications of elevated blood lead levels over specific occupational groups in Queensland during the years 2000 to 2005 (Source: Queensland Health)**

Cause of lead exposure	2000	2001	2002	2003	2004	2005
Removal of paint from domestic buildings	8	7	1	2	1	2
Removal of paint from other structures (e.g. boats, bridges)	0	0	0	1	1	0
Maintenance or demolition work	0	0	0	0	0	2

The number of notifications for occupational exposures has dropped from 2000 to 2005 though the median blood lead levels are largely unchanged.

In New South Wales there are no prescribed cut-off differences for determining if blood lead levels from an occupationally exposed person are notifiable compared with non-occupational exposure. However, under the *Public Health Act (1991)* (NSW) all blood lead readings at or above 15 µg/dL (0.72 µmol/L) require notification regardless of the exposure history of the patient. Under the *Occupational Health and Safety Regulations (2001)* (NSW) employers are required to notify WorkCover if a person is removed from lead risk work due to excessive blood lead levels, which are declared in the regulations and are consistent with the levels set out in the *National Standard for the Control of Inorganic Lead at Work (NOHSC, 1994)*, (see section 10.2.1).

Though occupational blood lead data for the years 2000, 2001, 2005 and 2006 were provided by Workcover New South Wales, these data were incomplete with a full twelve months monitoring data only available for the year 2000. No conclusions can be drawn from the NSW data. The companies in NSW providing monitoring data to Workcover on workers with blood lead levels at or above 15 µg/dL (0.72 µmol/L), were not from industries associated with surface coating and inks import, manufacture or use. No data on occupational blood lead levels from industries associated with surface coating and inks import, manufacture or use were available from the other states and territories. Victoria indicated that in the past ten years only one company in those industries had notified WorkSafe that an activity was classed as “lead risk”, however, no blood lead levels above the notifiable cut off were reported from that company.

Information was sought by NICNAS from the states and territories, on the extent of compliance by industry with the requirements of the states and territories for companies to monitor blood lead levels of workers. No state or territory could

provide data on compliance by industry with the requirements for “lead risk jobs” and for monitoring of workers involved in “lead risk jobs”. Therefore, it is not known whether the decreases in notified occupational blood lead levels seen in Queensland and the lack of data on notified blood lead levels in industries associated with surface coating and inks in other jurisdictions, reflect the effectiveness of the controls in reducing lead exposure in the workplace or a lack of compliance by industry with its obligations.

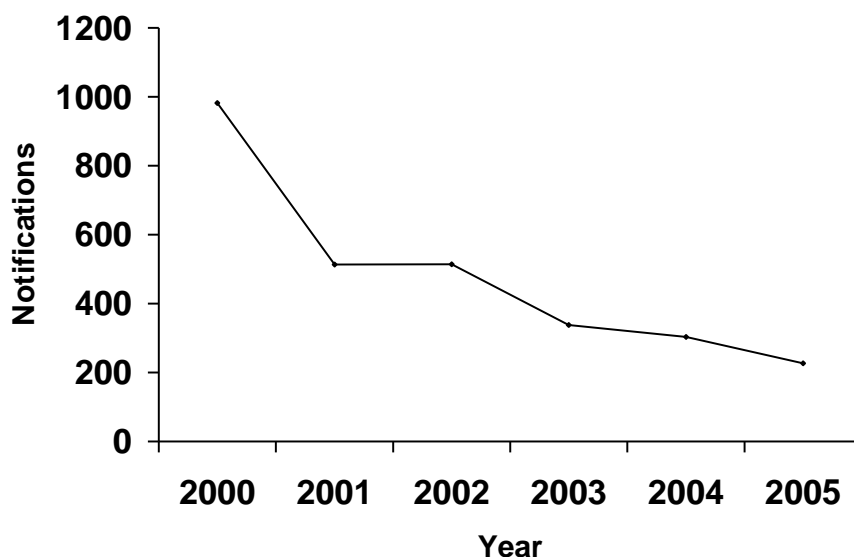
## 6.2 Consumer exposure

Lead compounds have been eliminated from surface coatings used in a domestic setting. Consumer exposure would largely be from renovations of buildings where leaded paints were used in the past, or through inappropriate use of industrial coatings in a domestic situation. Hobbyists such as car restorers or persons who undertake their own panel repairs on vehicles could also be exposed to paints containing lead. Consumer exposure to inks containing lead would be minimal.

### 6.2.1 Biological monitoring of the general population

Under the *Public Health Act 1991* (NSW) blood levels of lead above 15 µg/dL (0.72 µmol/L) are defined as elevated lead levels and must be notified. New South Wales Health provided data for this assessment on the number of notifiable blood lead samples taken at public hospitals throughout the state from the year 2000 to 2005. The number of notifiable blood samples has decreased since the year 2000 (Figure 6.3) as has the number in defined age groups (Table 6.3) It can be seen from Table 6.2 that by the year 2005, the number of notifiable samples in the population aged from 15 to 64, was greater than in other age groups. The years from 15 to 64 are the ones a person would most likely be working. As New South Wales requires all blood lead levels above 15 µg/dL (0.72 µmol/L) to be notified it is likely that the data in Figure 6.3 and Table 6.3 include persons who were occupationally exposed to lead.

**Figure 6.3 - Number of notifiable blood samples containing lead in NSW during the years 2000 to 2005 (Source: NSW Health)**

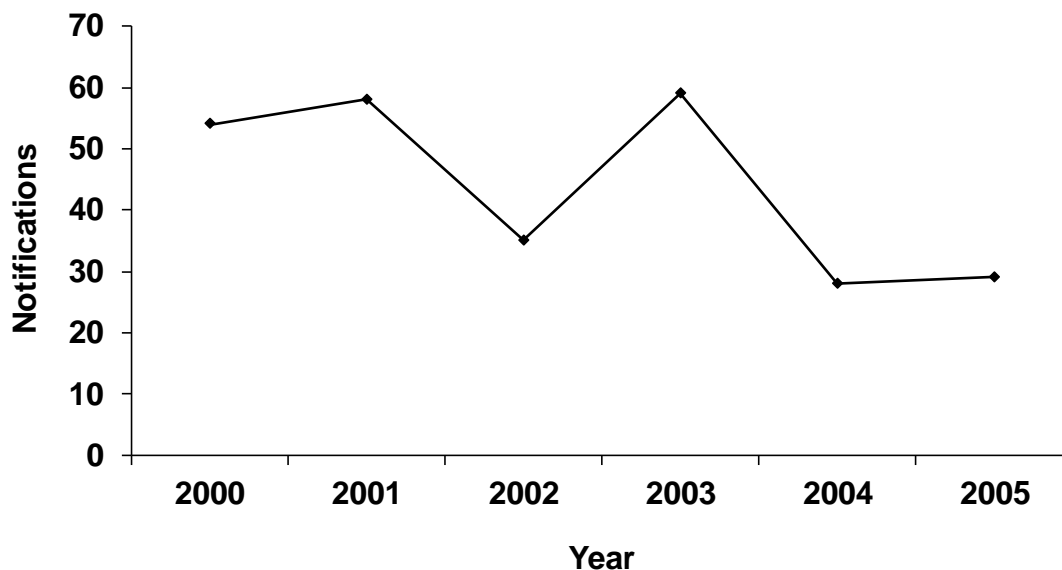


**Table 6.3 - Number of blood lead notifications in NSW during the years 2000 to 2005 by age group (Source: NSW Health)**

<b>AGE (Years)</b>	<b>2000</b>	<b>2001</b>	<b>2002</b>	<b>2003</b>	<b>2004</b>	<b>2005</b>
0-4	53	51	49	50	40	7
5-9	2	10	8	4	2	0
10-14	3	2	1	0	0	0
15-19	24	15	14	9	9	14
20-24	102	47	53	32	24	17
25-29	123	74	53	37	25	21
30-34	135	68	64	34	36	28
35-39	140	70	56	38	32	23
40-44	129	55	64	41	38	29
45-49	97	33	53	26	28	22
50-54	79	45	40	32	26	29
55-59	51	17	27	16	26	17
60-64	23	12	22	12	11	10
65-69	12	5	6	3	3	4
70-74	4	2	1	2	3	4
75-79	1	4	1	1	0	1
80-84	1	1	2	1	0	1
85+	3	1	0	0	0	0
unknown	0	1	0	0	0	0
<b>TOTAL</b>	<b>982</b>	<b>513</b>	<b>514</b>	<b>338</b>	<b>303</b>	<b>227</b>

In Queensland, the cut-off under the *Public Health Act (2005)* (Qld) to determine whether a non-occupational exposure blood lead level is notifiable is 0.73  $\mu\text{Mol/L}$  ( $\sim 15 \mu\text{g/dL}$ ) or greater. Figure 6.4 shows the total non-occupational blood lead notifications over the years 2000 to 2005 in Queensland.

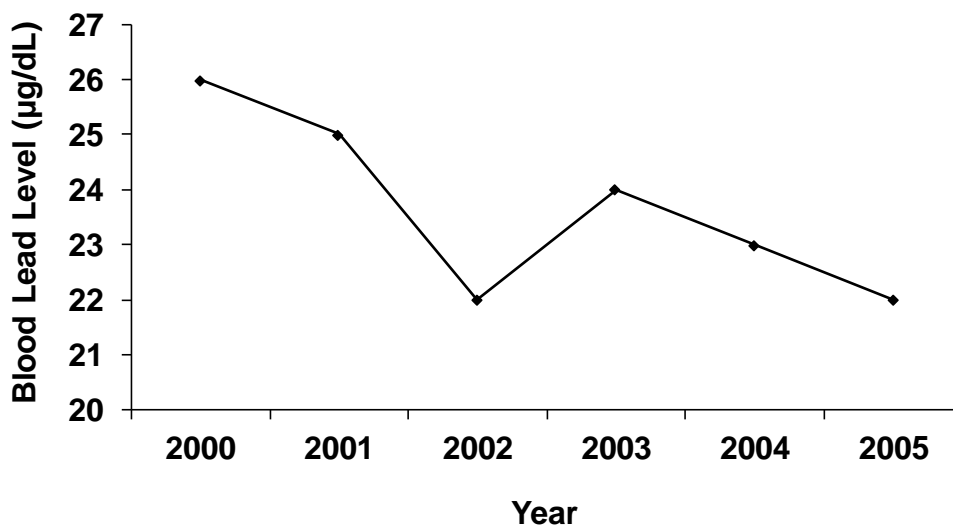
**Figure 6.4 – Total non-occupational notifiable blood lead levels in Queensland from 2000 to 2005 (Source: Qld Health)**



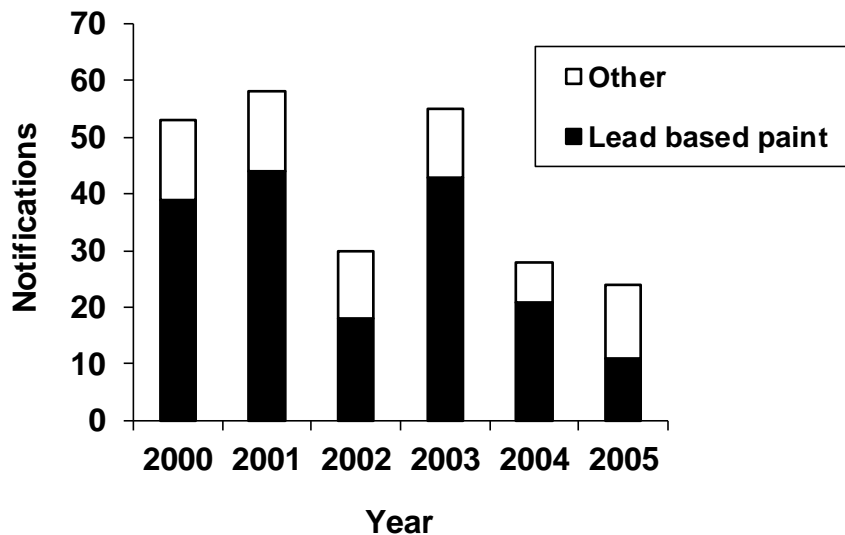
Median blood lead levels for non-occupational exposure are shown in Figure 6.5 and follow the downward trend observed for numbers of notifications.

In 2005 in Queensland, 38% of non-occupational notifications were by exposure to lead-based paint of which 14% arose during maintenance and demolition work (Figure 6.5).

**Figure 6.5 – Median blood lead levels over all non-occupational notifications in Queensland during the years 2000 to 2005 (Source: Queensland Health)**



**Figure 6.6 – Notified blood lead levels by cause over all non-occupational notifications in Queensland during the years 2000 to 2005 (Source: Queensland Health)**



No data from other states or territories were available for non-occupational exposure.

In Queensland, the number of notifications for non-occupational exposures has dropped over the years from 2000 to 2005. Also, in Queensland (no data from NSW) the median blood lead levels fell by around 15% for the non-occupational exposure group over that period.

In NSW in 2005, the lower numbers of notifiable blood samples in the age groups under 15 and over 65, compared with the other age groups suggest that lead exposure in the home, whilst a problem, may not be as significant when compared with occupational exposure.

## 7. Kinetics and Metabolism

In this section, a short discussion based on the draft ATSDR is presented. It is not a comprehensive review of the available literature and references marked with an asterisk have not been sighted.

### 7.1 Absorption

Inorganic lead can be absorbed orally, dermally or by inhalation, with the dermal route being the least efficient (ATSDR, 2005).

Solubility is an important factor in absorption and the water solubilities of the declared lead compounds are given in Table 7.1.

**Table 7.1 - Partition coefficient and water solubility of the declared lead compounds**

<b>Chemical Name</b>	<b>Partition Coefficient (Log Pow)</b>	<b>Solubility in Water</b>
Lead monoxide	No data	17 mg/L
Lead chromate	No data	0.2 mg/L
Lead sulfate	No data	42.5 mg/L
Lead molybdate	No data	Insoluble
Lead sulfo-chromate	No data	No data
Lead chromate molybdate sulfate red	No data	No data
Lead chromate oxide	No data	No data
Lead octanoate	No data	No data
Lead 2-ethylhexanoate	No data	No data
Lead oxide	No data	Insoluble
Lead nitrate	No data	565 g/L
Lead naphthenate	No data	No data
Lead peroxide	No data	No data
Lead carbonate (white lead)	No data	0.17 mg/100g
Lead chrome 1244	No data	No data

Though there is no data on partition coefficients, it can be assumed that the more soluble inorganic leads would ionise and partition to water whilst the compounds with an organic anion would partition to the lipid phase.

### **7.1.1 Inhalation**

Absorption after inhalation depends on a number of factors. The site of deposition, airways clearance and aerodynamics of particles of different sizes will influence the extent of absorption. The particle size is an important determinant of the site of deposition, determining the level of the bronchial tree in which the particle could be deposited.

Inhaled particles less than 1  $\mu\text{m}$  in diameter can be deposited in the alveolar region of the lungs. Particles of diameter greater than 2.5  $\mu\text{m}$  deposit in the nasopharyngeal and tracheobronchial regions from where mucociliary transport can move them into the oesophagus from where the particles are swallowed. Of particles less than 1  $\mu\text{m}$  in diameter, 95% of deposited lead is absorbed (ATSDR, 2005).

Following inhalation of particulates, the lead dust comes into contact with lung macrophages, which are primarily responsible for phagocytosis. Damage to alveolar macrophages has been demonstrated in vitro and in vivo by inorganic lead. This damage to the lung defence mechanisms could affect the rate of absorption of inhaled particles (EHC, 1977).

### **7.1.2 Dermal**

Dermal absorption is likely to be minimal and varies with the compound. Less than 0.3% of lead from lead acetate in cosmetics was absorbed in male volunteers over 12 h. Thirty percent of a dose of lead nitrate applied to the skin was absorbed but it is not known whether this dose remained in the skin layers or was systemically absorbed (ATSDR, 2005).

### **7.1.3 Oral**

Gastrointestinal absorption of inorganic lead is influenced by many factors, the most important being age. Estimates in infants and children aged up to eight years, indicate that 40% to 50% of an ingested dose of lead is absorbed while in adults absorption is 3% to 10% of the ingested dose. Particle size is also important in lead absorption with smaller particles being absorbed more compared to larger particles (ATSDR, 2005). It is unclear whether different lead compounds are absorbed at differing rates following ingestion (SCOEL, 2002).

## **7.2 Distribution**

Inorganic lead is distributed throughout the body and is independent of the route of absorption. Distribution is similar in children and adults but a larger proportion is distributed to bone in adults compared with children. In blood, 99% of lead is bound to proteins within erythrocytes (Smith et al., 2002\*; Al-Modhefer et al., 1991\*).

In children, 73% of the body burden of lead is deposited in bone compared with 94% in adults (Barry, 1975\*). Bone provides a reservoir of lead which can be slowly released to the blood well after exposure to lead has ceased (Flemming et al., 1997\*). Lead in bone can also transfer to the foetus during formation of the

foetal skeleton (Franklin et al., 1997\*). Distribution within bone is not uniform and lead accumulates in the areas undergoing active calcification at the time of exposure (Aufderheide and Wittmers, 1992\*).

Bone lead is exchanged with lead in blood and soft tissues, with bone lead contributing up to 70% of the lead in blood (Smith et al., 1996\*). Mobilisation of lead from bone increases during pregnancy when maternal bone is catabolised to produce the foetal skeleton. Up to 80% of lead in human foetal cord blood derives from maternal bone stores (Gulson et al., 2003\*). Lactation and osteoporosis also increase blood lead levels because of increased mobilisation of bone lead stores (Gulson et al., 2004\*). In a study in monkeys, up to 39% of the maternal body lead that was transferred to the foetus derived from the maternal skeleton (Franklin et al., 1997\*).

In soft tissue most of the lead is found in the liver. Relative percentages found in soft tissues from autopsy specimens are shown in Table 7.2.

**Table 7.2 - Distribution of inorganic lead in soft tissues from human autopsy specimens (after Schroeder and Tipton, 1968\*)**

<b>Tissue</b>	<b>Percent (%) of Total Soft Tissue Lead</b>
Liver	33
Skeletal muscle	18
Skin	16
Dense connective tissue	11
Fat	6.4
Kidney	4
Lung	4
Aorta	2
Brain	2
Other soft tissue	< 1

### **7.3 Metabolism**

Inorganic lead is metabolised by complexing with a variety of protein and non-protein ligands such as plasma albumin (Al-Modhefer et al., 1991\*). Bone turnover rates decrease with increasing age which results in bone lead levels slowly increasing with age and continued lead exposure (Barry, 1975\*), however, compared with bone, lead levels in soft tissue remain relatively constant with continued exposure (Treble and Thompson, 1997\*).

### **7.4 Excretion**

Lead is cleared from blood in around one month in human adults (Barry, 1975\*). Absorbed lead is excreted mainly in the faeces, urine, saliva, breast milk, sweat, hair and nails (Stauber et al., 1994\*). About 33% of the dose of lead is excreted in the faeces (Chamberlain et al., 1978\*).



## 8. Health Effects

This chapter summarises the health effects of lead and the declared lead compounds. As lead has an extensive database and the toxicity profile is well known, most of the studies in this section have been primarily summarised from the draft Agency for Toxic Substances and Disease Registry review (ATSDR, 2005), though occasionally studies have been summarised from the World Health Organization review (WHO, 1995). In addition, a comprehensive literature search was carried out on studies conducted from 2005 to date, for additional material of relevance to the hazard assessment that is not included in the draft ATSDR review. References that have not been sighted are marked with an astrix.

The database on the health effects of lead is extensive in both humans and animals, with the effects of lead on human health having been known for a number of centuries (ATSDR, 2005). Furthermore, there is a substantial amount of recent data on the health effects of lead, which is available from both human and animal studies (ATSDR, 2005; WHO, 1995). Most of the recent human health effects data comes from studies of occupationally exposed groups from a variety of industries and from studies of adults and children in the general population. In general the health effects of lead in animals supports the observations from human studies. However, animal data are generally considered less suitable as the basis for determining health effects in humans than human data, and there is no absolutely equivalent animal model for the effects of lead on humans (ATSDR, 2005). Consequently, in this section, animal studies will only be briefly discussed and only to the extent they support the results of human studies.

It should be noted that the primary data source (i.e. ATSDR, 2005) did not separate human data by routes of exposure as the dose data for humans are generally expressed in terms of absorbed dose in studies and not external exposure levels, or milligrams per kilogram per day. Blood lead concentration (PbB) is the most widely used measure of lead exposure, although other measures are also occasionally used, in particular bone lead concentration. The concentration of lead in the blood reflects mainly the exposure history of the previous few months, whereas lead in bone is considered a biomarker of cumulative or long-term exposure because lead accumulates in bone over the lifetime.

Most data on the health effects of lead compounds relate to the health effects of lead itself. There is much less data available on the specific health effects of the declared lead compounds used in industrial surface coatings and inks. Therefore, section 1 of this chapter summarises the information available on the health effects of lead. Section 2 of this chapter summarises the information available on the specific health effects of the declared lead compounds used in industrial surface coatings and inks.

## 8.1 Health effects of lead

### 8.1.1 Acute toxicity

There are relatively few data available for acute exposures in humans. Most data have been derived from cases of accidental exposure or intentional ingestion of dirt containing lead or lead-based paint in adults and children (ATSDR, 2005).

Acute exposure to high levels of lead may cause encephalopathy (symptoms include hyperirritability, ataxia, convulsions, stupor and coma) (ATSDR, 2005), and gastrointestinal effects such as colic (symptoms include abdominal pain, constipation, cramps, nausea, vomiting, anorexia, and weight loss) (WHO, 1995; ATSDR, 2005). Some data suggest that severe encephalopathy is generally not observed in adults except at very high PbBs (>460 µg/dL) (Kehoe, 1961\*). In contrast, other data indicate that symptoms of acute toxicity (severe gastrointestinal symptoms and/or signs of encephalopathy) may occur in adults at PbBs ranging from 50 to >300 µg/dL, although the data are old and ambiguous (Smith et al., 1938\*). Similarly, divergent results have been seen in children with respect to the PbB levels that neurological effects were seen.

The most extensive compilation of dose-response information on a paediatric population reported symptoms of encephalopathy in children at PbBs of 90-800 µg/dL (mean 330 µg/dL), though the distribution of PbBs associated with death (mean PbB 327 µg/dL) was almost the same as for levels associated with encephalopathy. In some children, there were no symptoms of acute lead poisoning at PbBs of 60-300 µg/dL (mean 105 µg/dL), while symptoms of acute lead poisoning, other than encephalopathy, were observed at PbBs of 60-450 µg/dL (mean 178 µg/dL) (National Academy of Sciences, 1972\*, unpublished data reported in Chisolm, 1962, 1965\* and Chisolm and Harrison, 1956\*). Furthermore, evidence from medical reports suggest that acute encephalopathy in the most susceptible children may be associated with PbBs of 80-100 µg/dL (Bradley and Baumgartner, 1958\*; Bradley et al., 1956\*; Gant, 1938\*; Rummo et al., 1979\*; Smith et al., 1983\*). However, a further study reported 19 cases of acute encephalopathy in infants with a mean age of 3.8 months and with mean PbB of 74.5 µg/dL following the use of traditional medicines containing lead (Al Khayet et al., 1997\*).

With regard to gastrointestinal effect, colic is a consistent early symptom of lead poisoning in individuals exposed to high levels of lead and typically occurs in workers at PbBs of 100 – 200 µg/dL (ATSDR, 2005). While in children, the National Academy of Sciences (1972\*) summary of paediatric data reported symptoms of acute colic (such as severe constipation, anorexia, and intermittent vomiting) occurring at PbBs  $\geq$ 60 µg/dL (unpublished data reported in Chisolm, 1962, 1965\* and Chisolm and Harrison, 1956\*).

Exposure to lead is also known to cause proximal renal tubular damage in the kidney (Chisolm, 1962). Furthermore, in children, exposure to lead has been shown to inhibit formation of the haem-containing protein cytochrome P-450, as reflected in decreased activity of hepatic mixed-function oxygenases. Two children with signs of acute lead poisoning did not metabolise the test drug antipyrine as rapidly as the controls (Alvares et al., 1975\*).

### 8.1.2 Irritation

There are no reports of skin, eye or respiratory irritation to lead in humans.

### 8.1.3 Sensitisation

Though altered immune parameters have been described in workers (ATSDR, 2005) there are no reports of skin or respiratory sensitisation to lead in humans.

The only available related information is from two studies of children that reported significant association between PbB and increases in serum IgE levels (Lutz et al., 1999\*; Sun et al., 2003\*), IgE is the primary mediator of type-1 hypersensitivity and is involved in various allergic diseases such as asthma.

### 8.1.4 Repeat dose toxicity

Due to the multi-modes of action of lead in biological systems, lead can potentially affect any system or organs in the body. However, the most sensitive target organs for lead toxicity are the nervous system, the haematological system, the cardiovascular system, and the kidney (ATSDR, 2005). Consequently, only the effects on the identified target organs are summarised below, and the reader is referred to the draft ATSDR (2005) report for a comprehensive review of the data and including the effects of lead on other biological systems/target organs.

#### Neurological effects

The most severe neurological effect of lead in adults is lead encephalopathy, which is generally not observed in adults except at extremely high PbBs. However, occupational exposure to lead has often been associated with signs of neurotoxicity. Numerous case reports and small cohort studies are available that report a higher incidence of the symptoms, including malaise, forgetfulness, irritability, lethargy, headache, fatigue, impotence, decreased libido, dizziness, weakness, and paresthesia at PbBs that range from approximately 40 to 120 µg/dL (ATSDR, 2005). Additionally, numerous studies have reported neuropsychological effects in lead workers.

For example the following neuropsychological effects have been reported in studies in lead workers.

- General performance on cognitive and visual motor coordination tasks and verbal reasoning ability impaired with PbBs 45 – 60 µg/dL (Campara et al., 1984\*).
- Disturbances in oculomotor function (saccadic eye movements) with mean PbBs of 57 – 61 µg/dL (Baloh et al., 1979\*; Spivey et al., 1980\*; Glickman et al., 1984\*).
- Deficits in hand-eye coordination and reaction time were seen for a mean PbB 60.5 µg/dL (NIOSH, 1974\*).
- Disturbances in reaction time, visual motor performance, hand dexterity, IQ tests and cognitive performance, nervousness, mood or coping ability were seen with PbBs of 50 – 80 µg/dL (Arnvig et al., 1980\*; Haenninen et al.,

1978\*; Hogstedt et al., 1983\*; Mantere et al., 1982\*; Valciukas et al., 1978\*).

- Impaired verbal concept formation, memory, and visual/motor performance were reported with PbBs > 40 µg/dL (Baker et al., 1983\*). Similar findings were reported for a mean PbB of 42 µg/dL (Maizlish et al., 1995\*).

In a study of 91 workers separated into groups with PbBs < 20, 21-40 and 41-80 µg/dL, evidence of impairment on tests of serial reaction time and category search was seen in workers in the high level group along with weak impairments on tasks measuring syntactic reasoning and delayed verbal free recall (Stollery et al., 1989\*, 1991\*). While in a study of 427 lead workers with a mean PbB of 27.5 µg/dL and mean duration of employment of 17.7 years, tasks that tested primarily visuomotor skills were significantly associated with a cumulative dose-estimate (Lindgren et al., 1996\*).

More recent studies of lead workers have also reported significant associations between longitudinal decrements in cognitive function and past high PbBs. The results of a study of 5 neuropsychological measures suggest that reversibility of function may occur when PbB is maintained below 40 µg/dL (Lindgren et al., 2003\*). While current not cumulative exposure was associated with impaired visual contrast sensitivity in workers with a current mean PbB of 27 µg/dL (range, 6-61 µg/dL) (Lucchini et al., 2000\*). Similarly, a significant correlation was seen between current not cumulative exposure (mean PbB, 31 µg/dL, range, 11-62 µg/dL) for cognitive deficits, particularly visuo-spatial abilities and executive functions (Barth et al., 2002\*).

Mixed results were observed in two recent meta-analyses that examined the magnitude of performance effects with PbBs <70 µg/dL due to occupational exposures. In a meta-analysis of 22 studies were mean PbBs of subjects ranged from 31-52 µg/dL compared to 6-20 µg/dL in controls, a small but statistically significant deficit was reported in the block design, logical memory and Santa Ana [dominant hand] test of the 13 tests assessed (Meyer-Baron and Seeber, 2000). A further meta-analysis was undertaken of 22 studies that assessed 22 tests. In this study, were mean PbBs of subjects ranged from 24-63 µg/dL compared to 0-28 µg/dL in controls, a small but significant effect was seen in the digit symbol and the D2 test but not in the block design, logical memory or Santa Ana [dominant hand] tests (Goodman et al., 2002).

Recently, the effect of lead exposure on neurobehavioural parameters in non-occupational cohorts of older persons (means > 65 years of age) has been evaluated. In 530 females participating in a osteoporotic fractures study, women with PbB ≥ 8 µg/dL performed significantly worse in tests of psychomotor speed, manual dexterity, sustained attention, and mental flexibility after adjustment for age, education and tobacco and alcohol consumption than women with PbB ≤ 3 µg/dL (Muldoon et al., 1996\*). A number of studies have been conducted using data from the Normative aging study. In a study of 141 men (mean PbB 5.5 µg/dL and mean patella and tibia lead levels 31.7 and 22.5 µg/g bone respectively) participants with higher blood and bone lead levels were reported to do worse in a range of cognitive tests than subjects with lower levels after adjusting for age and education (Payton et al., 1998). Furthermore, it was reported that both bone (mean patella Pb 29.5 ppm) and blood lead (mean PbB, 4.5 µg/dL)

were associated with poor cognitive test performance among 736 older men (Weisskopf et al., 2004\*; Wright et al., 2003). It was also reported that patellar lead levels showed a significant association with psychiatric symptoms among 526 participants (Rhodes et al., 2003\*). However, in these studies it is not known when lead exposure occurred and as cognition (which declines with age) is determined/affected by many factors confounding may have occurred. Furthermore, the mechanism of lead's effect on the brain is unclear. Consequently, it is considered that the limited evidence available from these studies does not robustly demonstrate that low levels of lead impair cognitive function among elderly people.

There are numerous studies available on peripheral nerve function that measured the conduction velocity of electrically stimulated nerves in the arm or leg of lead workers though mixed results have been observed with regard to the PbB level that effects have been observed. A prospective cohort study found decreased nerve conduction velocities (NCVs) in the ulnar and/or median nerves of workers with PbBs ranged from 30–48 µg/dL after 1, 2 or 4 years exposure, although the severity of effect appeared to lessen with continued exposure (Seppalainen et al., 1983\*). In contrast, a negative result was observed in a study that investigated ulnar and peroneal NCV in 55 workers exposed for 1 year or more with PbBs from 60 – 80 µg/dL (Spivey et al., 1980\*) and in a study that investigated median NCV in 58 male and 78 female ceramic workers with PbBs of 2.1 – 69.5 µg/dL (Ishida et al., 1996\*). In cross-sectional studies, a decrease in ulnar and median NCV were seen primarily in workers at PbBs > 70 µg/dL with 1 – 28 years exposure (Triebig et al., 1984\*), while a significant decrease in fibular and sural NCV was seen as a function of PbB but not duration of exposure in a further study (Rosen and Chesney, 1983\*).

A 1986 review of many of the studies of NVC effects concluded that a mild slowing of certain motor and sensory NCVs may occur at PbBs < 60 µg/dL, but the majority of the studies did not find correlations between PbB and NVC < 70 µg/dL. This review did not analyse the above studies by Rosen and Chesney (1983\*) and Seppalainen et al. (1983\*) (Ehle, 1986).

Recent studies have also produced mixed results. At the end of a 3-year study period a significant association was seen in 5 out of the 8 NCV parameters measured in median and ulnar nerves for lead-battery workers with PbB ≥ 40 µg/dL but not < 40 µg/dL when compared to unexposed referents with a mean PbB of 10.5 µg/dL (Chia et al., 1996\*). While in a further study in battery workers with a mean exposure duration of 30.4 months and a mean PbB of 63 µg/dL, a significantly increased distal latency was seen in the median nerve but not the ulnar, peroneal or tibial nerve in workers (Yeh et al., 1995\*).

Thus, while there is limited evidence of neuropsychological effects at blood lead levels of > 20 µg/dL such effects have been inconsistently seen and, overall, the data is considered to indicate that such effects occur with PbBs > 40 µg/dL. Similarly, while there is very limited evidence of affects on peripheral nerve function at PbBs > 30 µg/dL, overall, the data indicates that lead affects NCVs at > 40 µg/dL.

## Haematological effects

Lead has long been known to alter the haematological system inducing microcytic and hypochromic anaemia, that results primarily from both inhibition of heme synthesis and shortening of erythrocyte lifespan (ATSDR, 2005). The EPA (EPA, 1986\*) estimated the threshold PbB for a decrease in haemoglobin in occupationally exposed adults to be 50 µg/dL based on analysis of the data by Baker et al., 1979\*; Grandjean, 1979\*; Lilis et al., 1978\*; Tola et al., 1973\* and Wada et al., 1973\* (ATSDR, 2005). For children, the EPA (EPA, 1986\*) judged the PbB threshold for decreased haemoglobin levels to be approximately 40 µg/dL, based on analysis of the data by Adebonojo, 1974\*; Bettes et al., 1973\*; Pueschel et al., 1972\*; and Rosen et al., 1974\* (ATSDR, 2005). However, lead induced anaemia occurs through inhibiting the activities of several enzymes involved in heme synthesis. Of all the parameters examined the most sensitive indicator of effects on heme synthesis following lead exposure is δ-aminolevulinic acid dehydratase (ALAD), an enzyme occurring early in the heme synthesis pathway (ATSDR, 2005). ALAD depression is also a more sensitive indicator than the approach of defining anaemia as a hematocrit value less than 33%–35% of the normal range (Schwartz et al., 1990). Therefore, only studies investigating the effect of lead exposure on ALAD activity are summarised below. For a summary of studies investigating less sensitive biomarkers (such as urinary δ-aminolevulinic acid, blood erythrocyte protoporphyrin, blood zinc protoporphyrin, and haematocrit) the reader is referred to the draft ATSDR (2005) report.

Studies in the general population indicate that ALAD activity is inhibited at very low PbB levels. ALAD activity was inversely correlated with PbB over the entire range of 3–34 µg/dL detected in 26 medical students with no present or previous occupational exposure to lead (Hernberg and Nikkanen, 1970). In a study of volunteers exposed to different levels of particulate lead in air (0.003 or 0.01 mg Pb/m<sup>3</sup>) for 23 hours/day for 3-4 months, ALAD activity decreased to 80% of pre-exposure values in the exposure group whose mean PbB increased from 20 to 27 µg/dL after 5 weeks of exposure, and decreased to 53% of pre-exposure values in the exposure group whose mean PbB increased from 20 to 37 µg/dL after 4 weeks of exposure (Griffin et al., 1975\*). Similar observations were made in studies of volunteers who ingested lead acetate resulting in increased mean PbBs of 40 µg/dL (Stuik, 1974\*; Cools et al., 1976\*). An inverse correlation between PbB ranging from 3–30 µg/dL and ALAD activity was also seen in mothers (delivery) and their newborns (cord blood) (Lauwerys et al., 1978\*).

An inverse correlation between PbB levels and ALAD activity was observed in 142 workers with PbBs ranging from 11–94 µg/dL (Hernberg and Nikkanen, 1970). Several other occupational studies have also shown that ALAD activity correlated inversely with PbB (Alessio et al., 1976\*; Gurer-Orhan et al., 2004\*; Hernberg et al., 1970\*; Meredith et al., 1978\*; Schuhmacher et al., 1997\*; Tola et al., 1973\*; Wada et al., 1973\*).

Studies in animals, in general, support the findings in humans and indicate that the effects depend on the chemical form of lead, duration of exposure, and animal species (ATSDR, 2005).

Thus, decreased ALAD activity has been observed with PbBs within the range 3 µg/dL–94 µg/dL in humans. However, although inhibition of ALAD has been

observed at very low levels, there is some controversy as to the toxicological significance of a depression in ALAD activity in the absence of a detectable effect on haemoglobin levels. The threshold for depression of haemoglobin levels is estimated to be 50 and 40  $\mu\text{g/dL}$  in adults and infants respectively, while the threshold for anaemia as defined by a hematocrit value less than 35% of the normal range was determined to be  $\geq 20$   $\mu\text{g/dL}$  in young children (ATSDR, 2005).

### **Cardiovascular effects**

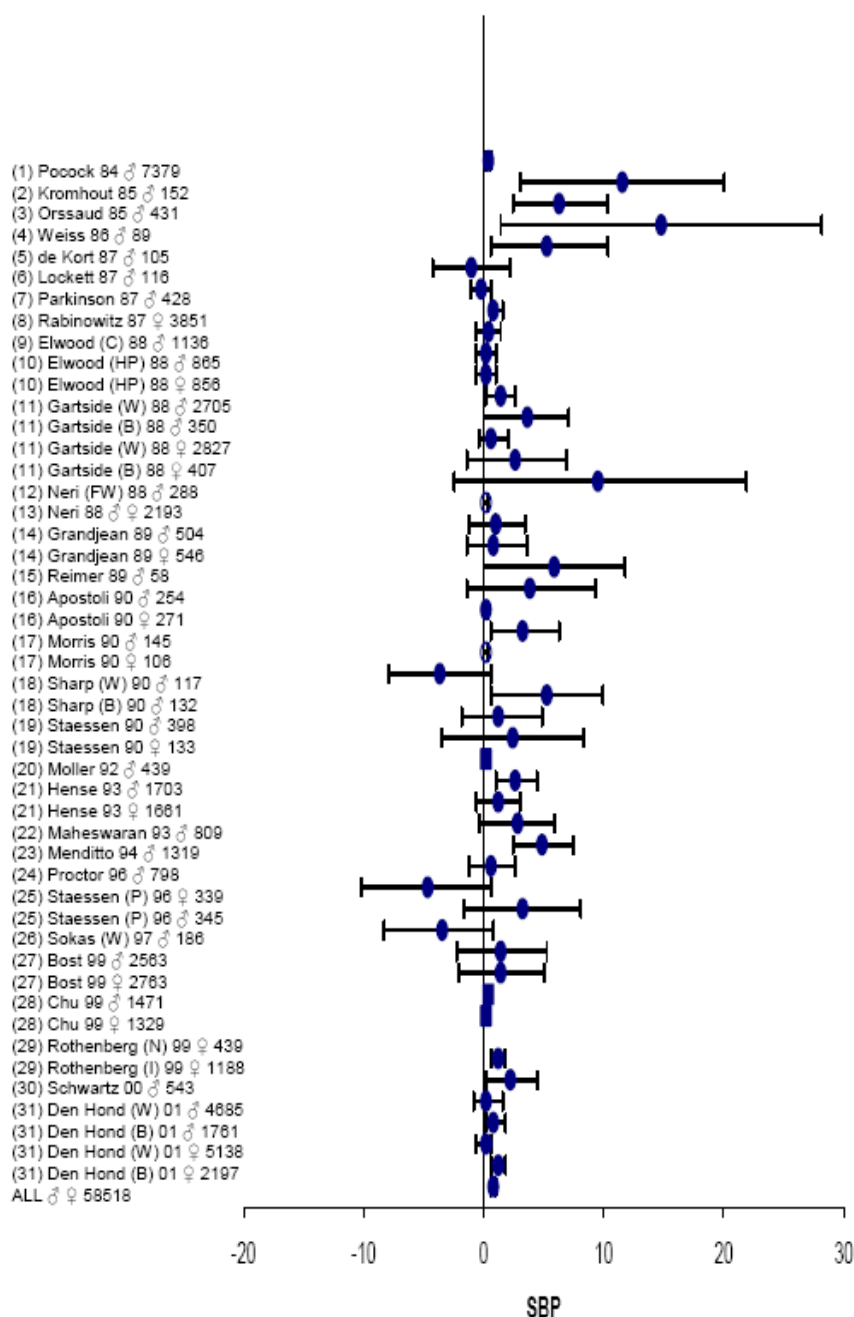
Lead has been shown to produce various cardiovascular effects in animals (Vaziri and Sica, 2004\*). The endpoints of greatest concern for humans at low exposures and low PbBs are elevations in systemic blood pressure. Consequently, only this health effect is addressed below, and the reader is referred to the draft ATSDR (2005) for a summary of other cardiovascular changes (e.g. changes in cardiac rhythm).

Numerous epidemiology studies have examined associations between lead exposure (as indicated by PbB or bone lead concentrations) and blood pressure. These include both occupational and general population studies. However, it should be noted that decrements in glomerular filtration rate may contribute to elevations in blood pressure, and elevated blood pressure may predispose people to glomerular disease, and both effects can be confounders and covariables in epidemiology studies.

Longitudinal occupational studies on the possible association between blood pressure and lead exposure have produced mixed results. In a study of 288 foundry workers the average covariate (age and body weight) adjusted regression coefficient (mmHg per  $\mu\text{g/dL}$  blood lead) was 0.210 (standard error [SE], 0.139,  $p = 0.064$ ) for systolic blood pressure and 0.298 (SE, 0.111,  $p < 0.05$ ) for diastolic blood pressure (Neri et al., 1988\*). In a population of 70 Boston policemen covariate adjusted linear regression coefficients (mmHg per  $\mu\text{g/dL}$ ) were determined for high (PbB  $\geq 30$   $\mu\text{g/dL}$ ) and low (PbB 20-29  $\mu\text{g/dL}$ ) exposure. After adjusting for covariates, high PbB was a significant predictor of subsequent elevation in systolic pressure of 1.5–11 mmHg while low PbB was not. Diastolic pressure was unrelated to PbB (Weiss et al., 1986\*, 1988\*). A study of 496 current and former employees of an inorganic lead manufacturing facility (mean age 56 years; mean PbB 4.6  $\mu\text{g/dL}$ ; mean bone lead 14.7  $\mu\text{g/g}$ ) reported that after adjustment for covariates a one standard deviation increase in PbB was associated with a 0.64 mmHg (95% CI 0.14 – 1.14) increase in systolic blood pressure and a 0.009 (95% CI –0.24 – 0.43) increase in diastolic blood pressure. In addition, a one standard deviation increase in tibia bone lead concentration was associated with a 0.73 mmHg (95% CI 0.23 – 1.23) increase in systolic blood pressure and a 0.07 mmHg (95% CI –0.29 – 0.42) increase in diastolic blood pressure. No data on family history of hypertension or dietary information on subjects was available for this study and, thus, these potential confounders could not be evaluated (Glenn et al., 2003).

A substantial number of studies are available in the general population that have also produced mixed results (see Figures 8.1 and 8.2). Summaries of these studies are provided in the draft ATSDR (2005), to which the reader is referred, while a summary of meta-analyses of the data is presented below.

**Figure 8.1 – Change in the diastolic pressure associated with a doubling of the blood lead concentration\***

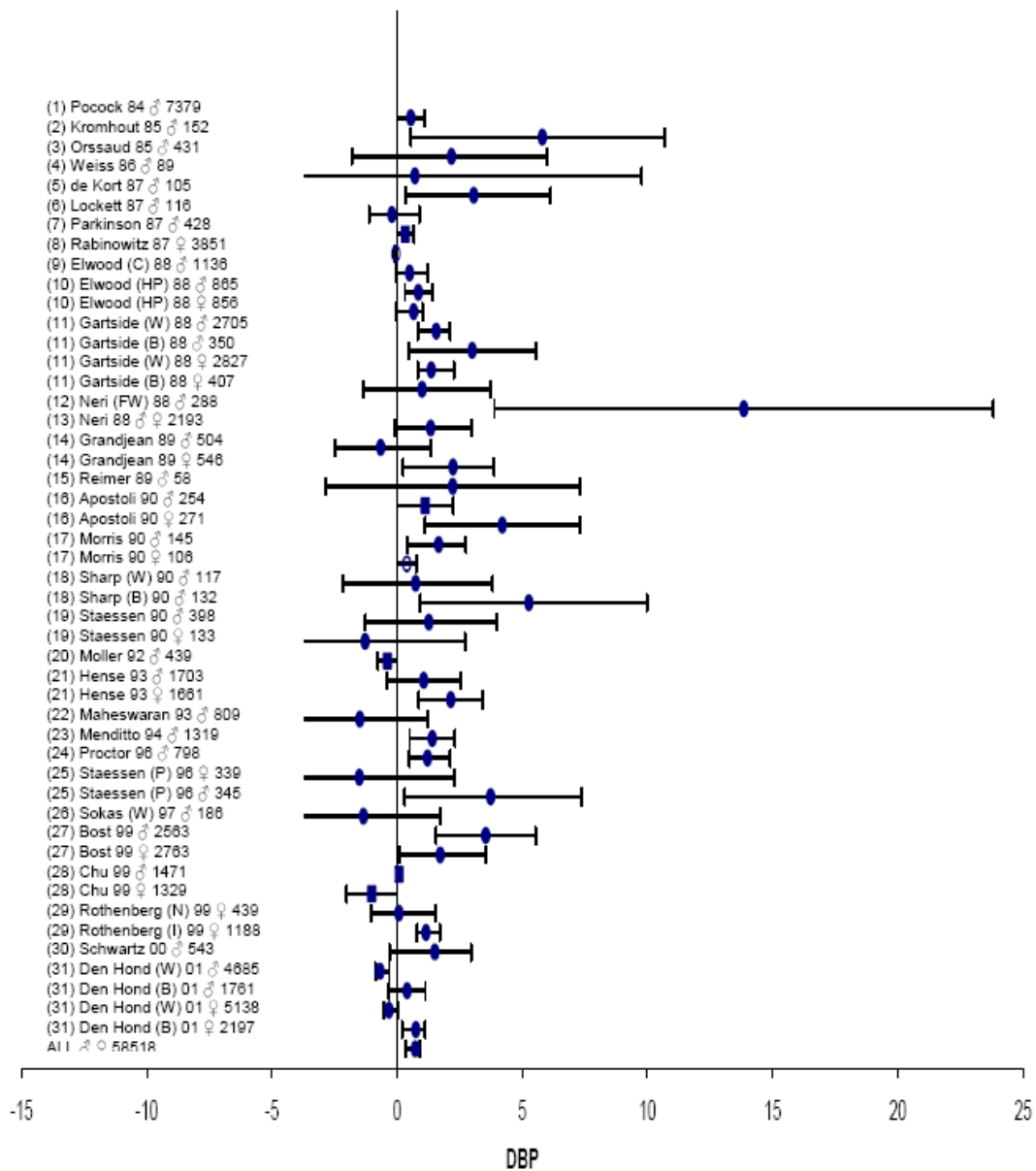


Data were digitised from Nawrot et al., 2002. Circles represent mean (mmHG) of individual groups; squares represent combined groups; open circles represent non-significant associations (plotted as zero). Bars represent 95% confidence limits. See Table 8.1 for more details on study groups.

B = blacks; C = Caerphilly study; CS = civil servants; FW = foundry workers; HP = Welsh Heart Program; I = Immigrants; NI = non-immigrants; P = Public Health and Environmental Exposure to Cadmium Study; W = whites



**Figure 8.2 – Change in the systolic pressure associated with a doubling of the blood lead concentration\***



Data were digitised from Nawrot et al., 2002. Circles represent mean (mmHG) of individual groups; squares represent combined groups; open circles represent non-significant associations (plotted as zero). Bars represent 95% confidence limits. See Table 8.1 for more details on study groups.

B = blacks; C = Caerphilly study; CS = civil servants; FW = foundry workers; HP = Welsh Heart Program; I = Immigrants; NI = non-immigrants; P = Public Health and Environmental Exposure to Cadmium Study; W = whites

**Table 8.1 – Characteristics of the study population in meta-analyses of effects of lead blood on blood pressure**

Reference	No.	Pop.	Men (%)	HT	Age (years)	SBP	DBP	Lead (µg/dL)
Pocock et al. 1984; Sharper et al. 1981	7379	GP	100	Y	49 (40–59)	145	82	15.13 (2.07-66.3) <sup>Ae</sup>
Kromhout 1988; Kromhout et al. 1985	152	GP	100	Y	67 (57–76)	154	92	18.23 (10.77-27.97) <sup>Ae</sup>
Moreau et al. 1982, 1988; Orssaud et al. 1985	431	WC	100	Y	41 (24-55)	131	75	18.23 (8.91-49.94) <sup>Ae</sup>
Weiss et al. 1986, 1988	89	WC	100	Y	47 (30-64)	122	83	24.45 (18.65-29.01) <sup>Mx</sup>
De Kort and Zwennis 1988; de Kort et al. 1987	105	BC	100	N	40 (25-80)	136	83	29.22 (4.35-83.29) <sup>Ae</sup>
Lockett and Arbunkle 1987	116	BC	100	Y	32 (UK)	119	80	37.5 (14.92-95.52) <sup>Ae</sup>
Parkinson et al. 1987	428	BC	100	Y	36 (18-60)	127	80	27.97 (6.01-49.52) <sup>Ae</sup>
Rabinowitz et al. 1987	3851	GP	0	Y	28 (18-38)	121	76	7.04 (3.73-10.15) <sup>Ae</sup>
Elwood et al. 1988a, 1988b <sup>a</sup>	1136	GP	100	Y	56 (49-65)	146	87	12.64 (6.01-26.11) <sup>Gc</sup>
Elwood et al. 1988a, 1988b <sup>b</sup>	1721	GP	50	Y	41 (18-64)	127	78	10.15 (4.56-23.21) <sup>Gc</sup>
Harlan 1988; Harlan et al. 1985; Gartside et al. 1988; Pirkle et al. 1985; Ravnskov 1992 <sup>c</sup>	6289	GP	53	Y	30 (10-74)	127	80	13.47 (2.07-95.93) <sup>Ge</sup>
Neri et al. 1988 <sup>d</sup>	288	BC	100	UK	UK (UK)	UK	UK	45.17 (6.01-65.06) <sup>Ae</sup>

**Table 8.1 – Characteristics of the study population in meta-analyses of effects of lead blood on blood pressure (cont.)**

Reference	No.	Pop.	Men (%)	HT	Age (years)	SBP	DBP	Lead ( $\mu\text{g}/\text{dL}$ )
Neri et al. 1988 <sup>e</sup>	2193	GP	UK	Y	45 (25-65)	UK	UK	23.41 (0-47.03) <sup>Me</sup>
Grandjean et al. 1989, 1991 <sup>f</sup>	1050	GP	48	Y	40 (40-40)	UK	UK	11.6 (3.94-60.09) <sup>Ac</sup>
Reimer and Tittlebach 1989	58	BC	100	UK	32 (UK)	134	81	39.99 (12.85-70.24) <sup>Ac</sup>
Apostoli et al. 1990	525	GP	48	Y	45 (21-60)	132	84	13.05 (2.07-28.18) <sup>Ac</sup>
Morris et al. 1990	251	GP	58	Y	UK (23-79)	UK	UK	7.46 (4.97-38.95) <sup>Ac</sup>
Sharp et al. 1988, 1989, 1990	249	WC	100	N	43 (31-65)	128	83	6.63 (2.07-14.92) <sup>Pe</sup>
Staessen et al. 1984 <sup>g</sup>	531	WC	75	Y	48 (37-58)	126	78	11.4 (4.14-35.22) <sup>Ge</sup>
Moller and Kristensen 1992 <sup>h</sup>	439	GP	100	Y	40 (40-40)	UK	UK	13.68 (4.97-60.09) <sup>Ac</sup>
Hense et al. 1993	3364	GP	51	Y	48 (28-67)	129	80	7.87 (1.24-37.09) <sup>Ac</sup>
Maheswaran et al. 1993	809	BC	100	Y	43 (20-65)	129	84	31.7 (0-98.01) <sup>Ac</sup>
Menditto et al. 1994	1319	GP	100	Y	63 (55-75)	140	84	11.19 (6.22-24.66)
Hu et al. 1996; Proctor et al. 1996 <sup>i</sup>	798	GP	100	Y	66 (43-93)	134	80	5.59 (0.41-35.02) <sup>Pe</sup>
Staesson et al. 1983, 1996a, 1996b <sup>j</sup>	728	GP	49.3	Y	46(20-82)	130	77	9.12 (1.66-72.52) <sup>Ge</sup>
Sokas et al. 1997 <sup>*</sup>	186	BC	99	Y	43 (18-79)	130	85	7.46 (2.07-30.04) <sup>Pe</sup>
Bost et al. 1999	5326	GP	48	Y	48 (16- UK)	135	75	63.82 (?-?) <sup>G</sup>
Chu et al. 1999	2800	GP	53	Y	44 (15-85)	123	78	6.42 (0.41-69) <sup>Ac</sup>

**Table 8.1 – Characteristics of the study population in meta-analyses of effects of lead blood on blood pressure (cont.)**

Reference	No.	Pop.	Men (%)	HT	Age (years)	SBP	DBP	Lead ( $\mu\text{g}/\text{dL}$ )
Rothenberg et al. 1997, 1999a	1627	GP	0	Y	27 (UK)	110	59	2.28 (?-?) <sup>G</sup>
Schwartz and Stewart 2000	543	BC	100	Y	58 (41-73)	128	77	4.56 (1.04-20.1) <sup>Ae</sup>
Den Hond et al. 2001 <sup>k</sup>	13781	GP	53.2	Y	48 (20-90)	125	73	3.11 (0.62-55.94) <sup>Ge</sup>

Source: Nawrot et al. 2002

No.: Number of persons in whom relevant data were available. Pop.: Study population: BC = blue collar workers; GP = sample from general population; WC = white collar employees. Men: Percentage of men. HT: Indicates whether the sample included (Y = yes) or did not include (N = no) hypertensive patients. Age: Mean age or midpoint of age span (range or approximate range given between parentheses). SBP, DBP: Mean systolic and diastolic blood pressures. Lead: Measure of central tendency: A = arithmetic mean, G = geometric mean, M = midpoint of range, P = P<sub>50</sub>(median). The spread of blood lead is given between parentheses: c = P<sub>5</sub>-P<sub>05</sub> interval, P<sub>10</sub>-P<sub>90</sub> interval, or interval equal to 4 times the standard deviation, e = extremes, x = approximate limits of distribution, UK = Unknown

<sup>a</sup>Caerphilly Study

<sup>b</sup>Welsh Heart Program

<sup>c</sup>NHANES (National Health and Nutrition Examination Survey)

<sup>d</sup>foundry workers

<sup>e</sup>Canadian Health Survey

<sup>f</sup>Glostrup Population Study, cross-sectional analysis (19767)

<sup>g</sup>London Civil Servants

<sup>h</sup>Glostrup Population Study, longitudinal analysis (1976-1987)

<sup>i</sup>Normative aging study

<sup>j</sup>PheeCad (Public Health and Environmental Exposure to Cadmium) Study

<sup>k</sup>NHANES III Survey

\*Because of missing information, only the effect in whites is included

A recent meta-analysis of occupational and general population studies (31 in total; total of 58518 subjects) was undertaken (Nawrot et al., 2002), and a summary of the changes in systolic and diastolic pressure as seen in each study, along with the studies included in the analysis and their characteristics is presented in Figures 8.1 and 8.2, and Table 8.1.

In the recent meta-analysis referred to above of studies published between 1980 and 2001, a total of 22 studies were excluded<sup>1</sup> as: reported only on young children (Jhaveri et al., 1979\*; Cramer et al., 1966\*); were case reports (Hu, 2001\*; McAllister et al., 1971\*); recruited less than 50 persons (Sandstead et al., 1970\*) analysis was only performed on less than 50 subjects (Navah et al., 1996\*), estimated exposure from other measurements than the blood lead concentration (de Castro and Medley, 1997\*; Sparrow et al., 1984\*; Staessen et al., 1984\*; Stern, 1996\*; Medeiros and Pllum, 1985\*); and did not provide enough information to compute the association size (Baker et al., 1979\*; Beevers et al., 1980\*; dos Santos et al., 1994\*; Cramer and Dahlberg, 1966\*; Cramer et al., 1974\*; Factor-Litvak et al., 1992\*; Granadillo et al., 1995\*; Ramirez-Cervantes et al., 1978\*; Reimer et al., 1989\*; Schuhmacher et al., 1994\*; Wolf et al., 1995\*).

In the analysis, the results were adjusted for age in all but 4 studies, and most studies took into account additional confounding factors such as body mass index and use of alcohol and medication. Results in men and women were similar. For all studies and both sexes combined a two-fold increase in blood lead concentration was associated with a 1.0 mm Hg rise in systolic pressure (95% CI +0.5 to +1.4 mm Hg;  $p < 0.001$ ) and with a 0.6 mm Hg increase in the diastolic pressure (95% CI +0.4 to +0.8 mm Hg;  $p < 0.001$ ). However, some of the large studies that supported a positive relationship between blood pressure and blood lead based their conclusions on a single blood pressure reading. In contrast 24-hour ambulatory blood pressure recordings are characterised by high reproducibility, are not subject to digit preference or observer bias and minimise the transient rise of a person's blood pressure in respect to the observer. Only one study used this (new) technique of blood pressure measurement (Staessen et al., 1996b) and did not support the hypothesis of a positive relationship between blood pressure and blood lead concentration (Nawrot et al., 2002) Thus, overall, the meta-analysis is not considered to conclusively prove an association between body burden of lead and blood pressure.

The finding of this recent meta-analysis is similar to two other meta-analyses. A meta-analysis of 23 studies published between 1984 and 1993 (33141 subjects) found a 1 mmHg (95% CI, 0.4 – 1.6) increase in systolic blood pressure and a 0.6 mmHg (95% CI 0.2 – 1.0) in diastolic pressure per doubling of PbB (Staessen et al., 1994\*), while a meta-analysis of 15 studies published between 1985 and 1993 found a 1.25 mmHg (95% CI, 0.87 – 1.63) increase in systolic blood pressure per doubling of PbB. Diastolic blood pressure was not reported (Schwartz, 1995\*).

Cohort studies by Gerr et al. (2002\*) and Hu et al. (1991\*) examined the potential effects of childhood exposure to lead on the association between bone lead levels and blood pressure in adulthood. The studies indicate that only high childhood exposures to

---

<sup>1</sup> See Nawrot et al. (2002) for the study references.

lead (e.g. bone lead levels of > 10 µg/g) may contribute to higher blood pressure in adulthood.

Thus, the human data is inconsistent with many studies not detecting an association or significance between blood pressure and low blood lead concentration. Additionally, covariables and confounders are likely to be a potential limitation of many of these studies. Furthermore, although a significant relationship has been reported following meta-analyses of the human data, measurement error may be an important factor in many of the studies that reported a relationship, as 24-hour measurements are more reproducible than single measurements and when such techniques have been employed no significant associations has been found between PbB and blood pressure. Consequently, overall, it is considered that from the available data no casual relationship can be demonstrated in humans between blood pressure and low blood lead concentrations.

### **Renal effects**

Lead nephrotoxicity is characterised by proximal tubular nephropathy, glomerular sclerosis and interstitial fibrosis (Diamond, 2005\*; Goyer, 1989\*; Loghman-Adham, 1997\*). Functional deficits in humans that have been associated with excessive lead exposure include enzymuria, proteinuria, impaired transport of organic anions and glucose, and decreased glomerular filtration rate (as indicated by decreases in creatine clearance or increases in serum creatine concentration). A few studies have identified histopathological effects of renal injury in humans, including intranuclear inclusion bodies and cellular necrosis in the proximal tubule and interstitial fibrosis (Biagini et al., 1977\*; Cramer et al., 1974\*; Wedeen et al., 1975\*, 1979\*).

There are a large number of studies on lead nephropathy in humans. A summary of the studies and endpoints investigated as provided in the draft ATSDR (2005) report is presented in Table 8.2.

The overall dose-effect pattern seen from the studies as provided in the draft ATSDR (2005) is presented in Figure 8.3. The data suggest an increasing severity of nephrotoxicity associated with increasing PbB, with effects on glomerular filtration reported at PbBs below 20 µg/dL, enzymuria and proteinuria becoming evident above 30 µg/dL, and severe deficits in function and pathological changes occurring in association with PbBs exceeding 50 µg/dL. This is consistent with observations made in animal models (ATSDR, 2005).

With regard to reduced glomerular filtration rate (i.e. indicated by decreases in creatinine clearance or increases in serum creatinine concentration) variable results have been observed at low levels (i.e. < 20 µg/dL). In larger studies multi-variate linear regression suggests that decrements in glomerular filtration rate may occur with PbBs < 20 µg/dL and, possibly, below 10 µg/dL (ATSDR, 2005). A summary of such studies as provided in the ATSDR (2005) is presented in Table 8.3.

**Table 8.2 – Selected studies of lead-induced nephrotoxicity in humans<sup>a</sup>**

<b>Reference</b>	<b>Exposure type</b>	<b>Number of subjects</b>	<b>Age (year)</b>	<b>Exposure duration (year)</b>	<b>Blood lead concentration (µg/dL)</b>	<b>Biomarker evaluated</b>
Muntner et al., 2003	Unknown	4831	>20	NA	5	SCr
Hu, 1991	Environmental	22	55	NA	6	CCr
Lin et al., 2001	Unknown	55	57	NA	7	CCr
Staessen et al., 1992	Environmental	1981	48	NA	8	CCr, SCr
Payton et al., 1994	Environmental	744	64	NA	8	CCr
Kim et al., 1996	Unknown	459	57	NA	10	SCr
Staeseen et al., 1990	Environmental	531	48	NA	10	SCr
Bernard et al., 1995	Environmental	154	13	NA	12	UNAG, URBP
Fels et al., 1998	Environmental	62	10	NA	13	SCr, UE, UP, ULMWP
Sonmez et al., 2002 <sup>1</sup>	Occupational	13	32	0.14	25	UNAG
Chia et al., 1994	Occupational	128	28	3	30	UNAG
Chia et al., 1995	Occupational	137	28	>0.5	30	SCr, Sβ <sub>2</sub> µG
Mortada et al., 2001	Occupational	43	33	10	32	SCr, UNAG, UAlb
Gerhardsson et al., 1992	Occupational	100	37–68	14 – 32	32	CCr, SCr, Uβ <sub>2</sub> µG, UNAG
Verberk et al., 1996	Environmental	151	4.6	NA	34	UNAG

**Table 8.2 – Selected studies of lead-induced nephrotoxicity in humans<sup>a</sup> (cont.)**

<b>Reference</b>	<b>Exposure type</b>	<b>Number of subjects</b>	<b>Age (year)</b>	<b>Exposure duration (year)</b>	<b>Blood lead concentration (µg/dL)</b>	<b>Biomarker evaluated</b>
Factor-Litvak et al., 1999	Environmental	394	6	6	35	UP
Omae et al., 1990 <sup>1</sup>	Occupational	165	18–57	0.1 – 26	37	CCr, CUA, U $\beta$ <sub>2</sub> $\mu$ G, C $\beta$ <sub>2</sub> $\mu$ G
Cardozo dos Santos et al., 1994 <sup>1</sup>	Occupational	166	33	4.5	37	SCr, UNAG, UAlb, UP
Wedeen et al., 1975	Occupational	4	36	5 – 8	40	GFR, RPF, TMPAH, HP
Hsiao et al., 2001	Occupational	30	38	13	40	SCr
Huang et al., 2002 <sup>1</sup>	Occupational	40	30	5	41	U $\beta$ <sub>2</sub> $\mu$ G, UP
Fels et al., 1994 <sup>1</sup>	Occupational	81	39	7	42	UP
Pergande et al., 1994	Occupational	82	30	7	42	SCr, UP, UE
Roels et al., 1994	Occupational	76	44	6-36	43	CCr, UNAG
Kumar et al., 1995 <sup>1</sup>	Occupational	22	32.5	NA	43	CCr, U $\beta$ <sub>2</sub> $\mu$ G, UNAG
de Kort et al., 1987	Occupational	53	42	12	47	SCr, BUN
Verschoor et al., 1987	Occupational	155	30-51	<2 - >10	47	UNAG, URPB
Cardenas et al., 1993	Occupational	41	39	14	48	SCr, UP, U $\beta$ <sub>2</sub> $\mu$ G, UNAG, UTBX, UPG

<sup>1</sup> The full reference for these unseen studies are not included in the draft ATSDR (2005) or WHO (1995) Reference list.



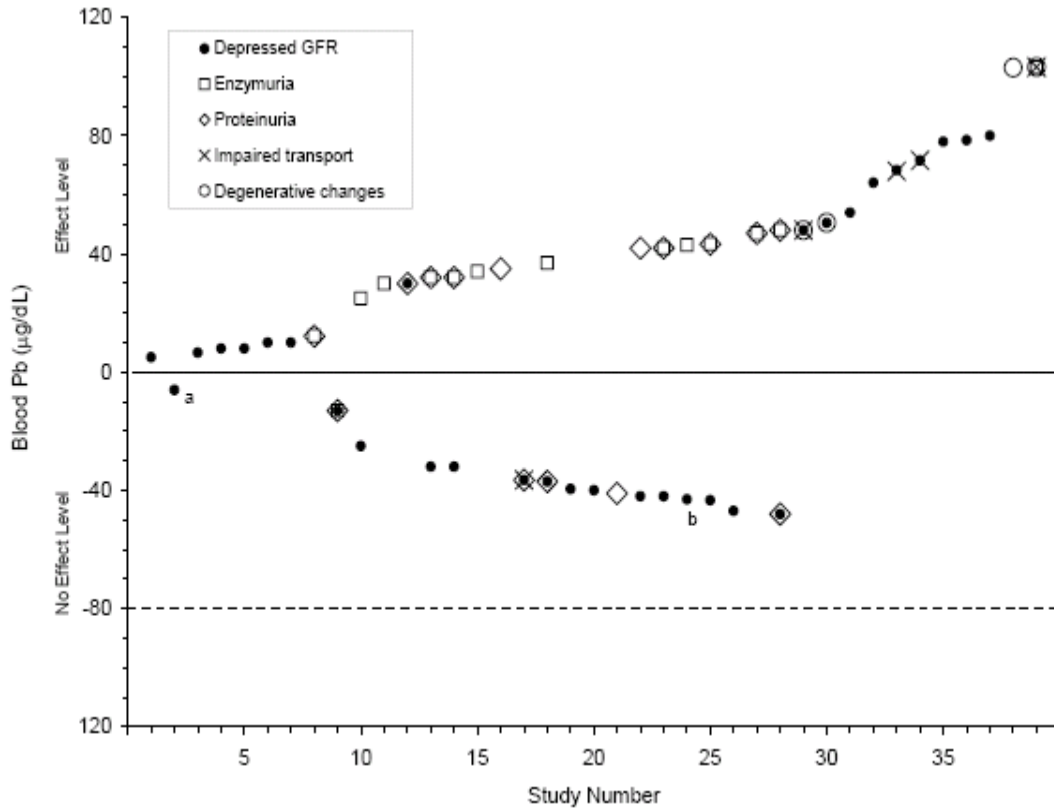
**Table 8.2 – Selected studies of lead-induced nephrotoxicity in humans<sup>a</sup> (cont.)**

Reference	Exposure type	Number of subjects	Age (year)	Exposure duration (year)	Blood lead concentration (µg/dL)	Biomarker evaluated
Wedeen et al., 1975	Occupational	1	40	5	48	GFR, TMPAH, HP
Wedeen et al., 1979	Occupational	15	41	14	51	GFR, HP
Ehrlich et al., 1998	Occupational	382	41	12	54	SCr, SUA
Pinto et al., 1987 <sup>1</sup>	Occupational	52	38	NA	64	SCr
Hong et al., 1980	Occupational	6	35	7	68	GFR, TMG
Wedeen et al., 1975	Occupational	3	28	3-5	72	GFR, RPF, TMPAH, HP
Baker et al., 1979	Occupational	160	29-62	4-31	77	GFR, BUN
Lilis et al., 1968	Occupational	102	32-61	>10	79	GFR, SCr
Lilis et al., 1980 <sup>1</sup>	Occupational	449	NA	12	80	SCr, BUN
Cramer et al., 1974	Occupational	7	45	9	103	GFR, HP
Biagini et al., 1977	Occupational	11	44	12	103	GFR, CPAH, HP

<sup>a</sup>Blood lead concentrations are reported central tendencies

BUN = blood urea nitrogen, CCr = creatinine clearance; C<sub>2</sub>µG = <sub>2</sub>µG clearance, CPAH=p-aminohippurate (PAH) clearance; CUA = uric acid clearance; GFR = glomerular filtration rate; HP = histopathology; S<sub>2</sub>µG = serum <sub>2</sub>µG; SCr = serum creatinine; SUA = serum uric acid; RPF= renal plasma flow; TMG = transport maximum for glucose; TMPAH = transport maximum for PAH; UAlb = urine albumin; U<sub>2</sub>µG = urine <sub>2</sub>µG; UE = urine enzymes; ULMWP = urine low molecular weight proteins; UNAG = urine N-acetyl-D-glucosaminidase; UP = urine protein; UPG = urine prostaglandins; URBP = urine retinol binding protein; UTBX = urine thromboxane

**Figure 8.3 - Indicators of renal function impairment observed at various blood lead concentrations in humans**



Source: derived from Diamond et al., 2005 as presented in draft ATSDR, 2005.

Refer to Table 8.2 for study details

a = Significant increase in serum creatine concentration (Hu, 1991); b= Significant increase in creatine clearance (Roels et al., 1994); GFR = glomerular filtration rate

**Table 8.3 – Summary of dose-response relationships for effects of lead exposure on biomarkers of glomerular filtration rate**

Reference	Exposure	Number of subjects	Mean PbB (range) ( $\mu\text{g/dL}$ )	End point	Change in end point (per 10-fold increase in blood lead)
Payton et al., 1994	Mixed <sup>a</sup>	744 M	8.1 (4 – 26)	CCr (mL/min)	-10 <sup>b</sup>
Staessen et al., 1992	Environmental	1016 F	7.5 (1.7 – 65)	CCr (mL/min)	-10 F <sup>c</sup>
		965 M			-13 M
Kim et al., 1996a	Mixed <sup>a</sup>	459 M	9.9 (0.2 – 54)	SCr (mg/dL)	0.08 <sup>d</sup> 0.14 <sup>e</sup>
Staessen et al., 1990	Environmental	133 F	12 (6 – 35)	SCr (mg/dL)	0.07 M <sup>f</sup>
		398 M			

<sup>a</sup>U.S. Veteran Administrative Aging Study

<sup>b</sup>Partial Regression coefficient, -0.040 ln mL/min creatine clearance per ln  $\mu\text{mol/L}$  blood lead concentration

<sup>c</sup>Partial Regression coefficient, -9.51 mL/min creatine clearance per log  $\mu\text{mol/L}$  blood lead concentration

<sup>d</sup>Partial Regression coefficient, 2.89  $\mu\text{mol/L}$  serum creatine per ln  $\mu\text{mol/L}$  blood lead concentration

<sup>e</sup>In subjects with blood lead concentrations less than 10  $\mu\text{g/dL}$ , the partial regression coefficient was 5.29  $\mu\text{mol/L}$  serum creatine per ln  $\mu\text{mol/L}$  blood lead concentration

<sup>f</sup>Reported 0.6 increase in serum creatine ( $\mu\text{mol/L}$ ) per 25% increase in blood lead concentration ( $\mu\text{mol/L}$ , log-transformed) in males (two subjects with serum creatine concentrations exceeding 180  $\mu\text{mol/L}$  excluded; regression coefficient not reported for females).

CCr = creatinine clearance; F = females; ln = natural logarithm; M = males; PbB = blood lead concentration; SCr = serum creatinine concentration

Thus, table 8.3 provides evidence of a reduced glomerular filtration rate at levels < 20  $\mu\text{g/dL}$ . However, hypertension can be both a confounder in studies of associations between lead exposure and creatinine clearance (Perenger et al., 1993\*) and a covariable with lead exposure (Harlan et al., 1985\*; Muntner et al., 2003; Payton et al., 1994; Pirkle et al., 1985\*; Pocock et al., 1984\*, 1988\*; Tsaih et al., 2004\*; Weiss et al., 1986\*). In the largest study population evaluated (not included in Table 8.1), an association was seen for elevated serum creatine and chronic kidney disease (defined as a glomerular filtration rate < 60 mL/min) in people with hypertension when comparing the highest to lowest quartile of blood lead. Though, after adjustment for age, race and gender no association was seen for elevated serum creatine (OR = 1.09 (95% CI 0.53 – 2.22) or chronic kidney disease (OR = 1.09 95% CI 0.41 – 2.89) among persons without hypertension (Muntner et al., 2003).

Thus, severe deficits in function and pathological changes have been clearly observed with PbBs exceeding 50  $\mu\text{g/dL}$ , enzymuria and proteinuria are evident above 30  $\mu\text{g/dL}$  and there is evidence from some studies based on multivariate linear regression (with

log-transformed PbB) that reduced glomerular filtration rate may occur < 20 µg/dL and possibly < 10 µg/dL. However, mixed results have been seen for effects on indicators of renal functional impairment from 20-50 µg/dL, and for the limited evidence of effects at PbBs < 20 µg/dL there is increasing uncertainty attached to any estimate of affect. Thus, overall, there is evidence of affects occurring at Pbs > 20 µg/dL that may have clinical significance.

### **Summary**

Overall, it is considered that from the available data no causal relationship can be demonstrated in humans between blood pressure and low blood lead concentrations. However in the other most sensitive target organs, the nervous system, haematology system and kidney, effects have been observed that can be attributed to lead at low blood lead concentrations. It is considered that the key lead induced health effect in adults on the nervous system, haematology system and kidney is a reduction in NCV with PbBs > 30 µg/dL, a depression of haemoglobin levels with PbBs > 50 µg/dL and reduced glomerular filtration rate with PbBs > 20µg/dL respectively. Though effects have been reported in each of these target organs at lower PbB levels in adults, either the effects have been inconsistently seen or the toxicological significance of the observed change is uncertain, meaning there is increasing uncertainty attached to any estimate of affect at these lower PbBs.

In children the key health effect observed at low levels of exposure (excluding effects on intellectual development which are reported in section 8.3.4) is anaemia, defined by a hematocrit value less than 35% of the normal range, at PbBs  $\geq$  20 µg/dL.

### **8.1.5 Mutagenicity**

The potential genotoxic effects of lead have been studied in lead workers and the general population, as well as in animals and in vitro systems.

#### **In vitro**

A large number of studies have been conducted in vitro with lead and a wide variety of endpoints assessed. Most of the tests in bacteria have been negative while conflicting results have been observed in mammalian cells. A summary of the in vitro data as presented in the draft ATSDR (2005) is presented in Table 8.4.

#### **Studies in Drosophila**

A negative result was reported in studies in *Drosophila melanogaster* (Ramel and Magnusson, 1979\*; Costa et al., 1988), however, for an insect system such as this there is too little comparative data with mammalian cells and the relevance of findings to the mammalian in vivo system is uncertain (Aardema et al., 1998).

**Table 8.4 Genotoxicity of lead in vitro**

Species (test system)	Endpoint	Results		Reference
		With activation	Without activation	
<i>Salmonella typhimurium</i> (reverse mutation); <i>Escherichia coli</i> (forward mutation, DNA modification); <i>Saccharomyces cerevisia</i> (reverse mutation); <i>Bacillus subtilis</i> (rec assay)	Gene mutation or DNA modification	-	-	Bruce and Heddle, (1979)*; Dunkel et al., (1984)*; Fukunaga et al., (1982)*; Kharab and Singh, (1985); Nestmann et al., (1979); Nishioka, (1975)*; Rosenkranz and Poirier, (1979)*; Simmon, (1979b)*
<i>S. cerevisiae</i>	Gene conversion – or mitotic recombination	-	-	Fukunaga et al., (1982)*; Kharab and Singh, (1985); Nestmann et al., (1979); Simmon, (1979a)*
<i>E. coli</i> RNA polymerase or Avian myetoblastosis DNA polymerase	RNA or DNA synthesis	NA	+	Hoffman and Niyogi, (1977)*; Sirover and Loeb, (1976)*
Chinese hamster ovary cells; Syrian hamster embryo cells	Chromosomal aberration, DNA repair, mitotic disturbance	NA	+	Ariza et al., (1998)*; Bauchinger and Schmid, (1972)*; Costa et al., (1982)*; Robison et al., (1984)*; Zelikoff et al., (1988)
Chinese hamster fibroblasts	Micronuclei	NA	+	Thier et al., (2003)*
Human melanoma cells	Micronuclei	NA	+	Poma et al., (2003)*
Human lymphocytes	Structural chromosomal aberration	NA	+	Beek and Obe, (1974)*;
			-	Deknudt and Deminatti, (1978)*;
			-	Gasiorek and Bauchinger, (1981)*;
			-	Schmid et al., (1972)*
Human lymphocytes	DNA double-strand breaks, DNA-protein cross-links	NA	+	Woźniak and Blasiak, (2003)*
Human lymphocytes	Sister chromatid exchange	NA	-	Beek and Obe, (1975)*;
			+	Niebuhr and Wulf, (1984)*
Human melanoma cells	Sister chromatid exchange	NA	+	Poma et al., (2003)*

- = negative result; + = positive result; DNA = deoxyribonucleic acid; NA = not applicable; RNA = ribonucleic acid

## **In vivo**

A number of occupational studies reported a significant increase in chromosomal aberrations in peripheral lymphocytes in lead workers (group sizes 8–26 workers) with mean PbBs in the range 40–80 ug/dL (Nordenson et al, 1978\*; Schwanitz et al, 1970\*; Forni et al, 1976\*; Al-Hakkak et al, 1986\*; Huang et al, 1988\*). In contrast, negative results have been reported in other studies investigating chromosomal aberrations in lead workers (groups sizes 11–70) with PbBs ranging from <40–120 ug/dL (Maki-Paakkanen et al., 1981\*; O’Riordan and Evans, 1974\*). Though exposure concentrations to lead or other potential agents were not reported in the above studies.

Negative results were also reported for chromosomal aberrations in the general population in 11 adults (Bulma and De France, 1976\*) and 30 children (Bauchinger et al., 1977\*) with PbBs of 40 ug/dL and 12–33 ug/dL respectively. Though again exposure concentrations were not reported.

Similarly, divergent findings have been found for SCEs in lead workers. A statistically significant increase in SCEs was reported in 11 of 23 lead workers who had TWA exposures of 0.19–10.32 mg/m<sup>3</sup> lead in air and a mean PbB of 32.5 ug/dL compared to controls (mean PbB of 4 ug/dL). No statistically significant increase in SCE was seen in the remaining 12 workers who had a mean PbB of 9.3 compared to controls (Wu et al., 2002). An increase in SCEs was also reported in 31 lead workers with a mean PbB of 36 ug/dL (Duydu et al., 2001\*). An increase in SCE frequencies was also reported in lead workers with a mean PbB > 80 ug/dL but not below this value (Huang et al., 1988\*). A slight increase or no increase in the incidence of SCEs was found in 10 lead workers with a PbB range of 29–75 ug/dL (Grandjean et al., 1983\*), while no significant increase in SCEs was seen in 18 lead workers exposed to concentrations of lead in air ranging from 0.05–0.5 mg/m<sup>3</sup> with a mean PbB of 49 ug/dL compared to controls (PbB <10 ug/dL)(Maki-Paakkanen et al., 1981\*).

A statistically significant increase in the incidence of micronuclei in peripheral lymphocytes was seen in a group of 22 lead workers exposed to concentrations of lead in air ranging from 0.13–0.71 mg/m<sup>3</sup> with a mean PbB of 61 ug/dL compared to external and internal control groups (mean PbB 18 ug/dL or 28 ug/dL respectively) (Vaglenov et al., 1998). In a subsequent study in which 103 lead workers were stratified into four exposure levels according to their blood lead levels, very low, moderate, high and very high, workers with moderate exposure and above (i.e. PbBs >25 ug/dL) showed a statistically significant increase in micronuclei frequency compared with controls (Vaglenov et al. 2001).

Significantly elevated levels of DNA breaks in lymphocytes were seen in 37 battery workers exposed to lead compared to un-exposed controls (Fracasso et al., 2002\*). Compared to controls, a statistically significant increase in DNA breaks was also observed in leucocytes from 45 workers exposed to an air lead concentration of 0.004 mg/m<sup>3</sup> who had a mean PbB of 25 ug/dL (Danadevi et al., 2003). DNA damage was also observed in a mice model of lead inhalation, which was seen in the liver and lung following a single 60-minute exposure to 6.8 ug/m<sup>3</sup> lead acetate (Valverde et al., 2002\*). In general subsequent exposure resulted in DNA damage in the lung, liver, and kidney that correlated with length of exposure and lead concentration in the tissue.

A summary of the in vivo data as presented in the draft ATSDR (2005) is presented in Table 8.5.



**Table 8.5 - Genotoxicity of lead in vivo**

Species (test system)	Endpoint	Results	Reference		
Mouse bone marrow, rat bone marrow, mouse leukocyte, monkey lymphocyte, rabbit lymphocyte	Structural chromosomal aberrations or gaps, micronucleus formation; unscheduled DNA synthesis, sister chromatid exchange	±	Bruce and Heddle, (1979)*; Deknudt and Gerber, (1979)*		
		+	Deknudt et al., (1977)*;		
		+	Jacquet and Tachon, (1981)*		
		-	Jacquet et al., (1977)*;		
		-	Muro and Goyer, (1969)*		
		+	Tachi et al., (1985)*		
		-	Willems et al., (1982)*		
		+	Jagetia and Aruna, (1998)		
		Lead workers peripheral lymphocytes	Micronuclei	+	Vaglenov et al., (2001); Vaglenov et al., (1998)
				Lead workers peripheral lymphocytes	DNA damage
Lead workers peripheral lymphocytes	Chromosomal aberration				
		-	Maki-Paakkanen et al., (1981)*		
		+	Nordenson et al., (1978)*		
		-	O'Riordan and Evans, (1974)*		
+	Schwanitz et al., (1975)*; Huang et al., (1988)				
Children, general population	Chromosomal aberration	-	Bauchinger et al., (1977)*		
Adults, general population	Chromosomal aberration	-	Bulsma and De France, (1976)*		
Lead workers peripheral lymphocytes	Sister chromatid exchange	±	Grangjean et al., (1983)*		
		-	Maki-Paakkanen et al., (1981)*		
		+	Huang et al., (1988)*; Duydu et al., (2001)*; Wu et al., (2002)		
Children, general population	Sister chromatid exchange	-	Dalpra et al., (1983)*		
Adults, general population	Altered cell division	+	Bulsma and De France, (1976)*		
Lead workers peripheral lymphocytes	Altered cell division	+	Sarto et al., (1978)*; Schwanitz et al., (1970)*		

- = negative result; + = positive result; ± = inconclusive result; DNA = deoxyribonucleic acid;



Thus, while most of the bacterial studies were negative, mixed results have been seen in mammalian cells in vitro and in human studies. Both negative and positive results have been observed for chromosomal aberrations and SCEs in the peripheral lymphocytes of lead workers while positive results were observed for micronuclei and DNA damage. However, while there is human evidence to suggest that lead is a clastogenic agent, the inconsistent results in studies that were mostly undertaken on small groups of workers with no information on lead exposure levels or exposure to other potentially toxic agents, means that overall no reliable conclusion can be drawn from the available data on the in vivo genotoxic potential of lead.

### 8.1.6 Carcinogenicity

Almost all of the information regarding lead exposure and cancer in humans is derived from studies of lead workers and involves exposure to inorganic lead. The human carcinogenicity data was comprehensively reviewed by the International Agency for Research on Cancer (IARC) in 1980. Furthermore, the overall evaluation of carcinogenicity for lead and lead compounds was updated in 1987 by IARC. The evidence for carcinogenicity in humans was considered inadequate. The summary as provided in the update for human data follows (IARC, 1987).

Three epidemiology studies of workers exposed to lead and lead compounds were reviewed previously (IARC, 1980\*): one on smelters and battery workers in the USA, one on workers exposed to tetraethyl lead in USA, and one on copper smelters in the USA; data on the first of these populations have been updated (Cooper et al., 1985\*). A study on battery workers in the UK is now available (Malcolm and Barnett, 1982\*), and studies of a US lead smelter (Selevan et al., 1985\*) and a Swedish copper smelter (Gerhardsson et al., 1986\*) have been reported. A statistically significant excess of cancers of the digestive system (21 observed, 12.6 expected) was found in the study of battery workers in the UK, spanning 1925 – 1976, although the excess was confined to the years 1963 – 1966 (Malcolm and Barnett, 1982\*). Significant excesses of stomach cancer (34 observed, 20.2 expected) and of respiratory cancers (116 observed, 93.5 expected) were seen in the study of US battery plant workers (Cooper et al., 1985\*), although there was a downward trend in standardized mortality ratio by number of years of employment; in the lead production facilities, the excesses noted for stomach and respiratory cancers were not significant (Cooper et al., 1985\*). A non-significant excess of respiratory cancer (41 observed, 36.9 expected) was reported in one of the studies of smelters (Selevan et al., 1985\*), with 28 observed and 25.7 expected in the group with high exposure to lead. Excesses were also noted in this study for kidney cancer (6 observed, 2.9 expected) and bladder cancer (6 observed, 4.2 expected) (Selevan et al., 1985\*). A small study of workers at a Swedish smelter (Gerhardsson et al., 1986\*) with long-term exposure to lead demonstrated a non-significant excess of lung cancers (8 observed, 5 expected). Two cases of kidney cancer in lead smelter workers have also been reported (Baker et al., 1980\*; Lilis, 1981\*).

The excesses of respiratory cancer in these studies were relatively small, showed no clear-cut trend with length or degree of exposure, and could have been confounded by factors such as smoking or exposure to arsenic.

A study of workers manufacturing tetraethyl lead revealed excesses of respiratory cancer (15 observed, 11.2 expected) and brain cancer (3 observed, 1.6 expected) (Sweeney et al., 1986\*).

More recent evaluations of the UK cohort for gastric cardiac (Cocco et al., 1998a\*) and stomach cancers (Wong and Harris, 2000\*) provide no reliable evidence of an association. Similarly, follow-up studies of the Swedish cohort provide no reliable evidence of an increased age-standardised mortality ratio for cancer that was specifically related to lead exposure (Gerhardsson et al., 1995\*; Lundstrom et al., 1997\*). While an excess of kidney cancer was reported in a follow-up of the US cohort of lead smelter workers (Steenland et al., 1992\*).

Since the 1987 IARC update data from further cohorts have become available. In a study of 20700 Finnish workers exposed to lead, a 1.4 fold increase in overall cancer incidence was observed along with a 1.8 fold increase in lung cancer in workers with a BbB  $\geq$  21 ug/dL, though the overall mortality of the cohort was less than expected (Anttila et al., 1995\*). Furthermore, though an increased risk in cancers of the nervous track, specifically gliomas, was seen in the same cohort, the small number of cases, short follow-up time and low response rate mean no reliable conclusions can be drawn (Anttila et al., 1996\*). In a study of cause-specific mortality among 1388 men at an Italian lead-smelting plant, no significant increase were seen in cancer rates compared with the national mortality rate. Compared to regional mortality rates, bladder, kidney and brain cancers were increased (Cocco et al., 1997\*).

A statistically significant increase in rectal cancer was reported in a study of workers exposed to tetraethyl lead (Fayweather et al., 1997\*). While an analysis of 27060 cases of brain cancer in 24 US states reported that data in men suggests that heavy exposure to lead may be associated with an increase in brain cancer risk (Cocco et al., 1998b\*).

A meta-analysis of lead worker studies found a significant excess risk of overall cancer, stomach cancer, lung cancer, and bladder cancer (Fu and Boffetta, 1995). In contrast a more recent meta-analysis, of 7 cohort<sup>1</sup> studies and 1 nested case-control<sup>2</sup> study in lead smelter or battery workers, concluded that overall there is only weak evidence associating lead with cancer; the most likely candidates being lung cancer, stomach cancer and gliomas (Steenland and Boffetta, 2000). Furthermore, confounding may have occurred in both analyses (e.g. little information was available for occupational exposure to other chemicals), particularly for lung cancer as none of the studies control for smoking.

In the only available study of the general population, using data from the US NHANES II Mortality Study, the relationship between PbB level and all cancer mortality was investigated. The study consisted of 203 deaths (177 men, 86 women) among 3992 whites (1702 men, 1890 women) with an average 13.3 years follow-up. After adjusting for confounding no robust evidence of an increased risk of cancer mortality was seen in men or women or both sexes combined. Additionally, none of the site-specific cancer relative risks were significant, and the authors concluded that individuals with PbB in the range of the NHANES II study (weighted median, 13  $\mu$ g/dL) do not appear to have an increased risk of cancer mortality (Jemal et al., 2002).

Overall, in occupationally exposed persons, the results show only weak evidence for an association of cancer, specifically kidney cancer and gliomas. Additionally, no association was seen in the only study of the general population.

---

<sup>1</sup> Wong and Harris, 2000\* (a smelter worker cohort and battery worker cohort); Cocco et al., 1997\*; Lundstrom et al., 1997\*; Anttila et al., 1995\*; Gerhardsson et al., 1995\*; Steenland et al., 1992\*

<sup>2</sup> Fanning, 1988\*

With regard to the animal data, there have been several experimental studies in rats and mice in which the most common tumour seen following long-term administration of a lead compound in food, drinking water or parental administration was kidney tumours (Moore and Meridith, 1979\*; Azar et al., 1973\*; Van Esch and Kroes, 1969\*). However, while some non-genotoxic mechanisms have been proposed the mechanism of lead-induced carcinogenicity in animals is presently not known (ATSDR, 2005).

### 8.1.7 Fertility

It has long been generally accepted from early literature that lead adversely affects the reproductive process in both man and women. Most recent studies are on occupational groups and general populations living near industrial plants (WHO, 1995). The available evidence is mostly qualitative and dose-effect relationships have not generally been established (WHO, 1995). However, the evidence suggests that moderately high PbBs may result in spontaneous abortion and pre-term delivery in women and alterations in sperm and decreasing fertility in men (ATSDR, 2005).

#### Effects in females

A nested control-case study was carried out within a cohort of 668 pregnant women in Mexico City seeking pregnancy diagnosis at public hospitals. Of the 562 women successfully followed to week 20 of pregnancy, 36 women experienced spontaneous abortion, though 1 case was excluded as abortion was due to an accident. The PbB level in these 35 women ranged from 3.1–29 ug/dL (mean 12.0 ug/dL) and they were matched with 60 controls whose pregnancies survived beyond week 20 on age, hospital, date of enrolment and gestational age at enrolment. PbB levels in controls ranged from 1.3–26 ug/dL (mean 10.1 ug/dL). After adjustment for potential confounders such as medical conditions, reproductive characteristics, socio-demographic variables and life-style factors using a conditional logistic regression model, a statistically significant increased risk of spontaneous abortion was seen for each 1 ug/dL increase in blood lead levels (odds ratio [OR] = 1.13, 95% confidence interval [CI] 1.01 – 1.3) (Borja-Aburto et al., 1999). An increased frequency of spontaneous abortion was also reported in 511 women who worked in a lead smelter in Sweden that responded to a questionnaire. Spontaneous abortion rates were highest in cases where the mother was employed during the pregnancy (13.9%) or had been employed at the smelter before the pregnancy and was living within 10 km of the smelter (17%). However, it is noted that the smelter produced other chemical products and the effects reported may not be attributable to lead alone (Nordstrom et al., 1979\*). Furthermore, no association was found between PbBs and spontaneous abortions in a cohort study of women living in a lead smelter community in South Australia (Baghurst et al., 1987\*). In this study, mean mid-pregnancy PbBs in women living in (531 subjects) or outside of town (171 subjects) were 10.6 and 7.6 ug/dL respectively (Baghurst et al., 1987\*; McMichael et al., 1986\*). Similarly in a prospective study, no significant difference in rates of spontaneous abortion were seen between women living near a smelter in Kosovo, Yugoslavia (304 subjects, mean PbB 15.9 ug/dL) and females living 25 miles away (335 subjects, mean PbB 5.2 ug/dL) (Murphy et al., 1990).

Similarly, limited evidence is available that lead exposure may result in preterm delivery (delivery before the 37<sup>th</sup> week). In the cohort study of Australian women mentioned above, the rate of preterm delivery was significantly higher in women living in town with a mean PbB of 11.2 ug/dL at the time of delivery (5.3%) compared to women living outside of town with a mean PbB of 7.5 ug/dL at the time of delivery

(2.9%) (McMichael et al., 1986\*). Additionally, after adjusting for confounding preterm births were almost three times more frequent in women from Mexico City with umbilical PbB  $\geq 5.1$  ug/dL compared to women with PbB  $< 5.1$  ug/dL. However no dose response was evident: OR = 2.72 95% CI 1.03 – 7.19 for PbB 5.1 – 9.0 ug/dL, OR = 2.82 95% CI 1.13 – 7.02 for PbB 9.1 – 14.9 ug/dL, and OR = 2.60 95% CI 1.01 – 6.71. (Torres Sanchez et al., 1999). In contrast, no evidence of alterations in time to pregnancy or decreased fecundability was seen in a Finish study, where women were categorised as having very low exposure (PbB  $< 10$  ug/dL), low exposure (PbB between 10 and 19 ug/dL), or moderate-to-high exposure (PbB  $\geq 20$  ug/dL) (Sallmen et al., 1995\*).

Experimental studies in animals have showed that high maternal exposure to lead in rats can reduce litter size (WHO, 1995). Treatment of Sprague-Dawley rats with lead in drinking water on gestation days 5 – 21 resulted in 19% incidence of stillbirth compared to 2% observed in the control group. PbBs in the dams and offspring in this experiment were  $> 200$  ug/dL (Ronis et al., 1996\*). In subsequent studies in rats using a similar experimental protocol, treatment on gestation days 5 – 21 resulted in a 28% incidence of stillbirth. The mean PbB level in pups at birth was 197 ug/dL (Ronis et al., 1998\*). Studies are also available in monkeys that have suppressed circulating levels of progesterone, LH, FSH and/or estradiol in monkeys with PbBs of approximately 70 ug/dL (Franks et al., 1989\*) and 35 ug/dL (Foster, 1992). A PbB level of 70 ug/dL in monkeys also resulted in longer and more variable menstrual cycles and shorter menstrual flow (Franks et al., 1989\*).

Thus, conflicting results have been seen in women for effects on fertility, and the evidence is mostly qualitative and dose-effect relationships have not generally been established. However, the evidence suggests that moderately high PbBs may result in spontaneous abortion and pre-term delivery in women. While in experimental studies in rats high maternal exposure to lead (approximately 200 ug/dL) can reduce the number of offspring.

### **Effects in males**

A significant and dose related reduction in fertility (defined as non-occurrence of a marital pregnancy) was seen in a Finish study of 2111 lead workers compared to 681 unexposed men: for PbB of 10 – 20, 21 – 30, 31 – 40, 41- 50 and  $\geq 51$  ug/dL the risk ratio was 1.27 (95% CI 1.08 – 1.51), 1.35 (1.12 – 1.63), 1.37 (1.08 – 1.72), 1.50 (1.08 – 2.02) and 1.90 (1.30 – 2.59) respectively. However, there was no evidence of decreased fertility in couples who had achieved at least one pregnancy (Sallmen et al, 2000). A significant reduction in fertility has been observed in 74 workers with a mean PbB of 46.3 ug/dL compared to controls (Gennart et al, 1992\*) and for duration of exposure in a study of 4256 workers: at a PbB  $\geq 25$ , though the most marked reduction was for the greatest cumulative exposure of  $> 40$  ug/dL (Lin et al., 1996\*). A significant reduction in fertility was also seen in Taiwanese battery workers with PbBs in the range 30-39 ug/dL and  $\geq 40$  ug/dL but not  $\leq 29$  ug/dL (Shiau et al., 2004\*). In contrast, there was no association between occupational exposure to lead and reduced fertility in a multi-country (Belgium, Finland, Italy and England) study of 638 men with mean PbB from 29.3–37.5 ug/dL though most were  $< 50$  ug/dL (Joffe et al., 2003\*). Additionally, no effect on fertility was seen in battery workers exposed to lead among 229 men in a French study (Coste et al., 1991\*) and 1349 men in a Danish study (Bonde and Kolstad, 1997\*).

A number of studies have reported an association between lead exposure and sperm quality. An increased risk of reductions in normal sperm, total sperm, functional maturity of sperm and/or semen volume has been observed in: a study of 81 lead smelter workers with PbB  $\geq 40$  ug/dL compared to PbBs  $< 15$  ug/dL (Alexander et al., 1996\*); a study of 150 workers with a mean PbB of 52.8 ug/dL compared to 41 or 23 ug/dL (Lancranjan et al., 1975\*); a Swedish study of battery workers with a mean PbB of 45 ug/dL compared to about 21 ug/dL (Wildt et al., 1983\*); and a cross-sectional survey of 503 European workers with a PbB  $> 50$  ug/dL compared to men with a mean PbB of 10-50 ug/dL (no dose related linear trend was observed from 10–50 ug/dL) and controls with a PbB of  $\leq 10$  ug/dL (Bonde et al., 2002). Detrimental changes in sperm quality have also been seen in studies that assessed  $< 40$  men/study (Lerda, 1992\*; Assennato et al., 1987\*; Chowdhury et al., 1986\*).

Experimental studies in rats have shown adverse sperm effects at relatively high PbBs (Barratt et al., 1989\*; Hsu et al., 1998a\*, 1998b\*). In contrast, no significant effect on sperm parameters have been seen in male rats exposed to lead from gestation to 9 months after weaning (PbBs ranged from 4.5 to 67 ug/dL) (Fowler et al., 1980\*), and in a lifetime study in monkeys who had a mean PbB of 56 ug/dL (Foster et al., 1996\*). However, numerous studies in animals have reported testicular effects (i.e. histopathological changes) following exposure to lead (Bizarro et al., 2003\*; Adhikari et al., 2001\*; Batra et al., 2001\*). For example, electron microscopic analysis revealed disruption of the general architecture of the seminiferous tubule that involved Sertoli cells, basal lamina, and spermatids in lifetime and infancy (postnatal day 0 to 400) exposed monkeys with PbBs of approximately 35 ug/dL and  $< 1.0$  ug/dL respectively compared to controls and post-infancy (postnatal day 300 to 10 years of age) exposed animals with PbBs of  $< 1.0$  ug/dL and approximately 35 ug/dL respectively (Foster et al., 1998\*).

Thus, although the evidence for reduced fertility is not conclusive, overall, it appears that a threshold in men could be in the PbB range of 30–40 ug/dL. Furthermore, most of the available studies suggest that sperm quality may be affected in men with mean PbB  $> 40$  ug/dL but not in men with lower PbBs. While in experimental animals adverse sperm effects have been reported in rats at relatively high PbBs.

### **8.1.8 Developmental toxicity**

Lead is known to cause developmental toxicity in humans, a casual relationship between human exposure to lead and subsequent developmental toxic effects in the progeny having been established.

Prenatally, there is some evidence of an association between lead exposure and low birth weights in infants. A significant reduction in birth weight associated with prenatal (maternal) PbB levels was reported in an American prospective study after adjustment for covariates (Bornschein et al., 1989), while in a cross-sectional study a significant association was reported between reduced birth weight and placental lead concentration (Ward et al., 1987\*). In an Australian prospective study, the proportion of pregnancies resulting in low birth weights was more than twice as high in a group of women living in a smelter town (Port Pirie, Australia) with a mean PbB of 10.4 ug/dL than in women outside the town with a mean PbB of 5.5 ug/dL (McMichael et al., 1986\*). However, multiple regression analysis of the data showed no significant association between low birth weight and maternal PbB level. Additionally, other studies have not shown a

significant association between lead exposure and low birth weight (Murphy et al, 1990; Ernhart et al, 1986\*).

Similarly, while some studies have reported an association between decreased length of gestation in women with PbBs greater than 23 ug/dL (Moore et al., 1982\*), 12 ug/dL (Dietrich et al., 1986\*), or 15 ug/dL (McMichael et al., 1986\*), no decreases in gestational length has been observed in other studies in women with elevated PbB levels (Bellinger et al., 1991\*; Graziano, 1990\*).

However, increased attention in recent years has been on the effect of low-level exposure to lead and intellectual development of babies and young children, for which an extensive database is available. A comprehensive review of this data was recently undertaken by the Australian Institute of Health and Welfare (AIHW, 1996a, b) and is presently being undertaken by the US Department of Health and Human Services (ATSDR, 2005). Overall, both assessments concur that blood levels greater than 10 ug/dL can affect intellectual development (and lead has also caused neurobehavioral alterations in developing animals at PbBs similar to those reported in children [ATSDR, 2005]). However, although there are reports of neurobehavioral deficits in children associated with PbBs < 10 ug/dL it is considered that there is increasing uncertainty attached to any estimate of affect.

Consequently, as the postnatal hazard of lead is well known and documented, a summary of the studies is not presented here. Instead the reader is referred to the 1996 AIHW review and the 2005 draft ATSDR review for a comprehensive summary of the available data.

At higher levels of lead exposure than those observed to affect intellectual development, effects on peripheral nerve function have been documented in children. Frank peripheral neuropathy has been observed in children at PbBs of 60–136 ug/dL (Erenberg et al., 1974\*). While for nerve conduction velocity (NCV) studies mixed results have been observed. Although re-analysis of the data from children living near a smelter in Kellogg, Idaho revealed evidence for of a threshold for peroneal NCV at PbBs of 20–30 ug/dL (Schwartz et al., 1988\*), no significant difference was seen for NCV in peroneal nerves in young adults living near a lead smelter in Silver Valley, Idaho whose mean childhood exposure was determined to be 45 ug/dL. Similarly, several studies of associations between lead exposure and hearing thresholds in children have been reported, with mixed results. However, high-level exposure to lead is known to produce encephalopathy in children. Numerous studies clearly show that childhood lead poisoning with encephalopathy results in a greatly increased incidence of permanent neurological and cognitive impairments (ATSDR, 2005).

Thus, lead is a known developmental toxicant in both humans and experimental animals. In humans the critical health effect seen at low levels of exposure is intellectual development (IQ) of babies and young children, for which it has been reliably determined from meta-analyses conducted on cross-sectional studies or a combination of cross-sectional and prospective studies that PbBs greater than 10 ug/dL can result in an IQ decline. Furthermore, while there are some reports of neurobehavioral deficits at PbBs < 10 ug/dL there is increasing uncertainty attached to any estimate of affect.

## 8.2 Health effects of specific lead compounds

This section summarises the available information on the human health effects of the declared lead compounds used in industrial surface coating and/or inks. So as to compare the toxicity pattern of lead and lead compounds data is only provided for those endpoints as addressed in Section 1 (i.e. for repeat dose toxicity only information on effects on the nervous system, the haematological system, the cardiovascular system, and the kidney are presented). However, most studies have focused on the health effects of lead itself, and in human studies exposure to specific lead compound was often not reported. Thus, the availability of information on the specific health effects of lead compounds used in industrial surface coatings and inks are generally dependent on how widely these compounds have been used to study lead toxicity in animal studies. As can be seen below, the information available on specific lead compounds used in industrial surface coating and inks varies widely.

With the exception of specific lead chromium compounds, the available information on these specific lead compounds taken together indicates qualitatively a similar pattern of toxicity to that reported for lead in Section 1 (e.g. neurological effects following repeated exposure and conflicting results in mammalian cells in vitro for mutagenicity). There is evidence to suggest that quantitative differences seen between lead compounds for health effects is due to differences in physical chemical properties (e.g. solubility; Zelikoff et al., 1988 and/or bioavailability; Dieter et al., 1993) related to the ions comprising different lead compounds. This is most apparent for certain lead chromium compounds, where the observed carcinogenicity has been attributed to the chromate ion (see below).

### 8.2.1 Lead monoxide

In a 5-month study 14 female albino Swiss mice fed 10 mg/kg bw/day lead monoxide powder daily in the diet. The study also included a control group of 6 animals. Animals were sacrificed at the end of the treatment period and haematology investigations undertaken. Compared to controls, a statistically significant decrease was seen in haemoglobin percentage (10%), packed cell volume (18%), erythrocytes (11%) and leucocytes (8%), along with an increase in the incidence of irregular shaped erythrocytes. The results of this study indicate the development of anaemia in mice following ingestion of lead monoxide (Bazzaz et al., 1989).

A study was conducted to investigate potential for lead monoxide alloy to induce dominant lethal mutations in Balb-C Swiss mice. From weaning, male mice (17–22 per group) ingested 0, 0.096 and 0.194 mg lead alloy in the diet daily for 35 days and were then mated with 2 untreated females until evidence of mating was obtained or up to 1 week. Approximately half the females were allowed to deliver and half sacrificed on gestation day 14. The results showed pregnancy rates to be comparable between the groups. However, compared to controls a dose-related decrease was seen in the mean number of live pups (6.50, 5.36 and 5.10) that was statistically significant at the mid dose and above. Additionally, a decrease was seen in the mean number of total implants (7.73, 7.67 and 6.59) and viable implants (7.67, 7.27 and 6.12) that was statistically significant at the top dose, together with an increase in mean non-viable implants (0.06, 0.40 and 0.47) that was statistically significant at the mid dose and above (Al-Hakkak et al., 1988). However, the methodological limitations of this study such as only 2 lead monoxide dose levels, no mating intervals, only 15–17 pregnant females per group to

determine the number of viable and non-viable foetuses and absence of a positive control, limits the significance that can be attached to this positive finding.

Five males per group from the above study were also sacrificed after treatment and the effect of lead monoxide alloy on spermatogenesis investigated. No statistically significant decrease in the numbers of spermatogonia was seen. Compared to controls, a dose-related statistically significant decrease was observed in the number of spermatocytes (24%) at the top dose. Although statistically significant changes were seen in spermatocyte substages a dose response relationship was not evident. However, this study does provide evidence of reduced spermatogenic activity after ingestion of lead monoxide alloy (Al-Hakkak et al., 1988).

### **8.2.2 Lead sulfate**

In 3 cases of lead poisoning in workers in a plastics factory exposed to powdered lead sulfate stabilizer, PbBs at the time of diagnosis were 159, 114, and 108 µg/dL. The worker with a PbB of 159 µg/dL had clinical symptoms of abdominal pain, constipation, normocytic anaemia, fatigue and reversible azotemia, and renal insufficiency as indicated by an increased serum creatine level and reduced creatinine clearance rate. In this worker, bone lead concentrations were determined to be 102, 219, and 182 ppm in the tibia, calcaneous and patella respectively (Coyle et al., 2005).

Parkinsonian symptoms were observed in 7 of 9 postal workers exposed to lead sulfate batteries over a period of up to 30 years. However workers were also exposed to sulphuric acid and other sulphur compounds. Consequently, the high prevalence and cause of Parkinsonism in these workers is unclear (Kuhn et al., 1998).

Blood samples from two non-smoking volunteers with no history of exposure to heavy metal compounds were exposed to 250–2000 µM lead sulfate for 72 hours without metabolic activation. Compared to controls a small but statistically significant dose-related increase (30%–56%) was seen in the incidence of chromatid exchanges. It is not reported whether a second independent experiment was conducted to confirm the observed finding (Sahu et al., 1989). Thus, the absence of a positive control and information on the reproducibility of the finding means the results of this study are considered inconclusive. In the same study (Sahu et al., 1989) using the same methodology and dose levels, a greater increase in sister chromatid exchanges was seen for lead nitrate treatment with exposure to UV light than treatment with the metal alone, though the significance of this finding is unclear.

### **8.2.3 Lead molybdate**

No data are available.

### **8.2.4 Lead octanoate**

No data are available.

### **8.2.5 Lead 2-ethylhexanoate**

No data are available.



### 8.2.6 Lead oxide

The effect of exposure to lead oxide on the reproductive system and fertility of male rats and their offspring was investigated. Male Sprague-Dawley rats (20 per group) were exposed whole body to 0 or 5 mg/m<sup>3</sup> lead monoxide dust for 6 hours/day, 5 days/week for 10 weeks. At the end of treatment 8 animals per group were sacrificed and plasma follicle stimulating hormone, luteinizing hormone and testosterone levels determined along with the reproductive organs being examined. Fertility was assessed in 12 animals per group that were mated with unexposed females (2 per male): half of these dams were sacrificed on gestation day 20 and half were allowed to deliver. Twelve offspring from the treated and control animals were killed when 90-days old and hormone levels determined and reproductive organs examined. The fertility of the offspring was also determined in 12 males from treated animals and 24 males from controls being mated with unexposed females (3 per male). Pregnant females were sacrificed on day 20.

Hormone levels in exposed males and male offspring were similar to controls. Compared to controls a statistically significant decrease in seminal vesicle weight (23%) was seen in treated males. In contrast no effect on organ weight was seen in male offspring. Conversely, a statistically significant decrease in the number of spermatozoa from the cauda epididymides (12%) was seen in male offspring but absent in treated males. Furthermore, the mobility and morphology of spermatozoa were similar to controls in treated animals and offspring, while no histopathological changes to the reproductive organs or effects on fertility were seen in treated males or male offspring (Pinon-Lataillade et al., 1993). Thus, overall, this study with a single lead oxide exposure level of 5 mg/m<sup>3</sup> provides no robust evidence of an adverse effect on reproductive organs or function in treated males and their male offspring.

In a study in Sprague-Dawley rats, the effect of exposure to lead oxide during pregnancy on the reproductive system and fertility of offspring was investigated. Pregnant animals (9-10 per group) were exposed whole body to 0 or 5 mg/m<sup>3</sup> lead oxide dust for 6 hours/day on gestation days 2, 3, 6, 7, 8, 9, 10, 13, 14, 15, 16, 17 and 20. On day 21 of gestation approximately half the dams were sacrificed and follicle stimulating hormone, luteinizing hormone and testosterone levels determined, while the remaining animals were allowed to deliver. Reproductive organs were examined in 13 and 10 males from the exposed and control group respectively when 90 days old. The fertility of the offspring was also determined with 10–12 males from each group being mated with 20–24 females similarly exposed females. Pregnant females were sacrificed on day 20.

Hormone levels in exposed dams were similar to controls, as were the histopathology and weight of male reproductive organs, and mobility and morphology of spermatozoa, in offspring. Furthermore, no effect was seen on fertility in the offspring. Thus, this study provides no evidence of an effect on reproductive function in male offspring following exposure of dams to 5 mg/m<sup>3</sup> lead oxide (Coffigny et al., 1994).

### 8.2.7 Lead nitrate

The possibility of an effect of lead nitrate on microtubule formation in a non-physiological, cell free system was investigated. Microtubule proteins were purified from pig brain and incubated with lead nitrate in the absence of metabolic activation for

20 minutes. The ability of the proteins to form microtubules in this system was then assayed spectrophotometrically. It is reported the polymerisation of microtubule proteins was inhibited at concentrations of 20–60  $\mu\text{M}$  and the tubulin tended to aggregate at  $> 70 \mu\text{M}$ . Though examination of tubules incubated with up to 60  $\mu\text{M}$  by electron microscopy showed no significant difference in morphology from controls. Additionally, a microtubule gliding assay indicated an inhibition in gliding velocities of microtubules at  $> 10 \mu\text{M}$  with a 50% decrease seen at approximately 50  $\mu\text{M}$  and almost complete inhibition at 500  $\mu\text{M}$  (Bonacker et al., 2005). Thus, the data from this study shows that in a crude cell free system, lead nitrate disrupts microtubule formation.

In an assay to detect both mitotic gene conversion and reverse mutation, negative results were obtained when *Saccharomyces cerevisiae* strain D7 were exposed to lead nitrate at concentrations up to 0.06 mg/ml (concentration in  $\mu\text{g}/\text{plate}$  not stated) in the absence of metabolic activation. At the top dose cell survival was reduced by 84% compared to controls. It is not reported whether a positive control was used in this study (Kharab and Singh, 1985).

In a gene mutation test Chinese hamster V79 cells were exposed to 50–2000  $\mu\text{M}$  lead nitrate for 5 days without metabolic activation. At the top dose cell survival was reduced by 44% compared to controls. Positive results were clearly seen at 500  $\mu\text{M}$  and above (pooled results from two separate experiments) though the increase in mutation frequency were not dose related. The positive control gave clear increases in mutation frequency (Zelikoff et al., 1998).

In a sister chromatid exchange assay, Chinese hamster V79 cells (*hprt* locus) were exposed to 500–3000  $\mu\text{M}$  lead nitrate for 24 hours without metabolic activation. Compared to controls no significant increase was in the incidence of chromatid exchanges in pooled results from two separate experiments. No information on cytotoxicity is reported. The positive control gave a result that confirmed the validity of the test (Zelikoff et al., 1998).

Female ICR Swiss Webster mice (5 per group) received a single intravenous (i.v.) injection of 0, 12.5, 25, 50 or 75 mg/kg bw lead nitrate on day 9 of gestation and then sacrificed on day 18 of gestation. Compared to controls, G-banding analysis showed a slight increase in chromosomal aberrations, mainly deletions, in fetal liver and maternal bone marrow cells at 12.5 mg/kg and above. However, no statistical analysis was undertaken, the increases was not dose related and only 20–40 cells were analysed per tissue per dose level (Nayak et al., 1989). Consequently, no reliable conclusions on the genotoxic potential of lead nitrate can be reached from this non-standard study that also used i.v. administration which is not a relevant route of human exposure.

An in vivo neutral comet assay leukocytes from male Swiss mice (5 per group) were isolated from blood samples taken 24, 48 and 72 hours and 1 and 2 weeks after receiving a single oral (gavage) dose of 0, 0.7, 1.4, 2.8, 5.6, 11.2, 22.4, 44.8 and 89.6 mg/kg bw lead nitrate. Dose selection was based on a range finding study. Compared to controls, a statistically significant increase in comet tail length (i.e. DNA damage) was seen in leukocytes (50 per sample per mouse) in all treatment groups at all sample times. The increase in DNA damage was not dose-related (213%–366% at 24 hours), though DNA damage was seen to decrease after 72 hours in all lead nitrate treatment groups. The positive control gave a clear and statistically significant increase in DNA damage (Devi et al., 2000). However, only limited significance can be attached to the

finding of an increase in DNA damage that is not dose related in a non-regulatory study (i.e. no OECD test guideline exists).

In a micronucleus assay, groups of Swiss mice (6 per sex per dose per sampling time) received a single intraperitoneal injection of 0, 0.625, 1.25, 2.5, 5, 10, 20, 40 or 80 mg/kg bw/day lead nitrate. Bone marrow was sampled at, 12, 24 and 36 hours post-treatment. The study did not include a positive control. Compared to negative controls, the mean ratios of polychromatic to normochromatic erythrocytes (P/N ratios) were increased in both sexes in all treatment groups at 12, 24 and 36 hours. While this increase was statistically significant it was not dose related. Similarly, an increase in micronuclei was seen in both sexes from 0.625 mg/kg bw/day that was often statistically significant but not dose related at 12 (12% to 146% in males, -26% to 88% in females) and 24 hours (-16% to 94%, -59% to 51%). (Jagetia and Aruna, 1998). Consequently, the observation of an increase in micronuclei that is not dose related in the absence of a positive control limits the value of this study.

In a study investigating free radical oxidation and damage to the lungs in offspring, 3 pregnant female rats received 200 mg/kg bw lead nitrate on days 5 and 12 of gestation while 5 control dams received distilled water only. Offspring were killed when 40 days old (i.e. 18 treatment and 42 control animals) and histological examination of the lungs undertaken with free radical oxidation determined by chemiluminescence. Chemiluminescence analysis revealed a statistically significant increase in free radical oxidation in treated animals compared to controls. This was due to activation of lipid peroxidation and decrease in antioxidant antiradical activity of the lungs (data not presented here). However, examination of the lungs revealed no morphological differences between the offspring of treated and control animals (Lebed'ko and Ryzhavskii, 2005).

### **8.2.8 Lead naphthenate**

In an old and poorly reported study the oral LD<sub>50</sub> in rats for lead naphthenate was stated to be 5100 mg/kg bw and that deaths appeared to result from gastrointestinal disturbances. No further details are available (Rockford, 1955).

In an old and briefly reported feeding study, groups of 20, 5 and 5 male rats (strain not reported) received 20 daily doses of 1% lead naphthenate in peanut oil, peanut oil only or no treatment over a 4-week period respectively. At the end of the study histopathological examination was undertaken on 5 treatment, 2 vehicle control and 2 un-treated animals. No deaths or clinical signs of toxicity were seen, and body weight gain was similar between the 3 'treatment' groups. Additionally, no treatment related changes were reported at necropsy. No further details are available (Rockford, 1955).

### **8.2.9 Lead peroxide**

No data are available.

### **8.2.10 Lead carbonate (white lead)**

In a study undertaken to investigate the potential additive or synergistic effects of exposure to lead and trichloroethylene, groups of 10 males Sprague-Dawley rats received 0 or 2000 mg/kg bw/day lead carbonate by gavage for 9 days. Neurotoxic effects were evaluated through functional observational battery (FOB) tests. In lead

carbonate treated animals clinical signs of toxicity were observed, and effects were seen on many of the FOB parameters that indicated a neurotoxic effect. The mean PbBs in these animals was 423 µg/dL. Additionally, at necropsy, histopathological changes were seen in the kidney (number of animals not reported) along with necrosis in the stomach (7 animals) and testes (4 animals) (Nunes et al, 2001).

In a study to investigate neurotoxicity in suckling pups, approximately 100 pregnant female Long Evan rats received a diet containing 4% lead carbonate post partum. Offspring (number not reported) were sacrificed up to 71 days of age, having received 4% lead carbonate in the diet after cessation of suckling, and macroscopic and microscopic examination of the central nervous system undertaken. The level of the lead in the milk was reported to be 4.59 mg% of this metal. Retardation in growth was seen in offspring. No further details available. At 23–29 days of age approximately 90% of the offspring developed paraplegia for up to 2 weeks, with about 85%–90% of these animals dying. During the period of paraplegia in the suckling rat, histopathological changes were seen in the cerebellum indicative of encephalo-myelopathy (Pentschew and Garro, 1966).

#### 8.2.11 Lead chrome 1244

No data are available.

#### 8.2.12 Lead chromate

In a briefly reported Ames test, *Salmonella typhimurium* strains TA98, TA 100, TA1535, TA1537 and TA1538 were exposed to lead chromate at concentrations up to 400 µg/plate in the presence and absence of metabolic activation (S9). A positive response was detected in strains TA98, TA1537 and TA1538 with and without S9. Additionally in the same study a positive result was seen in a recombination assay in *Saccharomyces cerevisiae* strain D5 without S9 while a negative result was seen in a mitotic gene conversion assay in *Escherichia coli* strain K-12/343/113 without S9. Positive controls in all three assays gave results that confirmed the validity of the test (Nestmann et al., 1979).

Chinese hamster ovary cells were exposed to 1–10 µM lead chromate for 24 hours and DNA damage investigated by an alkaline or neutral filter elution method. Cells irradiated with x-ray radiation served as an internal standard/positive control. A dose-dependent increase in DNA single strand breaks and DNA-protein crosslinks were observed immediately after treatment with 1 µM and above. Unlike DNA-protein crosslinks, DNA strand breaks were absent 24 hours later. No DNA double-strand breaks were seen. Cytotoxicity was seen at the top dose: 10% cloning efficiency compared to 50% and 94% at 5 and 1 µM respectively (Xu et al., 1992).

In a neutral comet assay in human lung fibroblast WTHBF-6 cells were exposed to lead chromate at concentrations of 0, 0.1, 0.5, 1.0 and 5 µg/cm<sup>2</sup>. The top dose had been determined to cause 8% relative survival in cells compared to controls. A dose dependent increase in DNA double strand breaks was observed, as indicated by a statistically significant increase in comet tail length at 5 µg/cm<sup>2</sup> and a statistically significant and dose related increase in tail integrated intensity ratio (50%–109%) at 0.5 µg/cm<sup>2</sup> and above. In this study chromosomal damage was also measured in 1000 metaphases per dose level and a statistically significant and dose related increase in the

percentage of metaphases with damage seen at 0.1 µg/cm<sup>2</sup> and above: 9%, 23% and 34% compared to 2 % in controls (Xie et al., 2005).

The carcinogenicity data for chromium and chromium compounds was comprehensively reviewed by the International Agency for Research on Cancer (IARC) in 1980. Furthermore, the overall evaluation of carcinogenicity for chromium and chromium compounds was updated in 1987 by IARC. The evidence for carcinogenicity of hexavalent chromium compounds, such as lead chromate, in humans was considered sufficient. An overview of the summary provided in the update for human data follows (IARC, 1987).

*“An increased incidence of lung cancer has been observed among workers in both the bichromate-producing industry and chromate-pigment manufacturing. There is evidence of a similar risk among chromium platers and chromium alloy workers. However, a clear distinction between the relative carcinogenicity of chromium compounds of different oxidation states or solubilities has been difficult to achieve.*

*Recent studies of chromate-pigment makers and users (Dalager et al., 1980\*; Satoh et al., 1981\*; Alderson et al., 1981\*; Bertazzi et al., 1981\*; Sheffet et al., 1982\*; Korallus et al., 1982\*; Frentzel-Beyme, 1983\*; Langard and Vigander, 1983\*; Davies, 1984\*), chrome platers (Franchini et al., 1983\*), welders (Sjogren, 1980\*; Becker et al., 1985\*; Stern, 1983\*; Hernberg et al., 1983\*; Newhouse et al., 1985\*) and chrome-alloy foundry workers (Cornell and Landis, 1984\*) have shed some light on this problem. For chromate-pigment makers and users, respiratory cancer excesses have usually been found. Chromium pigments are usually hexavalent and commonly include zinc, lead or strontium chromate. Chrome platers have also been found to have excess lung cancer. Stainless-steel welding involves the greatest exposure to hexavalent chromium.”*

Additionally, IARC considered the evidence for carcinogenicity to animals to be sufficient for hexavalent chromium compounds (IARC, 1987).

The hexavalent chromium compound lead chromate is considered to have a significant carcinogenic potential as reflected in its present classification in the Hazardous Substances Information System (HSIS) (see section 10).

### **8.2.13 Lead chromate oxide**

Lead chromate oxide is a hexavalent chromium compound. As stated previously, IARC considered that the evidence for carcinogenicity of hexavalent chromium compounds in both humans and animals was sufficient (see comments made above for lead chromate). However, lead chromate oxide is not specifically listed in the Office of the Australian Safety & Compensation Council's (ASCC) *List of Designated Hazardous Substances*. Consequently, its classification in the HSIS is within the generic classification of lead compounds that are not classified for carcinogenicity (see section 10).

### **8.2.14 Lead chromate molybdate sulfate red**

In a briefly reported study investigating the mutagenic activity of several chromium compounds in the Ames test (*Salmonella typhimurium* strain TA100 only) chromium orange (a synonym for lead chromate molybdate sulphate red) was tested up to 160 µg/plate. In water a negative result was seen in the presence and absence of metabolic

activation while when suspended in sodium hydroxide it was reported to be clearly mutagenic. Furthermore, in the same study a statistically significant increase in the incidence of sister chromatid exchanges was reported for 0.1 µg/ml chromium orange following 30 hours incubation (Venier et al., 1985). However no further details are available and thus, limited significance can be attached to this finding.

Lead chromate molybdate sulphate red is a hexavalent chromium compound. As stated previously, IARC considered that the evidence for carcinogenicity of hexavalent chromium compounds in both humans and animals was sufficient (see comments made above for lead chromate). Lead chromate molybdate sulphate red is considered to have a significant carcinogenic potential as reflected in its present classification in the HSIS (see section 10).

### **8.2.15 Lead sulfo-chromate**

Lead sulfo-chromate is a hexavalent chromium compound. As stated previously, IARC considered that the evidence for carcinogenicity of hexavalent chromium compounds in both humans and animals was sufficient (see comments made above for lead chromate). Lead sulfo-chromate is considered to have a significant carcinogenic potential as reflected in its present classification in the HSIS (see section 10).

## 9. Risk Characterisation

There are no Australian measured exposure data available for manufacture, importation, formulation and use of the declared lead compounds in industrial surface coatings and inks. Exposure levels during spray painting were estimated using the EASE model. The margin of exposure (MOE) approach for risk characterisation was not undertaken, as animal data were not reviewed in detail and a NOAEL was not identified for health effects in animals.

In this section data on health hazards are analysed with regard to estimated exposure levels of relevance to human exposures. The resultant qualitative risk characterisation provides a basis for risk management strategies. Environmental risk is not considered in this assessment.

### 9.1 Occupational health risk

#### 9.1.1 Occupational exposure

Exposure to lead compounds in the workplace varies depending on the particular activity, quantities of lead compounds to which workers are exposed, the engineering controls and use of PPE. Engineering controls and PPE can reduce exposure and hence risks associated with lead compounds. PPE is however an adjunct to engineering controls and not a substitute.

All lead compounds used as pigments in industrial surface coatings and inks in Australia are imported either as pigment powder, concentrated liquid bases/pastes, finished powder coating products or in finished liquid products.

Industrial surface coating products formulated in Australia using the declared lead compounds are in liquid form. Powder coatings containing lead are not formulated in Australia. As the bulk of formulation involves use of lead chromates, workers may be exposed to both lead and chromates. During formulation of liquid products, exposure to lead and chromates can occur during debagging, transferring formulated product for packaging and distribution, and in maintenance operations. NICNAS obtained data on the formulation process from 7 medium to large companies. This sample may not be entirely representative of all the formulators, however, based on information provided exposure to lead compounds during formulation is likely to be low.

During use of industrial surface coatings, dermal exposure can occur by direct contact of solid or solutions with the skin or where workers come into contact with contaminated surfaces. Inhalation exposure can arise from dusts or aerosols. Stripping back of surfaces (e.g. car panels) prior to re-coating can expose workers via the dermal and inhalation routes to dusts of unknown composition. No data were provided on which elements of the hierarchy of controls (section 10.1) are used for surface preparation and removal of dusts.

In situations where old lead-based paint is being stripped from a surface, exposure to lead in the surface preparation is high, given that exposure is to the particulate form as opposed to liquid form. Similarly there is potential for high exposure during spray

painting as exposure is to aerosols or fine mists, though the degree of exposure depends on engineering controls and PPE used. A spray painter wearing a full suit and positive pressure hood in a dedicated spray booth would have low exposure.

Though the NOHSC spray painting guidance recommends use of wetted rags or wet vacuuming to clean up dusts this, and cleanup of paint contaminated equipment, will lead to the generating of wastes contaminated with hazardous substances.

### **9.1.2 Critical health effects**

#### **Acute effects**

Acute exposure to lead can cause encephalopathy and gastrointestinal effects. Symptoms include ataxia, convulsions and coma and GI effects, such as abdominal pain, anorexia, nausea and vomiting are reported.

#### **Effects from repeated exposure**

In adults, lead encephalopathy is the most severe neurological effect though it is generally only observed at high dose levels. Occupational exposures leading to blood lead levels between 40 to 120  $\mu\text{g}/\text{dL}$  have been reported to be associated with symptoms such as malaise, forgetfulness, irritability, headache, fatigue, paresthesia and impotence. General performance on cognitive and visual/motor coordination tasks have been impaired in lead workers. Neurophysiological effects occur consistently in workers with blood lead levels exceeding 40  $\mu\text{g}/\text{dL}$  and there are data to indicate that nerve conduction velocities are affected at these same doses.

Lead exposure induces anaemia by inhibition of haem synthesis and decreasing erythrocyte life span. In occupationally exposed adults the threshold for decreased haemoglobin levels was found to be a blood lead level of 40  $\mu\text{g}/\text{dL}$ . Reduced glomerular filtration rates are seen in lead workers with blood lead levels greater than 50  $\mu\text{g}/\text{dL}$ . There is evidence of an increased incidence of spontaneous abortion in females working at a lead smelter. Rates were highest where the mother was employed at the smelter during pregnancy. In males a significant reduction in fertility was seen in lead workers compared with unexposed men, although there are other data where no association between lead exposure and fertility was found. No cardiovascular effects or carcinogenicity arise from occupational exposure to lead. For the lead chromates however there is sufficient evidence for their carcinogenicity in humans due to the hexavalent chromium moiety.

### **9.1.3 Risk estimate(s)**

#### **Risks from physicochemical hazards**

No data are available on the flammability and explosive properties of the declared lead compounds. Lead nitrate is listed in the *Australian Code for the Transport of Dangerous Goods by Road and Rail* (FORS, 1988) (the ADG Code) where it is noted that mixtures of the chemical with combustible material are readily ignited and burn fiercely.



## Acute health risks

Acute effects in humans arise either from accidental or intentional ingestion of lead compounds. Skin, eye and respiratory irritation or skin and respiratory sensitisation are not reported as acute effects of lead. The risk of gastrointestinal and neurological effects during importation of the declared lead compounds is very low, given the low potential for exposure, except in cases of accidental spills.

Exposure and therefore risk of acute health effects during formulation of lead-based paints and inks is expected to be low because ingestion is unlikely and local engineering controls and use of PPE would limit inhalation of dusts.

During use, particularly spray painting, there is potential for dermal and inhalation exposure to liquids and aerosols if adequate engineering controls are not in place and PPE are not employed, however in the usual situation, risk of acute health effects is expected to be low because of engineering controls and use of PPE.

## Health risks from repeated exposure

Human data from repeated exposure to lead exposure are expressed in terms of blood lead levels rather than as a NOAEL. The following discussion therefore focuses on threshold levels of lead in blood for the various endpoints. The critical effect from exposure to lead is neurological. This is in the form of encephalopathy and neuropsychological changes. There are equivocal data regarding threshold lead levels however neurological effects are consistently seen in workers with blood lead levels greater than 40 µg/dL.

Reduced fertility in women manifesting as spontaneous abortion is seen in some cohort studies but not others and a threshold blood lead level is not determined. In male lead workers however, reduced fertility occurs at a threshold blood lead level of 30–40 µg/dL.

Exposure can result from ingestion, dermal contact with dusts or fluids or inhalation of dusts and aerosols. Dermal absorption of lead is expected to be minimal, as described in section 7, the dermal route is the least efficient method of absorption, though the actual proportion of an applied dose absorbed varies with the compound.

Potential risk of health effects due to repeated exposure during importation of lead compounds is low. This activity is not listed in the schedules to the *National Standard for the Control of Inorganic Lead at Work* (NOHSC, 1994). It would be unlikely that activities associated with import of the declared lead compounds, would present risk of adverse health effects from lead.

Formulation of industrial surface coatings and inks using lead compounds is a batch process with controls varying from fully automatic to manual handling. Repeated exposure during formulation, at workplaces where the process is manual with minimal controls, would lead to a risk of adverse health effects from exposure to lead. Work involving formulation of lead-based surface coatings would fit the definition in paragraph (a) of Schedule 1 of *National Standard for the Control of Inorganic Lead at Work* (NOHSC, 1994):

“(a) Any work which exposes a person to lead dust in air or lead fumes arising from the manufacture or handling of dry lead compounds, except galena (lead sulphide) when its character or composition remains unchanged.”

As formulation would be considered a ‘lead process’ and there is potential for exposure to lead where manual processes are involved using powdered compounds, there is a risk of long term health effects arising from inhalation of dusts.

Use of lead-based surface coatings in a spray painting situation involves not only application but prior surface preparation. For repair of old parts or items this could mean stripping paint which contains lead. Use including surface preparation would be considered a ‘lead process’ and during end use of industrial surface coatings the risk of adverse health effects from exposure to lead may be high.

Risk is likely to be reduced at workplaces with appropriate engineering controls and use of PPE. Of the 6000 panel shops in Australia, controls are in place at some workplaces to prevent exposure but a number of panel shops have little or no controls. The risk of adverse effects from exposure to lead, during end use of industrial surface coatings, will be high at places with minimal engineering controls as workers would have to rely on PPE which may or may not be available and if available may not be worn, or entirely effective.

There is likely to be a low risk of adverse effects from dermal exposure. Dermal exposure would only occur because of spills during mixing of paint or from leaks from the spray gun, however a high risk exists from inhalation of aerosols, dried overspray and surface preparation dusts.

Formulation using pastes is unlikely to expose workers to lead dust so would not fit the above definition. However, under Schedule 2 of the *National Standard for the Control of Inorganic Lead at Work* (NOHSC, 1994) a catch-all provision exists as follows:

“(e) Working in any lead process not listed in Schedule 1.”

This provision would capture the use of pastes in formulation.

## 9.2 Public health risk

Public exposure to lead from industrial surface coatings will occur from renovation of buildings which have been painted with a lead-based paint in the past and DIY panel repair on vehicles painted with lead-based paints. Exposure is also possible from inappropriate use of surface coatings intended for industrial use. Lead has not been a component of domestic surface coatings since the mid 1970s (FASTS, 2002). Inks containing lead compounds are not sold for consumer use.

The actual risk of long term health effects to the public from inappropriate use of industrial surface coatings in the home is not known. The data from NSW Health (Table 6.3) however shows that the number of notifications for blood lead levels above 15 µg/dL up to 15 years of age and after 65 years of age is low compared with the years between, when most people would be working in a non-domestic occupation. The significance of the lead exposure of the general public through domestic activities compared with occupational exposure cannot be determined from this data. It should also be noted that this data is for NSW only and is only representative of persons who have presented for blood testing.

# 10. Risk Management

This chapter discusses current regulatory controls and risk management practices in place to protect workers and the public from exposure to lead during use of industrial lead-based surface coatings and inks. This assessment also reports the elimination strategy by the Australian Paint Manufacturers' Federation, which is currently underway.

## 10.1 Assessment of current control measures

According to the NOHSC *National Model Regulations for the Control of Workplace Hazardous Substances* (NOHSC, 1994), exposure to hazardous substances should be prevented or, where this is not practicable, adequately controlled, so as to minimise risks to health and safety. The NOHSC *National Code of Practice for the Control of Workplace Hazardous Substances* (NOHSC, 1994) provides further guidelines in the form of a hierarchy of control strategies, namely:

- elimination;
- substitution;
- isolation;
- engineering controls;
- safe work practices; and
- personal protective equipment (PPE)

These measures are not mutually exclusive and effective control usually requires a combination of these strategies.

### 10.1.1 Elimination/substitution

Elimination is the removal of a chemical from a process and should be the first option considered in minimising risks to health.

The Australian Paint Manufacturers' Federation (APMF) acts as a primary interface between paint manufacturers, the Governments and the public in regards to issues impacting the manufacture, supply and use of industrial surface coatings. The APMF represents over 98 percent of Australia's coatings manufacturing capacity. Elimination and substitution are being actively pursued by the APMF and its industry members with the objective to eliminate all lead compounds where they continue to be used in industrial surface coatings and inks.

The main use of lead compounds in industrial surface coatings and inks is for pigmentation. Data based on industry surveys and information provided during assessment indicates that about 303 tonnes of lead in its various forms is imported annually for use in industrial surface coatings and 11 tonnes for use in inks (see Tables 5.1, 5.2 & 5.3).

Information on chemicals used as substitutes for lead compounds were provided by some companies that have commenced an elimination program (Table 10.1), but it is not known whether the list is exhaustive. All the substitutes in Table 10.1 are listed on the AICS however, this report has not assessed the hazards of these substitutes nor compared possible hazards of the substitutes with the hazards of lead and the declared lead compounds. The fact that parts of industry are working towards elimination and substitution would indicate that use of lead compounds in industrial surface coatings and inks may not be essential. However, some responders to the joint NICNAS/industry survey indicated that lead compounds were essential, as replacements do not provide the same colour, durability and economics.

**Table 10.1 – Chemicals used as substitutes for lead compounds in industrial surface coatings and inks**

<b>Chemical (AICS Name)</b>	<b>CAS Number</b>
<b><u>Pigments</u></b>	
Butanamide, 2-[(4-methoxy-2-nitrophenyl)azo]-N-(2-methoxyphenyl)-3-oxo-	6528-34-3
Bismuth vanadium oxide	14059-33-7
Pyrrolo[3,4-c]pyrrole-1,4-dione, 3,6-bis(4-chlorophenyl)-2,5-dihydro-	84632-65-5
Butanamide, 2,2'-[(3,3'-dichloro[1,1'-biphenyl]-4,4'-diyl)bis(azo)]bis[N-(4-chloro-2,5-dimethoxyphenyl)-3-oxo-	5567-15-7
2-Naphthalenol, 1-[(2,4-dinitrophenyl)azo]-	3468-63-1
Butanamide, 2,2'-[(3,3'-dichloro[1,1'-biphenyl]-4,4'-diyl)bis(azo)]bis[N-(4-methylphenyl)-3-oxo-	6358-37-8
Butanamide, 2-[(2-methoxy-4-nitrophenyl)azo]-N-(2-methoxyphenyl)-3-oxo-	6358-31-2
Benzoic acid, 2-[[1-[[[(2,3-dihydro-2-oxo-1H-benzimidazol-5-yl)amino]carbonyl]-2-oxopropyl]azo]-	31837-42-0
Butanamide, 2-[(4-chloro-2-nitrophenyl)azo]-N-(2,3-dihydro-2-oxo-1H-benzimidazol-5-yl)-3-oxo-	12236-62-3
3,6-bis[4-(1,1-dimethylethyl)phenyl]-2,5-dihydro-pyrrolo[3,4-c]pyrrole-1,4-dione	84632-59-7
Butanamide, 2,2'-[(3,3'-dimethoxy[1,1'-biphenyl]-4,4'-diyl)bis(azo)]bis[3-oxo-N-phenyl-	6505-28-8
Butanamide, 2,2'-[[3,3'-dichloro[1,1'-biphenyl]-4,4'-diyl]bis(azo)]bis[N-(2,4-dimethylphenyl)-3-oxo-	5102-83-0
2-Naphthalenecarboxamide, 3-hydroxy-N-(2-methylphenyl)-4-[(2,4,5-trichlorophenyl)azo]-	6535-46-2
1H-Isoindol-1-one, 3,3'-(1,4-phenylenediimino)bis[4,5,6,7-tetrachloro-	5590-18-1
Iron hydroxide oxide	20344-49-4
C.I. Reactive Yellow 98	61814-54-8
Iron oxide (Fe <sub>2</sub> O <sub>3</sub> )	1309-37-1
<b><u>Other</u></b>	
Naphthenic acids, zirconium salts	72854-21-8
Octanoic acid, zirconium salt	18312-04-0
Magnesium oxide	1309-48-4

## 10.2 Regulatory controls in Australia for industrial uses

This section outlines key standards, legislation and regulations, which cover both national controls and controls by states and territories in regard to industrial uses of lead-based surface coatings and inks. It is important to note that national standards do not become legally enforceable until they are adopted by the State and Territory governments, and codified in legislation or regulations. Codes of Practice when taken up are generally not mandatory but must be followed unless health and safety can be achieved in other more practicable ways.

Each State and Territory has a principal Occupational Health and Safety (OHS) Act which sets out certain requirements for the workplace. Some activities, such as the use of inorganic lead, warrant specific regulations or codes of practice to address the risks associated with their use. Ultimate control rests with the States and Territories whose legislation is based on the National Framework.

### 10.2.1 National framework

This section outlines the national framework, and the state legislation and regulations.

#### **National Workplace Hazardous Substances Regulatory Package**

In Australia, it is the role of the Commonwealth and State and Territory governments to implement legislation directed towards the effective control of risks arising from the use of inorganic lead in the workplace. The National Occupational Health and Safety Commission, NOHSC, (now known as the Australian Safety and Compensation Council) developed a model workplace hazardous substances regulatory package to assist in achieving a uniform approach to the assessment and control of such risks and recommended essential requirements which may be adopted in State and Territory legislation or regulations. The package consists of the following documents:

*National Model Regulations for the Control of Workplace Hazardous Substances* [NOHSC:1005(1994)]

*National Code of Practice for the Control of Workplace Hazardous Substances* [NOHSC:2007(1994)];

*National Code of Practice for the Preparation of Material Safety Data Sheets* [NOHSC:2011(2003)];

*National Code of Practice for the Labeling of Workplace Substances* [NOHSC:2012(1994)];

*Approved Criteria for Classifying Hazardous Substances* [NOHSC:1008(2004)]; and

*List of Designated Hazardous Substances* [NOHSC:10005(1994)]. (NB this has been replaced by HSIS – discussed below)

These publications are supplemented by additional guidance material and notes:

*National Guidance Material for Spray Painting* (NOHSC 1999)

*Isocyanates (NOHSC 1990)*

*Guidance Note for the Assessment of Health Risks Arising from the Use of Hazardous Substances in the Workplace [NOHSC:3017(1994)]; and*

*Guidance Note for the Control of Workplace Hazardous Substances in the Retail Sector [NOHSC:3018(1994)].*

### **National Standard for the Control of Inorganic Lead at Work (NOHSC, 1994)**

In addition to the package, a more specific standard exists in relation to inorganic lead, namely the *National Standard for the Control of Inorganic Lead at Work* [NOHSC:1012(1994)]. Each jurisdiction gives effect to the national standard to achieve consistency with definitions used in their principal occupational health and safety legislation. NICNAS contacted the various state and territory OHS authorities to determine the extent of uptake of the national standard. Five responses were received, four of which indicated that the standard has been picked up in their legislation. According to the Standard, the major routes of entry of inorganic lead into the body are inhalation and ingestion of dust and fumes during various production and manufacturing processes, termed 'lead processes'. Exposure to lead presents health risks for all people and represents a serious health risk to the unborn child by way of elevated maternal blood lead levels because the toxic effects of lead on the developing embryo/foetus are reached at blood lead levels significantly lower than those for adults (NOHSC 1994).

According to NOHSC, a 'lead risk job' is one in which the blood level of the employee could be expected to rise above 1.45  $\mu\text{mol/L}$  (30  $\mu\text{g/dL}$ ).

Certain safety procedures outlined in the standard include the following:

- Employers must provide applicants for jobs where lead is used with information concerning the health risks and toxic effects associated with exposure.
- Material Safety Data Sheets (MSDS) must be received from the supplier on the first delivery of lead-containing hazardous substances, and they must be readily available for the employee to view.
- All hazardous products containing lead at work should be properly labeled, and these labels should not be defaced, modified or altered in any way
- The employer must also ensure that a register of all lead-containing hazardous products is kept and that this register is available to all employees in danger of lead exposure.
- The employer must provide the employee with an induction before starting work, and at least annually after that. In such sessions, information should be given concerning the health hazards involved in the use of hazardous materials containing lead, as well as the correct use of protective equipment.

- The employer should ensure that “suitable and sufficient” assessment is made as to the extent of the risks to health present in the workplace where there exists a potential for lead exposure.
- All engineering controls, safe work practice and protective equipment in the workplace should be adequately maintained
- Where possible the employer should make sure that the level of lead in the air likely to be inhaled by employees does not exceed the exposure standard (0.15mg/m<sup>3</sup> TWA). When this is not practical, the employer must provide the worker with suitable respiratory protective equipment, and place appropriate warning signs in the lead process area. Lead chromate is the only declared chemical with a specific exposure standard in the HSIS (0.05 mg/m<sup>3</sup> TWA) however the standard of 0.15mg/m<sup>3</sup> TWA applies to the general listing of “lead inorganic dusts and fumes, as Pb” (see table 10.4).
- Employers should ensure that contamination of areas outside the lead process area is avoided, and that employees do not “eat, drink, chew gum, smoke or carry smoking materials in any lead process area”. They should also provide a room away from the lead contaminated areas for eating and drinking, and employees must wash and remove any protective clothing before entering.
- People can be excluded from lead-risk jobs due to personal medical conditions, pregnancy and breast feeding.
- Employers should provide health surveillance for employees working in lead-risk jobs. This should be provided by a suitable authorised medical practitioner, at no extra cost to the employee. The results of these health surveillances should be maintained for 10 years after the date of the last entry.

The Standard provides for biological monitoring of workers, as an important part of this surveillance process. This consists of the measurement of lead in whole blood or packed red cells, sampled as capillary or venous blood as appropriate, and any other biological testing deemed necessary. Results of biological monitoring should be given to the employee as soon as possible after the testing is complete. If the results of the biological monitoring confirm that the employee has a blood lead level that is at or above certain levels the employer must immediately remove the employee from the lead-risk job and arrange for a medical examination. The levels are:

- 2.41 µmol/L (50 µg/dL): for males and females not of reproductive capacity,
- 2.41 µmol/L (50 µg/dL): for males of reproductive capacity,
- 0.97 µmol/L (20 µg/dL): for females of reproductive capacity,
- 0.72 µmol/L (15 µg/dL): for females who are pregnant or breast feeding

If a female employee advises the employer she is pregnant or breast feeding, she should immediately be removed from the lead-risk job. An employer should not allow an employee to return to a lead-risk job until the blood lead level is confirmed to be less than:

- 1.93 µmol/L (40 µg/dL): for males and females not of reproductive capacity,

- 1.93  $\mu\text{mol/L}$  (40  $\mu\text{g/dL}$ ): for males of reproductive capacity,
- 0.48  $\mu\text{mol/L}$  (10  $\mu\text{g/dL}$ ): for females of reproductive capacity, including females who have ceased their pregnancy and are not breast feeding;

and the employee is certified as fit to return back to work. If the results of biological monitoring show that the employee's blood lead level is at or above:

- 1.93  $\mu\text{mol/L}$  (40  $\mu\text{g/dL}$ ): for males and females not of reproductive capacity,
- 1.93  $\mu\text{mol/L}$  (40  $\mu\text{g/dL}$ ): for males of reproductive capacity,
- 0.72  $\mu\text{mol/L}$  (15  $\mu\text{g/dL}$ ): for females of reproductive capacity,

on three consecutive occasions; or at or above:

- 2.41  $\mu\text{mol/L}$  (50  $\mu\text{g/dL}$ ): for males and females not of reproductive capacity,
- 2.41  $\mu\text{mol/L}$  (50  $\mu\text{g/dL}$ ): for males of reproductive capacity,
- 0.97  $\mu\text{mol/L}$  (20  $\mu\text{g/dL}$ ): for females of reproductive capacity,

on a single occasion; an employer shall take action to identify and assess the source of lead exposure and control that lead exposure.

Though it is up to an employer to determine whether a particular job is 'lead risk' some activities are defined in the Standard as a lead process. A lead process is one which there is a requirement to conduct an assessment to determine whether the job is lead risk and if not to ensure it does not become a lead risk job.

Schedule I of the Standard includes:

*“(h) Machine sanding or buffing of surfaces coated with paint containing greater than one per cent by dry weight of lead.*

*“(i) Any process whereby electric arc, oxy-acetylene, oxy gas, plasma arc or a flame is applied, for the purposes of welding, cutting or cleaning, to the surface of any metal which is coated with lead or paint containing greater than one per cent by dry weight of lead.”*

Schedule II of the Standard includes:

*“(c) Spray painting with lead paint containing greater than one per cent by dry weight of lead.”*

Industrial lead-based paints contain > 1% dry weight of lead and the activities quoted from the schedules above would be undertaken in an automotive body repair shop.

The general requirements of the Standard have been substantially taken up by all states and territories, expressed in varying degrees of regulatory detail. This is discussed further under “Australian Standards for Industrial Settings” below.



## National Code of Practice for the Control and Safe Use of Inorganic Lead at Work (NOHSC, 1994a)

This Code of Practice provides a practical guide on how to comply with the *National Standard for the Control of Inorganic Lead at Work*. It does not include any additional requirements. The Code has been taken up, for example, by the ACT and Northern Territory. Victoria has developed its own *Code of Practice for Lead*.

### Hazardous Substances Information System (HSIS)

The Hazardous Substances Information System (HSIS) is an internet database that contains information on hazardous substances that have been classified in accordance with the *Approved Criteria for Classifying Hazardous Substances* [NOHSC:1008(2004)] and National Exposure Standards declared under the *NOHSC Adopted National Exposure Standards for Atmospheric Contaminants in the Occupational Environment* [NOHSC:1003(1995)] or subsequent updates (<http://hsis.ascc.gov.au/Default.aspx>)

The HSIS replaces the NOHSC List of Designated Hazardous Substances and provides the first reference point for importers, manufacturers and suppliers in determining whether a substance has been classified as hazardous. It is an important reference for determining the classification of hazardous substances. Classification and concentration cut-offs for the declared lead compounds under this assessment that have been listed in the HSIS are shown in Table 10.2. Not all the declared chemicals have separate listings in the HSIS, however all inorganic lead compounds are classified as hazardous on the HSIS under the umbrella listing.

**Table 10.2 - HSIS labeling cut-offs for separately listed lead compounds used in industrial surface coatings and inks in Australia**

Chemical Name	HSIS CLASSIFICATION *			HSIS Labeling Cut-Offs
Lead chromate (as Cr)	Carc.	Cat.3;	R40	Conc>=5%: T; R61; R62; R40; R33
	Repr.	Cat.1;	R61	>=1%Conc<5%: T; R61; R40; R33
	Repr.	Cat.3;	R62	>=0.5%Conc<1%: T; R61
	R33			
	N;	R50-53		
Lead sulfo- chromate	Carc.	Cat.3;	R40	Conc>=5%: T; R61; R40; R62; R33
	Repr.	Cat.1;	R61	>=1%Conc<5%: T; R61; R40; R33
	Repr.	Cat.3;	R62	>=0.5%Conc<1%: T; R61
	R33			
	N;	R50-53		
Lead chromate molybdate sulfate red	Carc.	Cat.3;	R40	Conc>=5%: T; R61; R40; R62; R33
	Repr.	Cat.1;	R61	>=1%Conc<5%: T; R61; R40; R33
	Repr.	Cat.3;	R62	>=0.5%Conc<1%: T; R61
	R33			
	N;	R50-53		

\* R33 – danger of cumulative effects

R40 - Limited evidence of a carcinogenic effect.

R 50 - Very toxic to aquatic organisms

R53 - May cause long-term adverse effects in the aquatic environment

R61 - May cause harm to the unborn child.

R62 - Possible risk of impaired fertility.

A concentration cut-off level for a substance in the list represents a level (expressed as a percentage on a weight/weight basis) at and above which that substance must be considered hazardous.

Other inorganic lead compounds are not individually classified in the HSIS however there is a general classification which is shown in Table 10.3. This general classification would apply to the other declared lead compounds.

**Table 10.3 - HSIS labeling cut-offs for general inorganic lead compounds**

<b>Chemical Name</b>	<b>HSIS CLASSIFICATION *</b>		<b>HSIS Labelling Cut-Offs</b>
Lead compounds, with the exception of those elsewhere specified	Repr. Repr. R20/22 R33 N; R50-53	Cat.1; Cat.3;	R61 R62 R33 Conc<5%: T; R61; R20/22; R33 Conc<1%: T; R61; R33

\* R 20/22 - Harmful by inhalation and if swallowed.

NOHSC (now known as ASCC) in 1995 published the *Exposure Standards for Atmospheric Contaminants in the Occupational Environment* (NOHSC, 1995). The standards are updated as required and are searchable through the HSIS database. Exposure standards consider absorption via inhalation. They represent airborne concentrations of individual chemical substances which, according to current knowledge should neither affect the health of nor cause undue discomfort to nearly all workers. Exposure standards do not represent no effect levels which guarantee protection to every worker, it is inevitable that a very small proportion of workers who are exposed to concentrations around or below the exposure standard may suffer mild transitory discomfort and a smaller number may exhibit symptoms of illness. Time Weighted Average (TWA) exposure standards apply to long term exposure of a substance over an eight hour day, for a five day working week, over an entire working life. Table 10.4 outlines the exposure standards for lead.

**Table 10.4 – Exposure standards for lead in Australian workplaces**

<b>Chemical Name</b>	<b>TWA* mg/m<sup>3</sup></b>
Lead chromate	0.05
Lead, inorganic dusts & fumes (as Pb)	0.15

\* Time Weighted Average concentration of the substance over an eight hour working day, for a five day working week.

### **National Guidance Material for Spray Painting (NOHSC 1999)**

Some guidance material is available for spray painting which aims to minimise exposure to hazardous substances.

The National Occupational Health and Safety Commission (now ASCC) has published the *National Guidance Material for Spray Painting* (NOHSC 1999). This document deals with spray painting in general and covers hazard identification, risk assessment, exposure control and health surveillance. The guidance recognises that surface preparation can be a significant source of exposure to hazardous substances and

recommends that a wet rubbing technique be used in surface sanding. Dry sanding is to be avoided unless there are adequate dust extraction systems employed. The guidance recommends that dusts should be cleaned up using wet rags or wet vacuum cleaners and should not be swept unless exhaust ventilation is used.

### **Isocyanates (NOHSC 1990)**

Guidance material has been published by The National Occupational Health and Safety Commission on isocyanates (NOHSC 1990) covering hazard and control. The application of the control measures to avoid exposure of workers to isocyanates would also prevent exposure to the lead component of the surface coating.

### **Guide to Lead Paint Management; Industrial Applications (Standards Australia, 1995)**

This document is very specific in its focus on the management of lead containing paint on industrial structures, however it does provide some information relevant to this assessment on lead containing paint used for industrial purposes. According to this standard, paint is considered to be lead containing if it contains in excess of 0.1% (w/w) lead in the form of lead or lead compounds within the dried film.

The standard outlines what form of action is most appropriate in certain situations where lead-containing paint is found on a structure. After evaluating the site, options include: no painting, overcoating, spot or localized repair, total coating removal and replacement, or demolition and replacement of the structure. When making this decision a variety of factors need to be taken into account, including the condition of the coating, the cost of the procedure, the risk to the public and to the workers involved, environmental risks, and the cleaning and clearing techniques to be used on the completion of the project.

There is also a *Guide to Lead Paint Management: Residential and Commercial Buildings* (AS 4361.2-1998), which contains the same threshold for lead-containing paint. This is discussed further below under “Australian Standards for Consumer Settings”.

Generally Australian Standards developed by Standards Australia do not have regulatory force and only provide a guide, unless the requirements in the standard are adopted in legislation or regulations.

## **10.2.2 State and Territory workplace legislation around Australia**

The national framework has greatly influenced the development of legislation and regulations in each State and Territory. The approach in each jurisdiction is outlined below.

### **Australian Capital Territory**

The primary legislation is the *Occupational Health and Safety Act 1989*, which details responsibilities regarding substances. The *Environment Protection Act 1997* deals with regulated waste, and human health considerations regarding contaminated land. However, neither of these Acts specifically refer to lead. As informed by Workcover ACT, in the absence of specific regulatory provisions, the Territory relies on the

National Code of Practice and National Standard as approved codes and therefore legally enforceable.

### **New South Wales**

The *Occupational Health and Safety Regulation 2001*, made under the *Occupational Health and Safety Act 2000*, limits the acceptable amount of lead in a workers blood to 1.45 µmol/L and also regulates certain work practices using lead. Part 7.6, (regarding Lead processes and Lead Risk Work), places a duty on employers to control risks from lead, and describes relevant types of work in relation to categories of employee. The regulation deals with hazardous substances and includes requirements relating to health surveillance and also sets requirements for spray painting. These requirements are generally consistent with the National Standard core requirements, as discussed in 10.2.1 above.

Under the *Protection of the Environment Operations (Waste) Regulation 2005* waste containing lead or lead compounds transported intra or inter state is to be tracked (Schedule 1). The *Protection of the Environment Operations (General) Regulation 1998* gives effect to the *National Environment Protection (National Pollutant Inventory) Measure* (made 27 February 1998) whereby lead and lead compounds are listed as NPI substances and have reporting requirements ([www.npi.gov.au](http://www.npi.gov.au)).

### **Northern Territory**

The Work Health (Occupational Health and Safety) Regulations 1992, made under the *Work Health Act 1986* cover hazardous substances in workplaces in general and also contains specific provisions regarding lead-based work, requirements regarding spray painting biological monitoring of lead in whole blood of different categories of workers and the removal from work where appropriate and incorporates the blood level standards of the National Standard.

Codes of Practice can be made under the *Work Health Act 1986*, and the Northern Territory has adopted the National Code of Practice.

Lead and lead compounds are also defined as listed wastes under the Waste Management and Pollution Control (Administration) Regulations (Schedule 2).

### **Queensland**

The primary workplace legislation is the *Workplace Health and Safety Act 1995*. This Act provides for regulations, advisory standards and industry codes of practice to be made to manage exposure to identified risks (sections 41-42). The Workplace Health and Safety Regulation 1997, deals specifically with lead and outlines requirements of manufacturers, importers, suppliers, employers and workers, regarding labelling and health surveillance. These import requirements from the National Standard. Further detail is provided by the Workplace Health and Safety (Codes of Practice) Regulation 2005, Schedule 1 – Hazardous Substances Advisory Standard 2003 (now known as a Code of Practice).

Lead waste is defined as regulated waste under the Environmental Protection Regulations 1998, made pursuant to the Environmental Protection Act 1994. Also, the

Environmental Protection (Waste Management) Regulation 2000 defines lead waste as trackable waste.

### **South Australia**

The primary workplace legislation is the *Occupational Health and Safety and Welfare Act 1986*. The Occupational Health and Safety and Welfare Regulations 1995 made under the Act deals with hazardous substances, lead processes in work places and managing health risks of spray painting, but does not specifically refer to lead components in paint.

Lead compounds and solutions are listed wastes under the *Environment Protection Act 1993* (Schedule 1: A - prescribed activities of environmental significance – activities producing listed wastes include spray painting, and B - listed wastes include “lead compounds and solutions”). Lead compounds are similarly listed as wastes under the Development Regulation 1993 (Schedule 22).

### **Tasmania**

The primary workplace legislation is the *Workplace Health and Safety Act 1995*, which provides for Codes of Practice to be made (section 22), and regulations to be made that may prescribe standards that must be complied with regarding substances and the monitoring of health (Schedule 1).

Under the Workplace Health and Safety Regulations 1998 the use of lead carbonate for spray painting or spray coating and the use of material containing more than 0.1% lead in abrasive blasting is prohibited (Schedule 6). Clause 68 regarding lead processes provides “at a workplace, an accountable person must ensure that any process at the workplace involving lead is undertaken in accordance with the National Standard for Control of Inorganic Lead at Work, issued by Worksafe Australia.” However, lead is not listed separately in Schedule 4 which prescribes hazardous substances for which health surveillance is required.

### **Victoria**

The Victorian government enacted the Occupational Health and Safety (Lead) Regulation (2000) under the *Occupational Health and Safety Act 1985*. The regulation regulates the use of inorganic lead, lead metal and lead alloys in workplaces that undertake lead processes, and imposes duties on employers and employees. The regulatory objective is to “protect people against risks to their health associated with the use of lead at workplaces.”

The updated *Code of Practice for Lead 2000*, was developed to provide practical guidance on how to comply with the Occupational Health and Safety (Lead) Regulation 2000. The code mirrors the National Standard and includes:

- • consultation with employees;
- • providing information, instruction and training (including in relation to MSDS and labels);
- • risk assessment and control, including maintaining the controls;

- • ensuring lead exposure standard is not exceeded;
- • lead-risk jobs;
- • medical examinations and biological monitoring; and
- • employees' duties.

The Environmental Protection (Prescribed Waste) Regulations (1998) defines lead and lead compounds as prescribed waste.

### **Western Australia**

The Occupational Safety and Health Regulations 1996, made under the *Occupational Safety and Health Act 1984*, covers hazardous substances in workplaces generally (5.28-5.41) and also contains specific provisions regarding lead-based work (5.53–5.67). The latter relate to information, training, duties, health surveillance and record keeping, consistent with the core elements of the National Standard.

The Occupational Safety and Health Regulations 1996 defines lead and compounds of lead in paint (which are contained in such quantity that lead, calculated as a percentage of the dried material, exceeds 1% by weight) as a toxic paint substance and prohibits use of material containing more than 1% lead for use as abrasive blasting material (Schedule 3.2 and 5.2 respectively). Similar to Tasmania, lead is not listed separately as a substance where health surveillance is required (Schedule 5.3). Clause 5.3 establishes the blood lead level required for a worker to be removed from a workplace, however it is set at a higher threshold than the National Standard (30 µg/dL for females of reproductive capacity, and 60 µg/dL for other employees).

The Environmental Protection (Unauthorised Discharges) Regulations lists lead as a material which must not be discharged into the environment.

## **10.3 Australian Standards for industrial settings**

Standards Australia is recognized through a Memorandum with the Commonwealth government as the peak non-governmental Australian body involved in the development of standards. Relevant standards covering lead are:

### **Guide to Lead Paint Management; Residential and Commercial Buildings (Standards Australia 1998)**

This standard provides guidelines for the management of lead-containing paint on non-industrial structures such as public, residential and commercial buildings. While it does discuss the control of risk of public exposure to lead, it deals only minimally with safety procedures for workers. According to this standard, paint is considered to be lead-containing if it contains in excess of 0.1% (w/w) lead or lead compounds within the dried film

The standard importantly notes that lead in paint only becomes a problem when it is on a friction or impact surface of a building or product, it is deteriorating or it is disturbed by paint removal methods. When the paint is fully intact, it is not considered to pose a threat to public safety.

## 10.4 Voluntary industry controls for industrial settings

There are no general voluntary controls implemented by the paint/surface coatings and inks industries regarding lead-based compounds. A number of companies however have phased out or are currently phasing out the use of lead compounds in their products. Members of the APMF have embarked on a phase out of all lead compounds in industrial surface coatings and inks over about the next three years according to the following schedule:

### Category One

#### *Paint & Coating Types & End Use Applications*

- Auto refinish car collision repair.
- Commercial vehicle and component builders, refurbishers & repairers.
- Aviation (heavy, general & light aviation) builders, refurbishers repairers.

#### *Timing*

- End of December 2008: Cease manufacture and importation of all lead-based paints and coatings used in Category One.
- End of December 2009: Cease distribution, sale and end use of all lead-based paints & coatings used in Category One.

### Category Two

#### *Paint & Coating Types & End Use Applications*

- All other remaining paint & coating types and end use applications (e.g. paints for automotive original equipment- car builders, industrial coatings, aerosol coatings, protective coatings, packaging coatings etc.) and inks.

#### *Timing*

- End of March 2008: Cease manufacture, importation distribution, sale and end use of all lead-based paints & coatings used in Category Two.

A number of guidance documents are published by the APMF for industries involved with surface coatings. These are based on the Coatings Care<sup>®</sup> program which has been adopted by the APMF in Australia and is not specifically aimed at leaded paints but is a general industry program covering manufacturing (including OH&S, occupational and process safety and environmental management); transport and distribution; product stewardship; community responsibility.

Some guidance documents published by the APMF include:

- Guidance note on paint handling areas in panel shops discusses the setting up of separate designated paint mixing areas and weighing areas and procedures to be followed when undertaking those tasks.

- Code of Practice for the Automotive Refinishing Industry (the APMF Code) – this covers safe practices in all areas of a panel beating shop and is not limited to painting. It discusses workshop operations such as surface preparation, welding and painting; personal protective equipment; storage and handling of dangerous goods and hazardous substances.
- Training Manual and Guide for Demonstrators and other Staff when visiting Bodyshops provides checklists in tabular form of various activities in a bodyshop and sets out the hazards, risks and precautions to be taken when engaged in those activities.

The APMF Code (APMF, 2005) only discusses spray painting as a method of application for paints, and not others such as brushing. A number of controls covering different parts of the repair process are specified in the Code. For spray painting these are aimed at reducing exposure to solvents and to isocyanates, when a two pack system is used, though by default, exposure to lead would also be reduced. Specific procedures for lead-based paints are:

- Follow state regulations when using lead-based paints.
- Maintain high standards of hygiene.
- Provide adequate protective clothing, clothes storage and change facilities.

General controls are:

- If painted surfaces are subjected to high temperature (e.g. welding) then respiratory protection must be worn to avoid inhalation of fumes.
- Dry sanding should be under vacuum to minimise dust.
- Protective gloves should be worn during sanding.
- Spraying should be in a confined spray booth with proper ventilation, preferably downdraught

Respiratory protection should be worn when spraying.

### **10.3 Regulatory controls in Australia for consumer goods**

While this assessment focuses on lead compounds used in industrial surface coatings and inks, controls for lead compounds in consumer products is included to provide a complete picture of the regulatory regimen for lead in Australia. Similar to controls in industrial settings as discussed above, it is important to outline the national standards and framework and how this has shaped State and Territory legislation and regulation.

#### **10.3.1 Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP)**

The Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) forms the basis of control of lead compounds in consumer products in Australia. The National Drugs and Poisons Schedule Committee (NDPSC) comprises State and Territory government members and other persons appointed by the federal Minister for Health and Ageing, such as technical experts and representatives of various sectional interests.



The decisions of the NDPSC in relation to the Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) have no force in the Commonwealth law nor in States and Territories until incorporated into the legislation of the relevant jurisdiction. Enforcement of compliance with the SUSDP is the responsibility of those States and Territories where it is incorporated into legislation.

Lead compounds are listed in Schedules 4, 5, 6 and in Appendix I of the SUSDP.

#### **Schedule 4**

The Schedule covers Prescription Only Medicine, or Prescription Animal Remedy. The general description of Schedule 4 drugs and poisons is:

Substances, the use or supply of which should be by or on the order of persons permitted by State or Territory legislation to prescribe and should be available from a pharmacist on prescription.

The Schedule 4 listing states:

LEAD COMPOUNDS for human therapeutic use except when separately specified in these Schedules.

#### **Schedule 5**

The general description of Schedule 5 poisons is:

Substances with a low potential for causing harm, the extent of which can be reduced through the use of appropriate packaging with simple warnings and safety directions on the label.

The Schedule 5 listing states:

LEAD COMPOUNDS in preparations for use in hair cosmetics

#### **Schedule 6**

The general description of Schedule 6 poisons is:

Substances with a moderate potential for causing harm, the extent of which can be reduced through the use of distinctive packaging with strong warnings and safety directions on the label.

The Schedule 6 listing states:

LEAD COMPOUNDS except:

- when included in Schedule 4 or 5;
- in zinc based paints or tinters containing 0.2 per cent or less of lead as an impurity in the zinc and calculated on the non-volatile content of the paint or tinter;
- in other paints or tinters containing 0.1 per cent or less of lead calculated on the non-volatile content of the paint or tinter;
- in preparations for cosmetic use containing 100 mg/kg or less of lead;

- in pencil cores, finger colours, showcard colours, pastels, crayons, poster paints/colours or coloured chalks containing 100 mg/kg or less of lead; or
- in ceramic glazes when labeled with the warning statement:

CAUTION – Harmful if swallowed. Do not use on surfaces which contact food or drink, written in letters not less than 1.5 mm in height. (p 205)

### **Uniform Paint Standard**

Appendix I of the SUSDP is known as the Uniform Paint Standard. This provides control of paints sold to consumers. In paragraph 3, the supply of paints containing lead at a level of greater than 0.1% is prohibited for application to:

1. a roof or for any surface to be used for the collection or storage of potable water; or
2. furniture; or
3. any fence, wall, post, gate, building (interior or exterior), bridge, pylon, pipeline, storage tank or any similar structure; or
4. any premises, equipment or utensils used for the manufacture, processing, preparation, packing or serving of products intended for human or animal consumption

Additionally:

“A person must not manufacture, sell, supply or use a paint for application to toys unless the paint complies with the specification for coating materials contained in Part 3 of the Australian Standard 1647 for Childrens Toys (Safety Requirements).”

### **10.3.2 Australian Standards for consumer settings**

#### **Children’s Toys (Safety Requirements) - Toxicological Requirement (Standards Australia 1995)**

According to this standard, the maximum acceptable lead migration from toy materials, modeling clay or finger paint is 90 milligrams leachable lead per kilogram of toy material. This document was superseded by AS/NZS ISO 8124.3 on 23<sup>rd</sup> May 2003 however AS 1647.3 is still referred to in the SUSDP appendix I.

#### **Safety of toys - Migration of certain elements (Standards Australia 2003)**

This document superseded the above document (AS 1647.3) on 23<sup>rd</sup> May 2003.

According to this standard, the maximum acceptable element migration from toy materials for lead is 90 milligrams per kilogram toy material. This includes migration of lead from toy materials and from parts of toys, except materials not accessible and includes the following types of toy materials:

- Coatings of paints, varnishes, lacquers, printing inks, polymers and similar coatings;

- Polymeric and similar materials, including laminates, whether textile-reinforced or not, but excluding other textiles;
- Paper and paper board, up to a maximum mass per unit area of 400 g/m<sup>2</sup> ;
- Natural or synthetic textiles;
- Glass/ceramic/metallic materials, excepting lead solder when used for electrical connections (see 8.5);
- Other materials, whether mass-coloured or not (e.g. wood, fibreboard, hardboard, bone and leather);
- Materials intended to leave a trace (e.g. the graphite materials in pencils and liquid ink in pens);
- Pliable modeling materials, including modeling clays, and gels;
- Paints to be used as such in the toy including finger paints, varnishes, lacquers, glazing powders and similar materials in solid or liquid form;

For the purposes of this part of ISO 8124, the following criteria are considered appropriate for the categorization of toys which can be sucked, licked or swallowed:

- All intended food/oral contact toys, cosmetic toys and writing instruments categorized as toys;
- Toys intended for children up to six years of age, i.e. all accessible parts and components where there is a probability that those parts or components may come into contact with the mouth.
- Toys and parts of toys which, due to their accessibility, function, mass, size or other characteristics, obviously exclude any hazard due to sucking, licking or swallowing, bearing in mind the normal and foreseeable behaviour of children, are not covered by this part of ISO 8124.

### **National Health and Medical Research Council**

The National Health and Medical Research Council (NH&MRC) published guidelines for lead in blood and ambient air, however the guidelines were rescinded on 31 December 2005.

The NH & MRC in its Australian Drinking Water Guidelines (NH & MRC, 2004) state that the concentration of lead in drinking water should not exceed 0.01 mg/L.

### **10.3.3 Consumer legislation around Australia**

The various pieces of legislation affecting levels of lead permitted under different circumstances around Australia are as follows. The list does not include monetary type legislation such as tax and tariff controls or legislation affecting lead in motor fuels.

#### **Commonwealth**

The Customs (Prohibited Imports) Regulation 1956, Schedule 2 deals with “Goods the importation of which is prohibited unless the permission in writing of the Minister or an authorised person has been granted.” This includes: toys or playthings coated with a

material the non-volatile content of which contains more than 250 mg/kg of lead or lead compounds, calculated as lead; cosmetic products containing more than 250 mg/kg of lead or lead compounds (except products containing more than 250 mg/kg of lead acetate designed for use in hair treatments); money boxes coated with a material that contains more than 250 mg/kg of lead or lead compounds; and pencils or paint brushes coated with a material the non-volatile content of which contains more than 250 mg/kg of lead or lead compounds, calculated as lead.

### **Australian Capital Territory**

The Schedules to the SUSDP are incorporated by the *Poisons and Drugs Act 1978*.

### **New South Wales**

Section 31 of the *Poisons and Therapeutic Goods Act NSW 1966* states that Commonwealth therapeutic goods laws apply as laws of New South Wales (NSW). The provisions of the SUSDP, which are regulations under the Commonwealth *Therapeutic Goods Act 1989*, are directly subsumed into the laws of New South Wales. This includes Appendix I, which is the Uniform Paint Standard. Hence, paints containing more than 0.1% of lead cannot be sold in NSW for use on domestic structures, both internal and external, on toys, furniture, fences, etc. For all other uses, supply of lead-based paint containing more than 0.1% is available for use with certain restrictions. The NSW Poisons List under section 8 of the *Poisons and Therapeutic Goods Act 1966* includes lead-based paints that contain more than 0.1% lead, under Schedule 6. Under section 9, the wholesale supply of poisons can only occur through licenced persons. Also, Schedule 6 poisons must be labelled "POISON" and there are general conditions for their storage. Hence, the regulatory intent for consumer controls is to prohibit the domestic use of lead-based paint in NSW, with industrial use permissible under strict licencing, labelling and storage conditions.

### **Northern Territory**

The SUSDP and its appendices, are expressly incorporated under section 6A of the *Poisons and Dangerous Drugs Act*. Hence, the same domestic and industrial prohibitions and restrictions on the use of lead-based paints apply.

### **Queensland**

The *Public Health Act 2005* defines certain activities involving lead or lead-based paints to be a public health risk. Section 58 states:

*"A person must not use or permit the use of lead in, or for the purposes of, constructing, erecting, altering, extending, improving, renovating or repairing a building or part of a building if the lead is, or may be, easily accessible to children."*

This provision applies to prohibit lead-based paints in buildings accessible by children. Furthermore, Section 60 of the Act incorporates Appendix I of the SUSDP into Queensland law. It stipulates that persons manufacturing, selling, supplying or using paint must comply with the SUSDP standards. Hence, the Uniform Paint Standard applies to prohibit lead for domestic use in Queensland. Furthermore, the Health

(Drugs and Poisons) Regulation 1996 incorporates Schedules 2 to 9 in the SUSDP, meaning that the industrial use of lead-based paints is regulated as it is in NSW and Victoria.

### **South Australia**

Regulation 5 of the Controlled Substances (Poisons) Regulations 1996 expressly incorporates the SUSDP, except as modified by Schedule A of the Act. The provisions affecting lead are not modified and therefore the same restrictions and prohibitions apply relating to the domestic and industrial use of lead-based paints as they do in other states.

### **Tasmania**

Section 14 of the *Poisons Act 1971* incorporates the SUSDP schedules. These are found in the *Poisons List Order (2001)*. Lead-based paints are therefore included in Schedule 6 and the same restrictions are placed on their industrial use as in the other states. To further supplement these provisions, the *Workplace Health and Safety Regulation 1998* prohibits the use of lead carbonate for spray painting or spray coating and also the use of material containing more than 0.1% lead in abrasive blasting.

### **Victoria**

Section 12(2) of the *Drugs, Poisons and Controlled Substances Act 1981* states that the Victorian Poisons Code must include a poisons list and any provisions (including appendices) of the National Standard concerning the labelling, storing, packaging or advertising of poisons and any other interpretative provisions. This means that the SUSDP provisions apply as law in Victoria, which includes Schedules 2 to 9 and the Uniform Paint Standard. Hence, the same lead paint standards apply as in NSW.

### **Western Australia**

The Poisons Regulation 1965 incorporates Appendix I of the SUSDP. Section 33B states:

*“(1) If a paint contains a substance listed in the First, Second or Third Schedule to Appendix I of SUSDP, a person shall not manufacture, sell or use that paint except in accordance with that Appendix.*

*(2) For the purposes of this regulation the interpretation provisions of Part 1 of the SUSDP shall be used to interpret Appendix I of the SUSDP.”*

This essentially subsumes the Uniform Paint Standard into Western Australian law. Thus, paint containing more than 0.1% lead cannot be sold or supplied for domestic purposes in Western Australia. Furthermore, Section 20 of the *Poisons Act 1964* incorporates the SUSDP schedules. Hence the industrial use of lead-based paint is regulated through licensing, storage and labeling requirements as with other states.

### **Discussion**

A number of Standards, Acts, Regulations and Codes exist at the national level and in each State and Territory jurisdiction relating to hazardous substances in general and specifically to lead. While there is significant take up of measures set out in the

National Standard, each jurisdiction works under a slightly different framework. All jurisdictions have a primary occupational health and safety statute, with some jurisdictions having additional safeguards concerning lead in environment protection and waste legislation. It is important to note the different legal status of different types of instruments. Regulations are legally enforceable, whereas Codes of Practice provide advice on how to meet regulatory requirements. Codes are therefore not legally enforceable, but can be used in courts as evidence that legal requirements have or have not been met.

In terms of comparison between controls in an industrial setting and consumer controls, generally the latter are more stringent, and often involve a ban of lead or lead compounds in surface coatings of particular products. There is merit in applying the same more stringent controls to industrial settings to better protect the health and safety of workers.

# 11. Discussion and Conclusions

## 11.1 Importation and use

The declared lead compounds are used in industrial surface coatings and inks as pigments to impart colour to the final product and as driers.

Import is either as powdered compound, in concentrated pigment bases or pastes, or in finished industrial surface coating products (paints or powder coats) or finished inks.

Lead compounds in powder form and pigment powders, bases or pastes are used to manufacture industrial surface coatings and inks. Imported finished industrial surface coating and ink products are not treated further but are used by the end-user in the form they are introduced.

## 11.2 Health hazards

Lead and inorganic lead compound are known to have diverse effects on multiple body systems including neurological, gastrointestinal, reproductive and cardiovascular systems. In the case of some compounds such as the lead chromates toxicity can arise from both the lead and the chromate portions. For example, the IARC considers that there is inadequate evidence for the carcinogenicity of lead in humans but for hexavalent chromium compounds IARC considers there to be sufficient evidence for carcinogenicity in animals.

Few data are available from acute exposure in humans to lead with most data derived from cases of accidental or intentional ingestion of dirt containing lead or lead-based paint. High acute exposure has resulted in encephalopathy, gastrointestinal and renal effects.

Long term exposure to lead induces anaemia from inhibition of haem synthesis and shortening of erythrocyte lifespan. In animals lead has been shown to produce a variety of cardiovascular effects but the endpoint of greatest concern to humans is possible changes in blood pressure. Although there have been numerous epidemiological studies examining the effect of low concentrations of lead on blood pressure (amongst other endpoints) no causal relationship can be demonstrated between blood pressure and low blood lead concentration in humans.

Lead nephrotoxicity is characterised by proximal tubular nephropathy, glomerular sclerosis and interstitial fibrosis. The data suggest an increasing severity of nephrotoxicity with increasing blood lead concentrations.

There is conflicting evidence of the effects of lead on fertility in human and animal females however there are data to suggest that moderately high blood lead levels may result in spontaneous abortion and pre-term delivery. In humans and animals, there is evidence of reduced fertility resulting from an effect on sperm quality.

The most important effects of long term exposure to lead are neurological, including encephalopathy and neurobehavioural changes. Neurological effects are particularly

important in the foetus and in children. Lead is a known developmental toxicant in humans. Blood lead levels greater than 10 µg/dL can result in decline in IQ and neurobehavioural deficits. Peripheral neuropathy has been observed in children at blood levels between 60 and 130 µg/dL. Numerous studies have shown that childhood lead poisoning with encephalopathy results in a greatly increased incidence of permanent neurological and cognitive impairments.

### 11.3 Occupational health and risk

Occupational exposure to lead compounds used in industrial surface coatings and inks can occur during import, formulation of finished industrial surface coating products and inks and during use of industrial surface coatings and inks. The greatest single use of industrial surface coating products containing lead is in the automotive refinishing industry where about 50% of the total import is used. The compounds used in automotive refinishing products are mostly lead chromates so workers in this industry can be exposed to both lead and chromates. The lead chromates are listed in the HSIS and, in addition to the lead hazard, are classified as category 3 carcinogens. Risk of adverse effects from exposure to lead compounds occurs at all stages of use of surface coatings with the highest potential exposure and therefore risk during surface preparation where lead dust is generated. Overseas data report excessive blood lead levels in workers involved in stripping paint from damaged vehicles, compared with workers who are not involved in paint stripping.

It is not known whether current controls to mitigate the risks arising from exposure to lead are effective. Although industry around Australia is required to monitor workers involved in lead risk jobs, no data were available from the states and territories on compliance with this requirement. Additionally it was found that not all states and territories have adopted the NOHSC *National Standard for Control of Inorganic Lead at Work*.

Lead compounds have been removed from domestic surface coatings and substitutes used. Apart from accidental releases, exposure of the general public to surface coatings containing lead however can still occur from renovation of houses painted with a lead-based paint or from inappropriate use of industrial surface coatings in a domestic setting or from DIY car restoration and body repair, where automotive coatings containing lead compounds would be used. Because the lead chromate type compounds are used as pigments in automotive paints, DIY car restoration and body work exposes the public to compounds which are possible carcinogens.

This assessment has found that lead compounds are not essential in industrial surface coatings and inks and that a number of substitutes are available. Use of these substitutes in formulation of industrial surface coatings and inks and use of surface coatings and inks that do not contain lead will avoid the risks associated with the formulation and use of industrial surface coatings and inks that contain lead compounds.



# Appendix A Chemical Identity, Composition and Physical and Chemical Properties

## A1.1 Lead monoxide

This substance is listed on the Australian Inventory of Chemical Substances (AICS) as:	Lead oxide (PbO)
CAS No.:	1317-36-8
Appearance	Litharge - Red solid. Above 489 °C it is yellow  Massicot – Yellow solid.
Synonyms:	C.I. 77577; Lead (II) oxide; Lead(II)oxide yellow; Plumbous oxide; Lead monoxide; Lead ochre; Lead oxide; Lead oxide, 99.99%; Lead oxide(mono); Lead oxide (PbO); Lead oxide yellow; Lead protoxide; Litharge; Litharge yellow L-28; Massicot; Massicotite; Pigment yellow 46; Yellow lead ochre.
Molecular Formula:	PbO
Molecular Weight:	223.20
Melting point °C	888
Boiling point °C	Decomposes at 1472
Density kg/m <sup>3</sup> at 20 °C	9300 (Litharge)  8000 (Massicot)
Solubility, water at 20 °C	17 mg/L
pKa	No data
Vapour pressure	No data
Partition coefficient (Log Pow)	No data
Decomposition temperature °C	1472

## A1.2 Lead chromate

This substance is listed on the Australian Inventory of Chemical Substances (AICS) as:	Chromic acid (H <sub>2</sub> CrO <sub>4</sub> ), lead(2+) salt (1:1)
CAS No.:	7758-97-6
Appearance	Orange-yellow solid
Synonyms:	Dianichi chrome yellow G; Canary chrome yellow 40-2250; Crocoite; Lead chromate; Lead chromate (VI); Plumbous Chromate; Lead (II) chromate; Lead (IV) chromate; Leipzig yellow; Lemon yellow; Paris yellow; Pigment green 15; Pure lemon chrome l3; Chrome green 61; Chrome green 74; Chrome green 76; Chromic acid, Lead(2+) salt (1:1); Chromium yellow; Chrome lemon; Chrome yellow; Chrome yellow 5g; Chrome yellow g; Chrome yellow gf; Chrome yellow lf; Chrome yellow light 1066; Chrome yellow light 1075; Chrome yellow medium 1074; Chrome yellow medium 1085; Chrome yellow medium 1298; Chrome yellow primrose 1010; Chrome yellow primrose 1015; CI 77600; C.I. Pigment yellow 34; Cologne yellow; C.P. Chrome yellow light; C.P. Chrome yellow medium; C.P. Phrome yellow primrose.
Molecular Formula:	PbCrO <sub>4</sub>
Molecular Weight:	323.19
Melting point °C	844
Boiling point °C	decomposes
Density kg/m <sup>3</sup> at 15 °C	6120
Solubility, water at 25 °C	0.2 mg/L
pKa	No data
Vapour pressure	No data
Partition coefficient (Log Pow)	No data
Decomposition temperature °C	No data

### A1.3 Lead sulfate

This substance is listed on the Australian Inventory of Chemical Substances (AICS) as:	Sulfuric acid, lead(2+) salt (1:1)
CAS No.:	7446-14-2
Appearance	White solid
Synonyms:	Anglesite; Fast white; Lead (II) sulfate; Lead sulfate; Lead sulfate 99.999%; Lead sulphate; Lead(II) sulfate; Milk white; Sulfuric acid, lead (2+) Salt (1:1); White lead; Lear monosulfate; Lead Bottoms
Molecular Formula:	PbSO <sub>4</sub>
Molecular Weight:	303.26
Melting point °C	1170
Boiling point °C	No data
Density kg/m <sup>3</sup> at 20 °C	6200
Solubility, water at 25 °C	42.5 mg/L
pKa	No data
Vapour pressure	No data
Partition coefficient (Log Pow)	No data
Decomposition temperature °C	No data

### A1.4 Lead molybdate

This substance is listed on the Australian Inventory of Chemical Substances (AICS) as:	Molybdic acid (H <sub>2</sub> MoO <sub>4</sub> ), lead(2+) salt (1:1)
CAS No.:	10190-55-3
Appearance	White solid
Synonyms:	Lead (II) molybdate; Lead molybdate; Lead molybdate 99.97%; Plumbous molybdate
Molecular Formula:	PbMoO <sub>4</sub>
Molecular Weight:	367.14
Melting point °C	1070
Boiling point °C	No data
Density kg/m <sup>3</sup> at 25 °C	6900

Solubility, water at 20 °C	insoluble
pKa	No data
Vapour pressure	No data
Partition coefficient (Log Pow)	No data
Decomposition temperature °C	No data

### A1.5 Lead sulfo-chromate

This substance is listed on the Australian Inventory of Chemical Substances (AICS) as:	C.I. Pigment Yellow 34
CAS No.:	1344-37-2
Appearance	No data
Synonyms:	Chromastral green M; Chromastral green Y; Chromastral green HM; Chrome fast green CP; Chrome orange; Chrome yellow; Chrome yellow A-241; Chrome yellow 10G; Chrome yellow 4G; Chrome yellow 5GF; Chrome yellow 62E; Chrome yellow GL medium; Chrome yellow lemon; Chrome yellow LF AA; Chrome yellow light; Chrome yellow light Y 434D; Chrome yellow light 4GL; Chrome yellow medium; Chrome yellow medium Y 469; Chrome yellow middle; Chrome yellow primrose; Chrome yellow 6GL primrose; CI 77600; CI 77603; CP Chrome yellow light 1066; CP Chrome yellow light 1074; CP Chrome yellow medium 1074; CP Chrome yellow medium 1085; CP Chrome yellow medium 1298; Dainichi chrome yellow 10G; Dainichi chrome yellow 5G; Krolor yellow KY 788D; Lemon chrome A 3G; Lemon chrome C 4G; Middle chrome BHG; Primrose chrome; Pure lemon chrome L 3G; Pure lemon chrome L 3GS; Pure lemon chrome 24882; Pure lemon chrome 3GN; Pure lemon chrome HL 3G; Pure middle chrome 24883; Pure middle chrome LG; Pure primrose chrome LG; Pure primrose chrome L 10G; Pure primrose chrome L 6G; Pure primrose chrome 24880; Pure primrose chrome 24881; Renol chrome yellow Y 2; Renol

	chrome yellow Y 2RS.
Molecular Formula:	Unspecified
Molecular Weight:	Unspecified
Melting point °C	No data
Boiling point °C	No data
Density kg/m <sup>3</sup> at 20 °C	No data
Solubility, water at 20 °C	No data
pKa	No data
Vapour pressure	No data
Partition coefficient (Log Pow)	No data
Decomposition temperature °C	No data

#### **A1.6 Lead chromate molybdate sulfate red**

This substance is listed on the Australian Inventory of Chemical Substances (AICS) as:	C.I. Pigment Red 104
CAS No.:	12656-85-8
Appearance	No data
Synonyms:	Molybdenum orange; C.I. 77605; Chrome Vermilion; Horna Molybdate Orange MLH 84SQ; Krolor Orange KO 906D; Krolor Orange RKO 786D; Mineral Fire Red 5DDS; Mineral Fire Red 5GGS; Mineral Fire Red 5GS; Molybdate Orange Y 786D; Molybdate Orange YE 421D; Molybdate Orange YE 698D; Molybdate Red; Molybdate Red AA 3; Molybden Red; Molybdenum orange; Molybdenum Red; Renol Molybdate Red RGS; Vynamon Scarlet BY; Vynamon Scarlet Y
Molecular Formula:	Unspecified
Molecular Weight:	Unspecified
Melting point °C	No data
Boiling point °C	No data
Density kg/m <sup>3</sup> at 20 °C	No data

Solubility, water at 20 °C	No data
pKa	No data
Vapour pressure	No data
Partition coefficient (Log Pow)	No data
Decomposition temperature °C	No data

### A1.7 Lead chromate oxide

This substance is listed on the Australian Inventory of Chemical Substances (AICS) as:	Lead Chromate Oxide
CAS No.:	18454-12-1
Appearance	No data
Synonyms:	Lead chromate, basic; Lead chromate oxide, Basic chromium lead oxide, Chromic acid lead(2+) salt (1:2), Chromium dilead pentaoxide, Chromium lead oxide, Chromium lead oxide, Lead chromate, Lead chromate oxide, Lead chromate(VI), Lead chromate(VI) oxide
Molecular Formula:	CrO <sub>4</sub> .O.Pb
Molecular Weight:	323.2
Melting point °C	No data
Boiling point °C	No data
Density g/cm <sup>3</sup> at 20 °C	No data
Specific gravity	No data
Solubility	No data
Water at 20 °C	
pKa	No data
Vapour pressure	No data
Partition coefficient ( <i>Log Pow</i> )	No data
Flash point	No data
Decomposition temperature °C	No data
Autoignition temperature	No data

### A1.8 Lead octanoate

This substance is listed on the Australian Inventory of Chemical Substances (AICS) as:	Octanoic acid, lead(2+) salt
CAS No.:	7319-86-0
Appearance	Yellowish liquid
Synonyms:	Lead(II) n-octanoate; Lead Octanoate (in Mineral Spirits), 24% Lead Content; Lead (II) octoate; Lead dioctanoate; Lead(II) caprylate; Lead(II) octanoate; Minico P 25; P 25
Molecular Formula:	$C_8H_{16}O_2 \cdot \frac{1}{2}Pb$
Molecular Weight:	247.8
Melting point °C	No data
Boiling point °C	No data
Density kg/m <sup>3</sup> at 20 °C	No data
Solubility, water at 20 °C	No data
pKa	No data
Vapour pressure	No data
Partition coefficient (Log Pow)	No data
Flash point	No data
Decomposition temperature °C	No data
Autoignition temperature	No data

### A1.9 Lead 2-ethylhexanoate

This substance is listed on the Australian Inventory of Chemical Substances (AICS) as:	Hexanoic acid, 2-ethyl-, lead(2+) salt
CAS No.:	301-08-6
Appearance	No data
Synonyms:	Lead (II) 2-ethylhexanoate.
Molecular Formula:	$C_8H_{16}O_2 \cdot \frac{1}{2}Pb$

Molecular Weight:	247.8
Melting point °C	No data
Boiling point °C	No data
Density kg/m <sup>3</sup> at 20 °C	No data
Solubility, wWater at 20 °C	No data
pKa	No data
Vapour pressure	No data
Partition coefficient (Log Pow)	No data
Decomposition temperature °C	No data

### A1.10 Lead oxide

This substance is listed on the Australian Inventory of Chemical Substances (AICS) as:	Lead oxide (Pb <sub>3</sub> O <sub>4</sub> )
CAS No.:	1314-41-6
Appearance	Red solid
Synonyms:	Lead (II,III) oxide; Lead(II,IV) oxide; Lead oxide; Lead Tetraoxide; Lead tetroxide; Mineral red; orange lead; Red lead oxide; Azarcon; C.I. 77578; C.I. Pigment Red 105; Entan; Flowsperse R 12; Gold Satinobre; Heuconin 5; Lead orthoplumbate; Lead oxide (3:4); Lead oxide red; Mennige; Mineral Orange; Minium; Minium (Pb <sub>3</sub> O <sub>4</sub> ); Minium Non-Setting RL 95; Paris Red; Red lead; Red lead (pigment); Sandix; Saturn Red; Trilead tetroxide
Molecular Formula:	Pb <sub>3</sub> O <sub>4</sub>
Molecular Weight:	685.60
Melting point °C	Decomposes at 500
Boiling point °C	No data
Density kg/m <sup>3</sup> at 25 °C	9100
Solubility, water at 20 °C	insoluble
pKa	No data
Vapour pressure	No data



Partition coefficient (Log Pow)	No data
Decomposition temperature °C	No data

### A1.11 Lead nitrate

This substance is listed on the Australian Inventory of Chemical Substances (AICS) as:	Nitric acid, lead(2+) salt
CAS No.:	10099-74-8
Appearance	Colourless or white solid
Synonyms:	Lead dinitrate; Lead (II) nitrate; Lead nitrate; Lead nitrate, 99.5%; Plumbous nitrate; Lead (II) nitrate; Lead(2+) bis(nitrate); Lead(2+) nitrate; Lead(II) dinitrate
Molecular Formula:	$\text{HNO}_3 \cdot \frac{1}{2}\text{Pb}$
Molecular Weight:	331.21
Melting point °C	Decomposes at 470
Boiling point °C	No data
Density $\text{kg/m}^3$ at 20 °C	4530
Specific gravity	No data
Solubility, water at 20 °C	565 g/L
pKa	No data
Vapour pressure	No data
Partition coefficient (Log Pow)	No data
Decomposition temperature °C	470

### A1.12 Lead naphthenate

This substance is listed on the Australian Inventory of Chemical Substances (AICS) as:	Naphthenic acids, lead salts
CAS No.:	61790-14-5
Appearance	Yellow to brown resinous liquid
Synonyms:	Lead naphthenate; Lead naphthenate 61% min in mineral spirits (24% Pb);

	Naphthenic acid, lead salts; Naphthenic acids, lead salts; Cyclohexanecarboxylic acid, lead salt; Naphthex Pb; Octa-Soligen Pb 24; Trokyd Lead
Molecular Formula:	Unspecified
Molecular Weight:	Unspecified
Melting point °C	No data
Boiling point °C	No data
Density kg/m <sup>3</sup> at 20 °C	No data
Solubility, water at 20 °C	No data
pKa	No data
Vapour pressure	No data
Partition coefficient (Log Pow)	No data
Decomposition temperature °C	No data

### A1.13 Lead oxide

This substance is listed on the Australian Inventory of Chemical Substances (AICS) as:	Lead oxide (PbO <sub>2</sub> )
CAS No.:	1309-60-0
Appearance	No data
Synonyms:	C.I. 77580; Lead dioxide; Lead brown; lead(IV) oxide; Lead oxide; Lead oxide brown; Lead peroxide; lead superoxide; Plumbic acid; Plumbic oxide
Molecular Formula:	PbO <sub>2</sub>
Molecular Weight:	239.2
Melting point °C	No data
Boiling point °C	No data
Density kg/m <sup>3</sup> at 20 °C	No data
Solubility, water at 20 °C	No data
pKa	No data
Vapour pressure	No data
Partition coefficient (Log Pow)	No data

Decomposition temperature °C	No data
------------------------------	---------

#### A1.14 Lead carbonate

This substance is listed on the Australian Inventory of Chemical Substances (AICS) as:	Lead, bis(carbonato(2-))dihydroxytri-
CAS No.:	1319-46-6
Appearance	White solid
Synonyms:	Carbonic acid, lead(2+) salt (1:1); Lead carbonate; Lead carbonate, 99.999%; Lead (II) carbonate; Basic lead carbonate; Almex; Basic carbonate white lead; Berlin White; Bis[carbonato(2-)]dihydroxytrilead; C.I. 77597; C.I. Pigment White 1; Carbonic acid, lead salt, basic; Dutch White Lead; Enpaku; Flake White; Krems White; Lead carbonate; Lead carbonate hydroxide; Lead carbonate oxide monohydrate; Lead hydroxide carbonate; Lead subcarbonate; Lead white; Lead, bis(carbonato)dihydroxytri-; Novade; Rolite lead; Silver White; Slate White; Stabilisator 5012NS; Venetian White; White lead; White Lead Wartburg
Molecular Formula:	C <sub>2</sub> H <sub>2</sub> O <sub>8</sub> Pb <sub>3</sub>
Molecular Weight:	775.6
Melting point °C	Decomposes at 340
Boiling point °C	No data
Density kg/m <sup>3</sup> at 25 °C	6600
Solubility, water at 20 °C	0.17 mg/100g
pKa	No data
Vapour pressure	No data
Partition coefficient (Log Pow)	No data
Decomposition temperature °C	No data

### **A1.15 Lead chrome 1244**

This chemical could not be identified hence no data could be found. Industry was not able to further identify the chemical beyond the trade name nor provide data on it. It appears the chemical was used in surface coatings and/or inks in the past but has not been used for some years.

# References

Aardema, M.J., Albertini, S., Arni, P., Henderson, L.M., Kirsch-Volders, M., Mackay, J.M., Sariff, A.M., Stringer, S.A and Taalman, R.D.F (1998). Aneuploidy: a report of an ECETOC task force. *Mutation Research*, 410, p3 – 79.

\*Adebonojo, F.O (1974). Hematologic status of urban black children in Philadelphia: Emphasis on the frequency of anemia and elevated blood lead levels. *Clinical Pediatrics* ,13, p874 - 888.

\*Adhikari, N., Sinha, N., Narayan, R., et al. (2001). Lead-induced cell death in testes of young rats. *Journal of Applied Toxicology*, 21, p275 - 277.

AIHW (1996a). Lead in Australian children. Australian Institute of Health and Welfare, Canberra.

AIHW (1996b). Lead in Australian children – Summary of the National survey of lead in children, Canberra.

\*Alderson, M.R., Rattan, N.S and Bidstrup, L (1981). Health of workmen in the chromate-producing industry in Britain. *British Journal of Industrial Medicine*, 38, p117 – 124.

\*Alessio, L., Bertazzi, P.A., Monelli, O., et al. (1976). Free erythrocyte protoporphyrin as an indicator of the biological effect of lead in adult males: II. Comparison between free erythrocyte protoporphyrin and other indicators of effect. *International Archives Occupational Environmental Health*, 37, p89 - 105.

\*Alexander, B.H., Checkoway, H., van Netten, C., et al. (1996). Semen quality of men employed at a lead smelter. *Occupational and Environmental Medicine*, 53, p411 - 416.

\*Al-Hakkak, Z.S.H., Hamamy, H.A., Murad, A.M.B., et al. (1986). Chromosome aberrations in workers at a storage battery plant in Iraq. *Mutation Research*, 171, p53 – 60.

Al-Hakkak, Z.S., Zahid, Z.R., Ibrahim, D.K., Al-Jumaily, I.S and Bazzaz, A.A (1988). Effects of ingestion of lead monoxide alloy on male mouse reproduction. *Archives of Toxicology*, 62, p97 – 100.

\*Al Khayat, A., Menon, N.S and Alidina, M.R (1997). Acute lead encephalopathy in early infancy-clinical presentation and outcome. *Annals of Tropical Paediatrics*, 17(1), p39 - 44.

\*Alvares, A.P., Kapelner, S., Sassa, S., et al. (1975). Drug metabolism in normal children, lead-poisoned children, and normal adults. *Clinical Pharmacology and Therapeutics*, 17, p179 - 183.

\*Anttila, A, Heikkila, P., Nykyri, E., et al. (1996). Risk of nervous system cancer among workers exposed to lead. *Journal of Occupational and Environmental Medicine*, 38 (2), P131 – 136.

\*Anttila, A., Heikkila, P., Pukkala, E., et al. (1995). Excess lung cancer among workers exposed to lead. *Scandinavian Journal of Work and Environmental Health*, 21, p460 – 469.

APMF (2005) Code of Practice for the Automotive Refinishing Industry Edition II. Australian Paint Manufacturers' Federation Inc. North Sydney, Australia

APMF (2006) Australian Paint Manufacturers' Federation Inc. Personal communication.

- \*Apostoli, P., Maranelli, G., Cas, L.D., et al. (1990). Blood lead and blood pressure: A cross sectional study in a general population group. *Cardiologia*, 35 (7), p597 -603.
- \*Ariza, M.E., Bijur, G.N and Williams, M.V (1998). Lead and mercury mutagenesis: Role of H<sub>2</sub>O<sub>2</sub>, superoxide dismutase, and xanthine oxidase. *Environmental and Molecular Mutagenesis*, 31, p352 - 361.
- \*Arnvig, E., Grandjean, P., Beckmann, J., (1980). Neurotoxic effects of heavy lead exposure determined with psychological tests. *Toxicology Letters*, 5, p399 - 404.
- \*Assennato, G., Paci, C., Baser, M.E., et al. (1987). Sperm count suppression without endocrine dysfunction in lead-exposed men. *Archives of Environmental Health*, 42, p124 - 127.
- ATSDR, (2005). Draft Toxicological profile for lead. Accessed at: <http://www.atsdr.cdc.gov/toxprofiles/tp13.html> in July 2006.
- \*Azar, A., Trochimowicz, H.J and Maxfield, M.E (1973). Review of lead studies in animals carried out at Haskell Laboratory: Two year feeding study and response to hemorrhage study. In: Barth D, Berlin A, Engel R, et al., eds. *Environmental health aspects of lead: Proceedings, International Symposium, October 1972, Amsterdam, The Netherlands. Luxembourg: Commission of the European Communities*, 199-210.
- \*Baghurst, P.A., Robertson, E.F., McMichael, A.J., et al. (1987). The Port Pirie cohort study: Lead effects on pregnancy outcome and early childhood development. *Neurotoxicology*, 8, p395 - 401.
- \*Baker, E.L., Feldman, R.G., White, R.F., et al. (1983). The role of occupational lead exposure in the genesis of psychiatric and behavioral disturbances. *ACTA Psychiatrica Scandinavica Suppl.*, 67, p38 - 48.
- \*Baker, E.L., Goyer, R.A., Fowler, B.A., et al. (1980). Occupational lead exposure, nephropathy, and renal cancer. *American Journal of Industrial Medicine*, 1, p138 - 148.
- \*Baker, E.L., Landrigan, P.J., Barbour, A.G., et al. (1979). Occupational lead poisoning in the United States: Clinical and biochemical findings related to blood lead levels. *British Journal of Industrial Medicine*, 36, p314 - 322.
- \*Baloh, R.W., Spivey, G.H., Brown, C.P., et al. (1979). Subclinical effects of chronic increased lead absorption--a prospective study: II. Results of baseline neurologic testing. *Journal of Occupational Medicine*, 21, p490 - 496.
- \*Barratt, C.L.R., Davies, A.G., Bansal, M.R., et al. (1989). The effects of lead on the male rat reproductive system. *Andrologia*, 21, p161 - 166.
- \*Barth, A., Schaffer, A.W., Osterodem W., et al. (2002). Reduced cognitive abilities in lead-exposed men. *International Archives of Occupational and Environmental Health*, 75, p394 - 398.
- \*Batra, N., Nehru, B and Bansal, M.P (2001). Influence of lead and zinc on rat male reproduction at 'biochemical and histopathological levels'. *Journal of Applied Toxicology*, 21, p507 - 512.
- \*Bauchinger, M., Dresch, J., Schmid, E., et al. (1977). Chromosome analyses of children after ecological lead exposure. *Mutation Research*, 56, p75 - 79.
- \*Bauchinger, M and Schmid, E (1972). Chromosomenanalysen in zellkulturen des chinesischen hamsters nach applikation von bleiacetat. *Mutation Research*, 14, p95 - 100.

Bazzaz, A.A., Muhsin, S.S and Sulaiman, N.A (1989). Haematological changes in blood of mice induced by subchronic ingestion of lead monoxide. *Journal of Biological Sciences Research*, 20 (1), p31 – 40.

\*Becker, N., Claude, J and Frentzel-Beyme, R (1985). Cancer risk of arc welders exposed to fumes containing chromium and nickel. *Scandinavian Journal of Work, Environment and Health*, 11, p75 – 82.

\*Beek, B and Obe, G (1975). The human leukocyte test system: VI. The use of sister chromatid exchanges as possible indicators for mutagenic activities. *Humangenetik*, 29, p127 - 134.

\*Beek, B and Obe, G (1974). Effect of lead acetate on human leukocyte chromosomes *in vitro*. *Experientia*, 30, p1006 - 1007.

\*Bertazzi, P.A., Zocchetti, C., Terzagli, G.F., Riboldi, L., Guercilena, S and Beretta, F (1981). Carcinogenic risk in the production of paints. A mortality study (Ital.). *Medicina del Lavoro*, 6, p465 – 472.

\*Bellinger, D.C., Sloman, J., Leviton, A., et al. (1991). Low-level lead exposure and children's cognitive function in the preschool years. *Pediatrics*, 87, p219 - 227.

\*Berlin, C.M., Gorman, R.L., May, D.G., et al. (1995). Treatment guidelines for lead exposure in children. *Pediatrics* 96 (1), p155 - 160.

\*Betts, P.R., Astley, R and Raine, D.N (1973). Lead intoxication in children in Birmingham. *British Medical Journal*, 1, p402 - 406.

\*Biagini, G., Caudarella, R and Vangelista, A (1977). Renal morphological and functional modification in chronic lead poisoning. In: Brown SS, ed. *Clinical chemistry and chemical toxicology of metals*. Elsevier/North-Holland Biomedical Press, p123 - 126.

\*Bizarro, P., Acevedo, S., Nino-Cabrera, G., et al. (2003). Ultrastructural modifications in the mitochondrion of mouse Sertoli cells after inhalation of lead, cadmium or lead-cadmium mixture. *Reproductive Toxicology*, 17, p561 - 566.

Bonacker, D., Stoiber, T., Bohm, K.J., Prots, I., Wang, M., Unger, E., Their, R., Bolt, H.M and Degen, G.H (2005). Genotoxicity of inorganic lead salts and disturbance of microtubule function. *Environmental and Molecular Mutagenesis*, 45, p346 – 353.

Bonde, J.P., Joffe, M., Apostoli, P., Dale, A., Kiss, P., Spano, M., Caruso, F., Giwercman, A., Bisanti, L., Porru, S., Vanhoorne, M., Comhaire, F and Zschiesche, W (2002). Sperm count and chromatin structure in men exposed to inorganic lead: lowest adverse effect levels. *Occupational and Environmental Medicine*, 59, p234 – 242.

\*Bonde, J.P and Kolstad, H (1997). Fertility of Danish battery workers exposed to lead. *International Journal of Epidemiology*, 26 (6), p1281 - 1288.

Borja-Aburto, V.H., Hertz-Picciotto, I., Lopez, M.R., Farias, P., Rios, C and Blanco, J (1999). Blood lead levels measured prospectively and risk of spontaneous abortion. *American Journal of Epidemiology*, 150 (6), p590 – 597.

Bornschein, R.L., Grote, J., Mitchell, T., Succop, P.A., Dietrich, K.N., Krafft, K.M and Hammond, P.B (1989). Effects of prenatal lead exposure on infant size at birth. In: Smith M.A., Grant, L.D and Sors, A.I ed. *Lead exposure and child development: An international assessment*. Dordrecht, London, Kluwer Academic Publishers, p307 – 319.

\*Bost, L., Primates, P., Dong, W., et al. (1999). Blood lead and blood pressure: Evidence from the health survey for England 1995. *Journal of Human Hypertension*, 13(22), p123 - 128.

\*Bradley, J.E and Baumgartner, R.J (1958). Subsequent mental development of children with lead encephalopathy, as related to type of treatment. *Journal of Pediatrics*, 53, p311 - 315.

\*Bradley, J.E., Powell, A.E., Niermann W, et al. (1956). The incidence of abnormal blood levels of lead in a metropolitan pediatric clinic: With observation on the value of coproporphyrinuria as a screening test. *Journal of Pediatrics*, 49, p1 - 6.

\*Bruce, W.R and Heddle, J.A (1979). The mutagenic activity of 61 agents as determined by the micronucleus, Salmonella and sperm abnormality assays. *Canadian Journal of Genetics and Cytology*, 21, p319 - 334.

\*Bulsma, J.B and De France, H.F (1976). Cytogenetic investigations in volunteers ingesting inorganic lead. *International Archives of Occupational and Environmental Health*, 28, p145 - 148.

\*Campara, P., D'Andrea, F., Micciolo, R., et al. (1984). Psychological performance of workers with blood-lead concentration below the current threshold limit value. *International Archives of Occupational and Environmental Health*, 53, p233 - 246.

\*Cardenas, A., Roels, H., Bernard, A.M., et al. (1993). Markers of early renal changes induced by industrial pollutants II. Application to workers exposed to lead. *British Journal of Industrial Medicine*, 50, p28 - 36.

CEPE (2005) Safe Powder Coating Guideline 7<sup>th</sup> Edition. European Council of the Paint, Printing Ink and Artists' Colours Industry (CEPE)

\*Chia, S.E., Chia, K.S., Chia, H.P., et al. (1996). Three-year follow-up of serial nerve conduction among lead-exposed workers. *Scandinavian Journal of Work, Environment and Health* 22 (5), p374 - 80.

\*Chia, K.S., Jeyaratnam, J., Lee, J., et al. (1995). Lead-induced nephropathy: Relationship between various biological exposure indices and early markers of nephrotoxicity. *American Journal of Industrial Medicine*, 27, p883 - 895.

\*Chia, K.S., Mutti, A., Tan, C., et al. (1994). Urinary N-acetyl-D-glucosaminidase activity in workers exposed to inorganic lead. *Occupational and Environmental Medicine*, 51, p125 - 129.

\*Chisolm, J.J. (1965). Chronic lead intoxication in children. *Developmental Medicine and Child Neurology*, 7, p529 - 536.

Chisolm, J.J (1962). Aminoaciduria as a manifestation of renal tubular injury in lead intoxication and a comparison with patterns of aminoaciduria seen in other diseases. *The Journal of Pediatrics*, 60 (1), p1 - 17.

\*Chisolm, J.J and Harrison, H.E (1956). The exposure of children to lead. *Pediatrics*, 18, p943 - 958.

\*Chowdhury, A.R., Chinoy, N.J., Gautam, A.K., et al. (1986). Effect of lead on human semen. *Advances in Contraceptive Delivery Systems*, 2, p208 - 211.

\*Chu, N-F., Liou, S-H., Wu, T-N., et al. (1999). Reappraisal of the relation between blood lead concentration and blood pressure among the general population in Taiwan. *Occupational and Environmental Medicine*, 56, p30 - 33.



\*Cocco, P., Dosemeci, M and Heineman, E.F (1998a). Brain cancer and occupational exposure to lead. *Journal of Occupational and Environmental Medicine*, 40 (11), p937 - 942.

\*Cocco, P., Ward, M.H and Dosemeci, M (1998b). Occupational risk factors for cancer of the gastric cardia. *Journal of Occupational and Environmental Medicine*, 40 (10) p855 - 861.

\*Cocco, P., Hua, F., Boffetta, P., et al. (1997). Mortality of Italian lead smelter workers. *Scandinavian Journal of Work and Environmental Health*, 23 (1), p15 - 23.

\*Cools, A., Salle, H.J.A., Verberk, M.M., et al. (1976). Biochemical response of male volunteers ingesting inorganic lead for 49 days. *International Archives of Occupational and Environmental Health*, 38, p129 - 139.

\*Cooper, W.C., Wong, O and Kheifets, L (1985). Mortality among employees of lead battery plants and lead producing plants, 1947-1980. *Scandinavian Journal of Work and Environmental Health*, 11, p331 - 345.

Comcare (2004) Approved Code of Practice on the Control and Safe Use of Inorganic Lead in Commonwealth Employment

\*Cornell, R.G and Landis, J.R (1984). Mortality patterns among nickel/chromium alloy foundry workers. IN: Sunderman, F.W., Jr, ed. *Nickel in the Human Environment (IARC Scientific Publications No. 53)*, Lyon, International Agency for Research on Cancer, p87 – 93.

Costa, R., Strolego, G and Levis A.G (1988). Mutagenicity of lead chromate in *Drosophila melanogaster* in the presence of nitrilotriacetic acid (NTA). *Mutation Research*, 204, p257 – 261.

\*Costa, M., Cantoni, O., DeMars, M., et al. (1982). Toxic metals produce S-phase-specific cell cycle block. *Research Communications in Chemical Pathology and Pharmacology*, 38, p405 - 419.

\*Coste, J., Mandereau, L., Pessione, F., et al. (1991). Lead-exposed workmen and fertility: A cohort study on 354 subjects. *European Journal of Epidemiology*, 7, p154 - 158.

Coyle, P., Kosnett, M.J and Hipkins, K (2005). Severe lead poisoning in the plastics industry: A report of three cases. *American Journal of Industrial Medicine*, 47, p172 – 175.

\*Cramer, K., Goyer, R.A., Jagenburg, R., et al. (1974). Renal ultrastructure, renal function, and parameters of lead toxicity in workers with different periods of lead exposure. *British Journal of Industrial Medicine*, 31, p113 - 127.

\*Dalager, N.A., Mason, T.J., Fraumeni, J.F., Hoover, R and Payne, W.W (1980). Cancer mortality among workers exposed to zinc chromate paints. *Journal of Occupational Medicine*, 22, p25 – 29.

\*Dalpra, L., Tibiletti, M.G., Nocera, G., et al. (1983). SCE analysis in children exposed to lead emission from a smelting plant. *Mutation Research*, 120, p249 - 256.

Danadevi, K., Rozati, R., Banu, S.B., Rao, P.H and Grover, P (2003). DNA damage in workers exposed to lead using comet assay. *Toxicology*, 187, p183 – 193.

D’Arcy, JB and Chan, TL. (1990) Chemical distribution in high-solids paint overspray. *Am Ind Hyg Assoc J*, 51(3): 132 - 138

\*Davies, J.M (1984). Lung cancer mortality among workers making lead chromate and zinc chromate pigment at three English factories. *British Journal of Industrial Medicine*, 41, p158 – 169.

- \*de Kort, W.L.A.M and Zwennis, W.C.M (1988). Blood lead and blood pressure: Some implications for the situation in the Netherlands. *Environmental Health Perspectives*, 78, p67 – 77.
- \*de Kort W.L., Verschoor, M.A., Wibowom A.A.E., et al. (1987). Occupational exposure to lead and blood pressure: A study of 105 workers. *American Journal of Industrial Medicine*, 11, p145 - 156.
- \*Deknudt, G and Gerber, G.B (1979). Chromosomal aberrations in bone-marrow cells of mice given a normal or a calcium-deficient diet supplemented with various heavy metals. *Mutation Research*, 68, p163 - 168.
- \*Deknudt, G., Colle, A and Gerber, G.B (1977). Chromosomal abnormalities in lymphocytes from monkeys poisoned with lead. *Mutation Research*, 45, p7 - 83.
- \*Deknudt, G and Deminatti, M (1978). Chromosome studies in human lymphocytes after *in vitro* exposure to metal salts. *Toxicology*, 10, p67 - 75.
- \*Den Hond, E., Nawrot, T and Staessen, J.A (2001). Relationship between blood pressure and blood lead in NHANES III. *Journal of Hypertension*, 19(2), S57.
- Devi, D.K., Banu, S.B., Grover, P and Jamil K (2000). Genotoxic effect of lead nitrate on mice using SCGE (comet assay). *Toxicology*, 145, p195 – 201.
- \*Diamond, G.L (2005). Risk assessment of nephrotoxic metals. In: Tarloff J, Lash L, eds. *The toxicology of the kidney*. London: CRC Press, p1099 - 1132.
- Dieter, M.P., Mathews, H.B., Jeffcoat, R.A and Moseman, R.F (1993). Comparison of lead bioavailability in F344 rats fed lead acetate, lead oxide, lead sulfide, or lead ore concentrate from Skagway, Alaska. *Journal of Toxicology & Environmental Health*, 39 (1), p79 – 93.
- \*Dietrich, K.N., Krafft, K.M., Bier, M., et al. (1986). Early effects of fetal lead exposure: Neurobehavioral findings at 6 months. *International Journal of Biosocial and Medical Research*, 8, p151 - 168.
- \*Dunkel, V.C., Zieger, E., Brusick, D., et al. (1984). Reproducibility of microbial mutagenicity assays: 1. Tests with *Salmonella typhimurim* and *Escherichia coli* using a standardized protocol. *Environmental Mutagenesis*, 6 (Suppl. 2), p1 - 254.
- \*Duydu, Y., Suzen, H.S., Aydin, A., et al. (2001). Correlation between lead exposure indicators and sister chromatid exchange (SCE) frequencies in lymphocytes from inorganic lead exposed workers. *Archives of Environment Contamination and Toxicology*, 41, p241 - 246.
- Ehle, A.L (1986). Lead neuropathy and electrophysiological studies in low level lead exposure: a critical review. *NeuroToxicology*, 7 (3), p203 – 216.
- \*Ehrlich, R., Robins, T., Jordaan, E., et al. (1998). Lead absorption and renal dysfunction in a South African battery factory. *Occupational and Environmental Medicine*, 55, p453 - 460.
- \*Elwood, P.C., Davey-Smith, G., Oldham, P.D., et al. (1988a). Two Welsh surveys of blood lead and blood pressure. *Environmental Health Perspectives*, 78, p119 - 121.
- \*Elwood, P.C., Yarnell, J.W.G., Oldham, P.D., et al. (1988b). Blood pressure and blood lead in surveys in Wales. *American Journal of Epidemiology*, 127, p942 - 945.
- Enander, R.T., Cohen, H.J., Gute, D.M., Brown, L.C., Desmaris, A.M., and Missaghian, R. (2004). *J Occup Environ Hyg*, 1(2): 119 – 125

Enander, RT., Gute, DM., Cohen, HJ., Brown, LC., Desmaris, AM., and Missaghian, R. (2002). *AIHA J*, 63(6): 741 - 749

\*EPA, (1986). Air quality criteria for lead. Research Triangle Park, NC: US. Environmental Protection Agency, Office of Research and Development, Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office. EPA600883028F.

\*Erenberg, G., Rinsler, S.S and Fish, B.G (1974). Lead neuropathy and sickle cell disease. *Pediatrics*, 54, p438 - 441.

\*Ernhart, C.B., Wolf, A.W., Kennard, M.J., et al. (1986). Intrauterine exposure to low levels of lead: The status of the neonate. *Archives of Environmental Health*, 41, p287 - 291.

EUBEES (2004) Environmental Emission Scenarios for Biocides used as Film Preservatives. European Commission DG ENV / RIVM TM104-item6-PTZ.doc

\*Factor-Litvak, P., Wasserman, G., Kline, J.K., et al. (1999). The Yugoslavia prospective study of environmental lead exposure. *Environmental Health Perspectives*, 107, p9 - 15.

\*Fanning, D (1988). A mortality study of lead workers, 1926-1985. *Archives of Environmental Health*, 43, p247 - 251.

FASTS (2002). Indoor Air Quality for Australia: A Strategy for Action. Federation of Australian Scientific and Technological Societies. Occasional Paper Series No. 5, October 2002.

\*Fayerweather, W.E., Karns, M.E., Nuwayhid, I.A., et al. (1997). Case-control study of cancer risk in tetraethyl lead manufacturing. *American Journal of Industrial Medicine*, 31, p28 - 35.

\*Fels, L.M., Wunsch, M., Baranowski, J., et al. (1998). Adverse effects of chronic low level lead exposure on kidney function - a risk group study in children. *Nephrology Dialysis Transplantation*, 13, p2248 - 2256.

\*Forni, A., Camiaghi, G and Sechi, G.C (1976). Initial occupational exposure to lead: Chromosome and biochemical findings. *Archives of Environmental Health*, 31, p73 - 78.

FORS (Federal Office of Road Safety) (1989) Australian code for the transport of dangerous goods by road and rail (ADG Code), 6<sup>th</sup> ed., Canberra, AGPS.

\*Foster, W.G., Singh, A., McMahon, A., et al. (1998). Chronic lead exposure effects in the Cynomolgus monkey (*Macaca fascicularis*) testis. *Ultrastructural Pathology* 22 (1), p63 - 71.

\*Foster, W.G., McMahon, A and Rice, D.C (1996). Sperm chromatin structure is altered in Cynomolgus monkeys with environmentally relevant blood lead levels. *Toxicology and Industrial Health*, 12 (5), p723 - 735.

Foster, W.G (1992). Reproductive toxicity of chronic lead exposure in the female Cynomolgus monkey. *Reproductive Toxicology*, 6, p123 - 131.

\*Fowler, B.A., Kimmel, C.A., Woods, J.S., et al. (1980). Chronic low-level lead toxicity in the rat: III. An integrated assessment of long-term toxicity with special reference to the kidney. *Toxicology and Applied Pharmacology*, 56, p59 - 77.

\*Fracasso, M.E., Perbellini, L., Solda, S., et al. (2002). Lead induced DNA strand breaks in lymphocytes of exposed workers: Role of reactive oxygen species and protein kinase C. *Mutation Research*, 515, p159 - 169.

- \*Franchini, I., Magnani, F and Mutti, A (1983). Mortality experience among chromeplating workers. Initial findings. *Scandinavian Journal of Work, Environment and Health*, 9, p247 – 252.
- \*Franks, P.A., Laughlin, N.K., Dierschke, D.J., et al. (1989). Effects of lead on luteal function in Rhesus monkeys. *Biology of Reproduction*, 41, p1055 - 1062.
- Fu, H and Boffetta, P (1995). Cancer and occupational exposure to inorganic lead compounds: a meta-analysis of published data. *Occupational and Environmental Medicine*, 52, p73 – 81.
- \*Frentzel-Beyme, R (1983). Lung cancer mortality of workers employed in chromate pigment factories. A multicentric European epidemiology study. *Journal of Cancer Research and Clinical Oncology*, 105, p183 – 188.
- \*Fukunaga M, Kurachi Y, Mizuguchi Y. 1982. Action of some metal ions at yeast chromosomes. *Chemical and Pharmaceutical Bulletin*, 30, p3017 - 3019.
- \*Gant, V.A (1938). Lead poisoning. *Industrial Medicine*, 7, p679 - 699.
- \*Gartside, P.S (1988). The relationship of blood lead levels and blood pressure in NHANES II: Additional calculations. *Environmental Health Perspectives*, 78, p31 - 34.
- \*Gasiorek, K and Bauchinger, M (1981). Chromosome changes in human lymphocytes after separate and combined treatment with divalent salts of lead, cadmium, and zinc. *Environmental Mutation*, 3, p513 - 518.
- \*Gennart, J-P., Buchet, J-P., Roels, H., et al. (1992). Fertility of male workers exposed to cadmium, lead or manganese. *American Journal of Epidemiology*, 135, p1208 - 1219.
- \*Gerhardsson, L., Hagmar, L., Rylander, L., et al. (1995). Mortality and cancer incidence among secondary lead smelter workers. *Occupational and Environmental Medicine*, 52, p667 - 672.
- \*Gerhardsson, L., Chettle, D.R., Englyst, V., et al. (1992). Kidney effects in long term exposed lead smelter workers. *British Journal of Industrial Medicine*, 49, p186 - 192.
- \*Gerhardsson, L., Lundstrom, N.G., Nordberg, G., et al. (1986). Mortality and lead exposure: A retrospective cohort study of Swedish smelter workers. *British Journal of Industrial Medicine*, 43, p707 - 712.
- \*Gerr, F., Letz, R., Stokes, L., et al. (2002). Association between bone lead concentration and blood pressure among young adults. *American Journal of Industrial Medicine*, 42, p98 - 106.
- Glenn, B.S., Stewart W.F., Links J.M., Todd, A and Schwartz, B (2003). The longitudinal association of lead with blood pressure. *Epidemiology*, 14, p30 - 36.
- \*Glickman, L., Valciukas, J.A., Lilis, R., et al. (1984). Occupational lead exposure: Effects on saccadic eye movements. *International Archives of Occupational and Environmental Health*, 54, p115 - 125.
- Goodman, M., LaVerda, N., Clarke, C., Foster, E.D., Iannuzzi, J and Mandel, J (2002). Neurobehavioural testing in workers occupationally exposed to lead: systematic review and meta-analysis of publications. *Occupational and Environmental Medicine*, 59, p217 – 223.
- \*Goyer, R.A (1989). Mechanisms of lead and cadmium nephrotoxicity. *Toxicology Letters*, 46, p153 – 162.

- \*Grandjean, P., Jorgensen, P.J., Viskum, S., et al. (1991). Temporal and interindividual variation in erythrocyte-zinc-protoporphyrin in lead exposed workers. *British Journal of Industrial Medicine*, 48, p 254 - 257.
- \*Grandjean, P., Hollnagel, H., Hedegaard, L., et al. (1989). Blood lead-blood pressure relations: Alcohol intake and hemoglobin as confounders. *American Journal of Epidemiology*, 129, p732 - 739.
- \*Grandjean, P., Wulf, H.C and Niebuhr, E (1983). Sister chromatid exchange in response to variations in occupational lead exposure. *Environmental Research*, 32, p199 - 204.
- \*Grandjean, P (1979). Occupational lead exposure in Denmark: Screening with the haematofluorometer. *British Journal of Industrial Medicine*, 36, p52 - 58.
- \*Graziano, J.H., Popovac, D., Factor-Litvak, P., et al. (1990). Determinants of elevated blood lead during pregnancy in a population surrounding a lead smelter in Kosovo, Yugoslavia. *Environmental Health Perspectives*, 89, p95 - 100.
- \*Griffin, T.B., Coulston, F and Wills, H (1975). [Biological and clinical effects of continuous exposure to airborne particulate lead.] *Arhiv za Higijenu Rada I Toksikologiju*, 26, p191 - 208. (Yugoslavian)
- \*Gurer-Orhan, H., Sabir, H.U and Ozgunesm, H (2004). Correlation between clinical indicators of lead poisoning and oxidative stress parameters in controls and lead-exposed workers. *Toxicology*, 195, p147 - 154.
- \*Haenninen, H., Hernberg, S., Mantere, P., et al. (1978). Psychological performance of subjects with low exposure to lead. *Journal of Occupational Medicine*, 20, p683 - 689.
- \*Harlan, W.R (1988). The relationship of blood lead levels to blood pressure in the US population. *Environmental Health Perspectives*, 78, p9 - 13.
- \*Harlan, W.R., Landis, J.R., Schmouder, R.L., et al. (1985). Blood lead and blood pressure: Relationship in the adolescent and adult U.S. population. *Journal of the American Medical Association*, 253, p530 - 534.
- \*Hense, H.W., Filipiak, B and Keil, U (1993). The association of blood lead and blood pressure in population surveys. *Epidemiology*, 4, p173 - 179.
- \*Hernberg, S., Westerholm, P., Schultz-Larsen, K., Degerth, R., Kuosma, E., Englund, A., Engzell, U., Sand Hanse, H and Mutanen, P (1983). Nasal and sinonasal cancer. Correction with occupational exposures in Denmark, Finland and Sweden. *Scandinavian Journal of Work, Environment and Health*, 9, p315 - 326.
- Hernberg, S and Nikkanen, J (1970). Enzyme inhibition by lead under normal urban conditions. *The Lancet*, 1, p63 - 64.
- \*Hernberg, S., Nikkanen, J., Mellin, G., et al. (1970).  $\delta$ -Aminolevulinic acid dehydrase as a measure of lead exposure. *Archives of Environmental Health*, 21, p140 - 145.
- \*Hoffman, D.J and Niyogi, S.K (1977). Metal mutagens and carcinogens affect RNA synthesis rates in a distinct manner. *Science*, 198, p513 - 514.
- \*Hogstedt, C., Hane, M., Agrell, A., et al. (1983). Neuropsychological test results and symptoms among workers with well-defined long-term exposure to lead. *British Journal of Industrial Medicine*, 40, p99 - 105.

- \*Hong, C.D., Hanenson, I.B., Lerner, S., et al. (1980). Occupational exposure to lead: Effects on renal function. *Kidney International*, 18, p489 - 494.
- \*Hsiao, C-Y., Wu, H-DI., Lai, J-S., et al. (2001). A longitudinal study of the effects of long-term exposure to lead among lead battery factory workers in Taiwan (1989-1999). *Science of the Total Environment*, 279, p151 - 158.
- \*Hsu, P.C., Hsu, C.C., Liu, M.Y., et al. (1998a). Lead-induced changes in spermatozoa function and metabolism. *Journal of Toxicology and Environmental Health*, A 55, p45 - 64.
- \*Hsu, P.C., Liu, M.Y., Hsu, C.C., et al. (1998b). Effects of vitamin E and/or C on reactive oxygen species-related lead toxicity in the rat sperm. *Toxicology*, 128, p169 - 179.
- \*Hu, H (1991). Knowledge of diagnosis and reproductive history among survivors of childhood plumbism. *American Journal of Public Health*, 81, p1070 - 1072.
- \*Hu, H., Aro, A., Payton, M., et al. (1996). The relationship of bone and blood lead to hypertension. The normative study. *Journal of the American Medical Association*, 275, p1171 - 1176.
- \*Hu, H., Pepper, L and Goldman, R (1991). Effect of repeated occupational exposure to lead, cessation of exposure, and chelation on levels of lead in bone. *American Journal of Industrial Medicine*, 20, p723 - 735.
- \*Huang, X.P., Feng, Z.Y., Zhai, W.L., et al. (1988). Chromosomal aberrations and sister chromatid exchanges in workers exposed to lead. *Biomedical and Environmental Science*, 1, p382 - 387.
- IARC (1987). Overall evaluations of carcinogenicity: An updating of IARC Monographs Volumes 1 to 42. Supplement 7. WHO International Agency for Research on Cancer.
- \*IARC (1980). Volume 23 - Some metals and metallic compounds. WHO International Agency for Research on Cancer.
- ISA (2006) Insurance Statistics Australia Motor Report June 2006
- \*Ishida, M., Ishizaki, M and Yamada, Y (1996). Decreases in postural change in finger blood flow in ceramic painters chronically exposed to low level lead. *American Journal of Industrial Medicine*, 29(5), p547 - 553.
- \*Jacquet, P and Tachon, P (1981). Effects of long-term lead exposure on monkey leukocyte chromosomes. *Toxicology Letters*, 8, p165 - 169.
- \*Jacquet, P., Leonard, A and Gerber, G.B (1977). Cytogenetic investigations on mice treated with lead. *Journal of Toxicology and Environmental Health*, 2, p619 - 624.
- Jagetia, G.C and Aruna, R (1998). Effect of various concentrations of lead nitrate on the induction of micronuclei in mouse bone marrow. *Mutation Research*, 415, p131 - 137.
- Jemal, A., Graubard, B.I., Devesa, S.S and Flegal, K.M (2002). The association of blood lead level and cancer mortality among whites in the United States. *Environmental Health Perspectives*, 10 (4), p325 - 329.
- \*Joffe, M., Bisanti, L., Apostoli, P., et al. (2003). Time to pregnancy and occupational lead exposure. *Occupational and Environmental Medicine*, 60, p752 - 758.

- \*Kehoe, R.A (1961). The metabolism of lead in man in health and disease: Present hygienic problems relating to the absorption of lead: The Harben lectures, 1960. *Journal of Royal Institute of Public Health and Hygiene*, 24, p177 - 203.
- Kharab, P and Singh, I (1985). Genotoxic effects of potassium dichromate, sodium arsenite, cobalt chloride and lead nitrate in diploid yeast. *Mutation Research*, 155, p117 - 120.
- \*Kim, R., Rotnitzky, A., Sparrow, D., et al. (1996). A longitudinal study of low-level lead exposure and impairment of renal function. The normative aging study. *Journal of the American Medical Association*, 275, p1177 - 1181.
- \*Korallus, U., Lange, H-J., Neiss, A., Wustefeld, E and Zwingers, T (1982). The relationship between active environmental measures taken in the chromate producing industry and lung carcinoma mortality (Ger.). *Arbeitsmed. Sozialmed. Pravent. Med*, 17, p159 – 167.
- \*Kromhout, D (1988). Blood lead and coronary heart disease risk among elderly men in Zutphen, The Netherlands. *Environmental Health Perspectives*, 78, p43 - 46.
- \*Kromhout, D., Wibowo, A.A.E., Herber, R.F.M., et al. (1985). Trace metals and coronary heart disease risk indicators in 152 elderly men (the Zutphen study). *American Journal of Epidemiology*, 122, p378 - 385.
- Kuhn, W., Winkel, R., Voitalla, D., Meves, S., Przuntek, H and Muller, T (1998). High prevalence of parkinsonism after occupational exposure to lead-sulfate batteries. *American Academy of Neurology*, 50 (6), p1885 – 1886.
- LaPuma, Pt and Rhodes, BS (2002) Chromate content versus particle size for aircraft paints. *Regul. Toxicol. Pharmacol.* 36 (3): 318 -324
- \*Lancranjan, I., Popescu, H.I., Gavanescu, O., et al. (1975). Reproductive ability of workmen occupationally exposed to lead. *Archives of Environmental Health*, 30, p396 - 401.
- \*Langard, S and Vigander T (1983). Occurrence of lung cancer in workers producing chromium pigments. *British Journal of Industrial Medicine*, 40, p71 – 74.
- \*Lauwerys, R., Buchet, J-P., Roels HA., et al. (1978). Placental transfer of lead, mercury, cadmium, and carbon monoxide in women: I. Comparison of the frequency distributions of the biological indices in maternal and umbilical cord blood. *Environmental Research*, 15, p278 - 289.
- \*Lerda, D (1992). Study of sperm characteristics in persons occupationally exposed to lead. *American Journal of Industrial Medicine*, 22, p567 - 571.
- Lebed'ko, O.A. and Ryzhavskii, B. Y. (2005). Effect of administration of lead nitrate to pregnant rats on the lungs of their offspring. *Bull. Exp. Biol. and Medicine*, 139 (6), 655 - 657
- \*Lilis, R (1981). Long-term occupational lead exposure, chronic nephropathy, and renal cancer: A case report. *American Journal of Industrial Medicine*, 2, p293 - 297.
- \*Lilis, R., Eisinger, J., Blumberg, W., et al. (1978). Hemoglobin, serum iron, and zinc protoporphyrin in lead-exposed workers. *Environmental Health Perspective*, 25, p97 - 102.
- \*Lilis, R., Gavrilesco, N., Nestoresco, B., et al. (1968). Nephropathy in chronic lead poisoning. *British Journal of Industrial Medicine*, 25, p196 - 202.
- \*Lin, J.L., Tan, D.T., Hsu, K.H., et al. (2001). Environmental lead exposure and progressive renal insufficiency. *Archives of Internal Medicine*, 161, p264 - 271.

- \*Lin, S., Hwang, S., Marshall, E.G., et al. (1996). Fertility rates among lead workers and professional bus drivers: A comparative study. *Annals of Epidemiology*, 6, p201 - 208.
- \*Lindgren, K.N., Ford, D.P and Bleecker, M.L (2003). Pattern of blood levels over working lifetime and neuropsychological performance. *Archives of Environmental Health*, 58(6), p373 - 379.
- \*Lindgren K.N., Masten, V.L., Ford, D.P., et al. (1996). Relation of cumulative exposure to inorganic lead and neuropsychological test performance. *Occupational and Environmental Medicine*, 53(7), p472 - 477.
- \*Lockett, C.J and Arbuckle, D (1987). Lead, ferritin, zinc, and hypertension. *Bulletin of Environmental Contamination and Toxicology*, 38, p975 - 980.
- \*Loghman-Adham, M (1997). Renal effects of environmental and occupational lead exposure. *Environmental Health Perspectives*, 105, p928 - 939.
- \*Lucchini, R., Albini, E., Cortesi, I., et al. (2000). Assessment of neurobehavioral performance as a function of current and cumulative occupational lead exposure. *Neurotoxicology*, 21(5), p805 - 812.
- \*Lundstrom, N.G., Nordberg, G., Englyst, V., et al. (1997). Cumulative lead exposure in relation to mortality and lung cancer morbidity in a cohort of primary smelter workers. *Scandinavian Journal of Work and Environmental Health*, 23 (1) p24 - 30.
- \*Lutz, P.M., Wilson, T.J., Ireland, J., et al. (1999). Elevated immunoglobulin E (IgE) levels in children with exposure to environmental lead. *Toxicology*, 134, p63 - 78.
- Macquarie (2005), *The Macquarie Dictionary*. Macquarie Library Pty Limited, Dee Why, Australia. (Reproduced with permission from Macquarie Library Pty Limited).
- \*Maheswaran, R., Gill, J.S and Beevers, D.G (1993). Blood pressure and industrial lead exposure. *American Journal of Epidemiology*, 137(6), p645 - 653.
- \*Maizlish, N.A., Parra, G and Feo, O (1995). Neurobehavioral evaluation of Venezuelan workers exposed to inorganic lead. *Occupational and Environmental Medicine*, 52, p408 - 414.
- \*Mäki-Paakkanen, J., Sorsa, M and Vainio, H (1981). Chromosome aberrations and sister chromatid exchanges in lead-exposed workers. *Hereditas*, 94, p269 - 275.
- \*Malcolm, D and Barnett, H.A.R (1982). A mortality study of lead workers: 1925-76. *British Journal of Industrial Medicine*, 39, p404 - 410.
- \*Mantere, P., Haenninen, H., Hernberg, S., (1982). Subclinical neurotoxic lead effects: Two-year follow-up studies with psychological test methods. *Neurobehavioural Toxicology and Teratology*, 4, p725 - 727.
- \*McMichael, A.J., Vimpani, G.V., Robertson, E.F., et al. (1986). The Port Pirie cohort study: Maternal blood lead and pregnancy outcome. *Journal of Epidemiology and Community Health*, 40, p18 - 25.
- Meniditto, A., Morisi, G., Spagnolo, A., et al. (1994). Association of blood lead to blood pressure in men aged 55 to 75 years: Effect of selected social and biochemical confounders. *Environmental Health Perspectives*, 102, p107 - 111.
- \*Meredith, P.A., Moore, M.R., Campbell, B.C., et al. (1978). Delta-aminolevulinic acid metabolism in normal and lead-exposed humans. *Toxicology*, 9, p1 - 9.



Meyer-Baron, M and Seeber, A (2000). A meta-analysis for neurobehavioural results due to occupational lead exposure with blood lead concentrations < 70 µg/100 ml. *Archives of Toxicology*, 73, p510 – 518.

\*Moller, L and Kristensen, T.S (1992). Blood lead as a cardiovascular risk factor. *American Journal of Epidemiology*, 136(9), p1091 - 1100.

\*Moore, M.R., Goldberg, A., Pocock, S.J., et al. (1982). Some studies of maternal and infant lead exposure in Glasgow. *Scottish Medical Journal*, 27, p113 - 122.

\*Moore, M.R and Meredith, P.A (1979). The carcinogenicity of lead. *Archives of Toxicology*, 42 p87 - 94.

\*Moreau, T., Hannaert, P., Orssaud, G., et al. (1988). Influence of membrane sodium transport upon the relation between blood lead and blood pressure in a general male population. *Environmental Health Perspectives*, 78, p47 - 51.

\*Moreau, T., Orssaud, G., Juguet, B., et al. (1982). [Blood lead levels and arterial pressure: Initial results of a cross sectional study of 431 male subjects.] *Revue d Epidemiologie et de Sante Publique*, 39, p395 - 397. (French)

\*Morris, C., McCarron, D.A and Bennett, W.M (1990). Low-level lead exposure, blood pressure, and calcium metabolism. *American Journal of Kidney Diseases*, 15, p568 - 574.

\*Mortada, W.I., Sobh, M.A., El-Defrawy, M.M., et al. (2001). Study of lead exposure from automobile exhaust as a risk for nephrotoxicity among traffic policemen. *American Journal of Nephrology*, 21, p274 - 279.

\*Muldoon, S.B., Cauley, J.A., Kuller, L.H., et al. (1996). Effects of blood lead levels on cognitive function of older women. *Neuroepidemiology*, 15(2), p62 - 72.

Muntner, P., He, J., Vupputuri, S., Coresh, J and Batuman, V (2003). Blood lead and chronic kidney disease in the general United States population: Results from NHANES III. *Kidney International*, 63, p1044-1050.

\*Muro, L.A and Goyer, R.A (1969). Chromosome damage in experimental lead poisoning. *Archives of Pathology*, 87, p660 - 663.

Murphy, M.J., Graziano, J.H., Popovac, D., Kline, J.K., Mehmeti, M., Factor-Litvak, P., Ahmedi, G., Shrout, P., Rajovic, B., Nenezic, D.U and Stein, Z.A (1990). Past pregnancy outcomes among women living in the vicinity of a lead smelter in Kosovo, Yugoslavia. *American Journal of Public Health*, 80 (1), p33 – 35.

Nawrot, T.S., Thijs, L., Den Hond, E.M., Roels, H.A and Staessen, J.A (2002). An epidemiological re-appraisal of the association between blood pressure and blood lead: a meta-analysis. *Journal of Human Hypertension*, 16, p123 – 131.

Nayak, B.N., Ray, M and Persaud, T.V.N (1989). Maternal and fetal chromosomal aberrations in mice following prenatal exposure to subembryotoxic doses of lead nitrate. *Acta Anatomica*, 135, p185 – 188.

\*Neri, L.C., Hewitt, D and Orser, B (1988). Blood lead and blood pressure: Analysis of cross-sectional and longitudinal data from Canada. *Environmental Health Perspectives*, 78, p123 - 126.

Nestmann, E.R., Matula, T.I., Douglas, G.R., et al. (1979). Detection of the mutagenic activity of lead chromate using a battery of microbial tests. *Mutation Research*, 66, p357 - 365.

\*Newhouse, M.L., Oakes, D and Woolley, A.J (1985). Mortality of welders and other craftsmen at a shipyard in NE England. *British Journal of Industrial Medicine*, 42, p406 – 410.

\*Niebuhr, E and Wulf, H.C (1984). Genotoxic effects. In: Grandjean P, ed. *Biological effects of organo-lead compounds*. Boca Raton, FL: CRC Press, p117 - 124.

\*NIOSH (1974). Evaluation of behavioral functions in workers exposed to lead. In: Xintaras, C., Johnson, B.L and De Groot, I, eds. *Behavioral toxicology: Early detection of occupational hazards*. Cincinnati, OH: U.S. Department of Health, Education and Welfare, National Institute for Occupational Safety and Health, p248 - 266.

\*Nishioka, H (1975). Mutagenic activities of metal compounds in bacteria. *Mutation Research*, 31, p185 - 189.

NOHSC (1994) National Standard for the control of Inorganic Lead at work [NOHSC:1012(1994)] National Occupational Health and Safety Commission, Canberra, AusInfo.

NOHSC (1994a) National Code of Practice for the control and Safe Use of Inorganic Lead at work [NOHSC: 1012(1994)] National Occupational Health and Safety Commission, Canberra, AusInfo.

NOHSC (1994b) National Model Regulations for the Control of Workplace Hazardous Substances [NOHSC: 1005 (1994)] National Occupational Health and Safety Commission, Canberra, AusInfo

NOHSC (1994c) National Code of Practice for the Control of Workplace Hazardous Substances [NOHSC: 2007(1994)] National Occupational Health and Safety Commission, Canberra, AusInfo.

NOHSC (1994d) National Code of Practice for the Labeling of Workplace Substances [NOHSC: 2012(1994)] National Occupational Health and Safety Commission, Canberra, AusInfo.

\*Nordensön, I., Beckman, G., Beckman, L., et al. (1978). Occupational and environmental risks in and around a smelter in northern Sweden: IV. Chromosomal aberrations in workers exposed to lead. *Hereditas*, 88, p263 - 267.

\*Nordstrom, S., Beckman, L and Nordensen, I (1979). Occupational and environmental risks in and around a smelter in northern Sweden: V. Spontaneous abortion among female employees and decreased birth weight in their offspring. *Hereditas*, 90, p291 - 296.

Nunes, J., Ehrich, M and Robertson, J (2001). Toxicosis associated with dual oral exposure of rats to lead and trichloroethylene. *Toxicologic Pathology*, 29 (4), p451 – 457.

\*O'Riordan, M.L and Evans, H.J (1974). Absence of significant chromosome damage in males occupationally exposed to lead. *Nature*, 247, p50 - 53.

\*Orssaud, G., Claude, J.R., Moreau, T., et al. (1985). Blood lead concentration and blood pressure. *British Medical Journal*, 290, p244.

\*Parkinson, D.K., Hodgson, M.J., Bromet, E.J., et al. (1987). Occupational lead exposure and blood pressure. *British Journal of Industrial Medicine*, 44 p744 - 748.

Payton, M., Riggs, K.M, Spiro III, A., Weiss, S.T and Hu, H (1998). Relations of bone and blood lead to cognitive function: The VA Normative Aging Study. *Neurotoxicology and Teratology*, 20 (1), p19 – 27.

- Payton, M., Hu, H., Sparrow, D and Weiss, S.T (1994). Low-level lead exposure and renal function in the normative aging study. *American Journal of Epidemiology*, 140 (9), p821 – 829.
- Pentschew, A and Garro, F (1966). Lead encephalo-myelopathy of the suckling rat and its implications on the porphyrinopathic nervous diseases. *Acta Neuropathologica*, 6, p266 –278.
- \*Perneger, T.W., Nieto, F.J., Whelton, P.K., et al. (1993). A prospective study of blood pressure and serum creatinine: Results from the ‘clue’ study and the ARIC study. *Journal of the American Medical Association*, 269, p488 - 493.
- \*Pergande, M., Junk, K., Precht, S., et al. (1994). Changed excretion of urinary proteins and enzymes by chronic exposure to lead. *Nephrology Dialysis Transplantation*, 9, p613 - 618.
- Pinon-Lataillade, G., Thoreaux-Manlay, A., Coffigny, H., Monchaux, G., Masse, R and Soufir J-C (1993). Effect of ingestion and inhalation of lead on the reproductive system and fertility of adult male rats and their progeny. *Human & Experimental Toxicology*, 12, p165 – 172.
- \*Pirkle, J.L., Schwartz, J., Landis, J.R., et al. (1985). The relationship between blood lead levels and blood pressure and its cardiovascular risk implications. *American Journal of Epidemiology*, 121, p246 - 258.
- \*Pocock, S.J., Shaper, A.G., Ashby, D., et al. (1988). The relationship between blood lead, blood pressure, stroke, and heart attacks in middle-aged British men. *Environmental Health Perspectives*, 78, p23 - 30.
- \*Pocock, S.J., Shaper A.G., Ashby D., et al. (1984). Blood lead concentration, blood pressure, and renal function. *British Medical Journal*, 289, p872 - 874.
- \*Poma, A., Pittaluga, E and Tucci, A (2003). Lead acetate genotoxicity on human melanoma cells *in vitro*. *Melanoma Research*, 13 (6), p563 - 566.
- \*Proctor, S.P., Rotnitzky, A., Sparrow, D., et al. (1996). The relationship of blood lead and dietary calcium to blood pressure in the normative aging study. *International Journal of Epidemiology*, 25(3), p528 - 536.
- \*Pueschel, S.M., Kopito, L and Schwachman, H (1972). Children with an increased lead burden: A screening and follow-up study. *Journal of the American Medical Association*, 222, p462 - 466.
- QLD, (2005). Blood Lead Notifications in Queensland 2005. Published at <http://www.health.qld.gov.au/phs/documents/ehu/31749.pdf> - accessed 7 May 2007
- \*Rabinowitz, M.B., Bellinger, D., Leviton, A., et al. (1987). Pregnancy hypertension, blood pressure during labor, and blood lead levels. *Hypertension*, 10, p447 - 451.
- \*Ramel, C and Magnusson J (1979). Chemical induction of nondisjunction in *Drosophila*. *Environmental Health Perspective*, 3, p59 - 66.
- \*Ravnskov, U (1992). Cholesterol lowering trials in coronary heart disease: Frequency of citation and outcome. *British Medical Journal*, 305, p15 - 19.
- \*Reimer, W and Tittelbach, U (1989). Verhalten von Herzfrequenz, Blutdruck und systolischen Zeitintervallen in Ruhe und während einhandarbeit bei bleiexponierten und Kontrollpersonen. *Z Gesamte Hyg* 35:491-492.
- \*Rhodes, D., Spiro, A., Aro, A., et al. (2003). Relationship of bone and blood lead levels to psychiatric symptoms: The normative aging study. *Journal of Occupational and Environmental Medicine*, 45, p1144 - 1151.

\*Robison, S.H., Cantoni, O and Costa, M (1984). Analysis of metal-induced DNA lesions and DNA-repair replication in mammalian cells. *Mutation Research*, 131, p173 - 181.

Rockhold, W.T. (1955). Toxicity of Naphthenic Acids and their metal salts. *AMA Arch. Ind. Health*, 12(5) 477 - 482

\*Roels, H.A., Lauwerys, R., Konings, J., et al. (1994). Renal function and hyperfiltration capacity in lead smelter workers with high bone lead. *Occupational and Environmental Medicine*, 51, p505 - 512.

Roels, H and Lauwerys, R (1987). Evaluation of dose-effect and dose-response relationships for lead exposure in different Belgian population groups (fetus, child, adult men and women). *Trace Elements in Medicine*, 4 (2), p80 – 87.

\*Ronis, M.J.J., Gandy, J and Badger, T (1998). Endocrine mechanisms underlying reproductive toxicity in the developing rat chronically exposed to dietary lead. *Journal of Toxicology and Environmental Health*, 54, p77 - 99.

\*Ronis, M.J.J., Badger, T.M., Shema, S.J., et al. (1996). Reproductive toxicity and growth effects in rats exposed to lead at different periods during development. *Toxicology and Applied Pharmacology*, 136, p361 - 371.

\*Rosen, J.F and Chesney, R.W (1983). Circulating calcitriol concentration in health and disease. *Journal of Pediatrics*, 103, p1 - 7.

\*Rosen, J.F., Zarate-Salvador, C and Trinidad, E.E (1974). Plasma lead levels in normal and lead-intoxicated children. *Journal of Pediatrics*, 84, p45 - 48.

\*Rosenkranz, H.S and Poirier, L.A (1979). Evaluation of the mutagenicity and DNA-modifying activity of carcinogens and noncarcinogens in microbial systems. *Journal of the National Cancer Institute*, 62, p873 - 892.

\*Rothenberg, S.J., Manalo, M., Jiang, J., et al. (1999a). Maternal blood lead level and blood pressure during pregnancy in South Central Los Angeles. *Archives of Environmental Health*, 54, p151 - 157.

\*Rummo, J.H., Routh, D.K., Rummo, N.J, et al. (1979). Behavioral and neurological effects of symptomatic and asymptomatic lead exposure in children. *Archives of Environmental Health*, 34, p120 - 125.

Sahu, R.K., Katsifis, S.P, Kinney, P.L and Christie, N.T (1989). Effects of nickel sulfate, lead sulfate, and sodium arsenite alone and with UV light on sister chromatid exchanges in cultured human lymphocytes. *Journal of Molecular Toxicology*, 2, p129 – 136.

Sallmen, M., Lindbohm, M.L., Anttila, A., Taskinen, H and Hemminki, K (2000). Time to pregnancy among the wives of men occupationally exposed to lead. *Epidemiology*, 11 (2), p141 - 147.

\*Sallmen, M., Anttila, A., Lindbohm, M-L., et al. (1995). Time to pregnancy among women occupationally exposed to lead. *Journal of Occupational and Environmental Medicine*, 37, p931 – 934.

\*Sarto, F., Stella, M and Acqua, A (1978). [Cytogenic studies in 20 workers occupationally exposed to lead.] *Medicina del Lavoro*, 69, p172 - 180. (Italian)

- \*Sato, K., Fukuda, Y., Torii, K and Katsuno, N (1981). Epidemiological study of workers engaged in the manufacture of chromium compounds. *Journal of Occupational Medicine*, 23, p835 – 838.
- \*Schwartz, J., Landrigan, P.J., Feldman, R.G., et al. (1988). Threshold effect in lead-induced peripheral neuropathy. *Journal of Pediatrics*, 112, p12 - 17.
- \*Selevan, S.G., Landrigan, P.J., Stern, F.B., et al. (1985). Mortality of lead smelter workers. *American Journal of Epidemiology*, 122, p673 - 683.
- \*Seppalainen, A.M., Hernberg, S., Vesanto, R., et al. (1983). Early neurotoxic effects of occupational lead exposure: A prospective study. *Neurotoxicology*, 4, p181 - 192.
- \*Schmid, E., Bauchinger, M., Pietruck, S., et al. (1972). [Cytogenic action of lead in human peripheral lymphocytes *in vitro* and *in vivo*.] *Mutation Research*, 16, p401 - 406. (German)
- \*Schuhmacher, M., Paternain, J.L., Domingo, J.L., et al. (1997). An assessment of some biomarkers indicative of occupational exposure to lead. *Trace Elements and Electrolytes*, 14(3), p145 - 149.
- \*Schwanitz, G., Gebhart, E., Rott, H.D., et al. (1975). [Chromosome investigations in subjects with occupational lead exposure.] *Deutsche Medizinische Wochenschrift*, 100, p1007 - 1011. (German)
- \*Schwanitz, G., Lenhert, G and Gebhart, E (1970). [Chromosome damage after occupational exposure to lead.] *Deutsche Medizinische Wochenschrift*, 95, p1630 - 1641. (German)
- \*Schwartz, J (1995). Lead, blood pressure, and cardiovascular disease in men. *Archives of Environmental Health*, 50, p31 - 37.
- \*Schwartz, B.S and Stewart, W.F (2000). Different associations of blood lead, meso 2,3-dimercaptosuccinic acid (DMSA)-chelatable lead, and tibial lead levels with blood pressure in 543 organolead manufacturing workers. *Archives of Environmental Health*, 55, p85 - 92.
- Schwartz, J., Landrigan, P.J., Baker, E.L., Orenstein, W.A and von Lindern, I.H (1990). Lead-induced anemia: Dose-response relationships and evidence for a threshold. *American Journal of Public Health*, 80 (2), p165 – 168.
- SCOEL (2002) Recommendation of the Scientific Committee on Occupational Exposure Limits for Lead and its Inorganic Compounds, SCOEL/SUM/83
- \*Sharp, D.S., Benowitz, N.L., Osterloh, J.D., et al. (1990). Influence of race, tobacco use, and caffeine use on the relationship between blood pressure and blood lead concentration. *American Journal of Epidemiology*, 131(5), p845 - 854.
- \*Sharp, D.S., Smith, A.H., Holman, B.L., et al. (1989). Elevated blood pressure in treated hypertensives with low-level lead accumulation. *Archives of Environmental Health*, 44, p18 - 22.
- \*Sharp, D.S., Osterloh, J., Becker, C.E., et al. (1988). Blood pressure and blood lead concentration in bus drivers. *Environmental Health Perspectives*, 78, p131 - 137.
- \*Sharper, A.G., Pocock, S.J., Walker, M., et al. (1981). British regional heart study: Cardiovascular risk factors in middle-aged men in 24 towns. *British Medical Journal*, 283, p179 - 186.
- \*Sheffet, A., Thind, I., Miller, A.M and Lourina, D.B (1982). Cancer mortality in a pigment plant utilizing lead and zinc chromates. *Archives of Environmental Health*, 37, p44 – 52.

\*Shiau, C.Y., Wang, J.D and Chen, P.C (2004). Decreased fecundity among male lead workers. *Occupational and Environmental Medicine*, 61, p915 – 923.

SI Chemical Data 3rd Edition, Aylward, G and Findlay, T. 1994 John Wiley and Sons, Brisbane Aust.

\*Simmon, V.F (1979a). *In vitro* assays for recombinogenic activity of chemical carcinogens and related compounds with *Saccharomyces cerevisiae* D3. *Journal of the National Cancer Institute*, 62, p901 - 909.

\*Simmon, V.F (1979b). *In vitro* mutagenicity assays of chemical carcinogens and related compounds with *Salmonella typhimurium*. *Journal of the National Cancer Institute*, 62, p893 - 899.

\*Sirover, M.A and Loeb, L.A (1976). Infidelity of DNA synthesis *in vitro*: Screening for potential metal mutagens or carcinogens. *Science*, 194, p1434 - 1436.

\*Sjogren, B (1980). A retrospective cohort study of mortality among stainless steel welders. *Scandinavian Journal of Work, Environment and Health*, 6, p197 – 200.

\*Smith, M., Delves, T., Tansdown, R., et al. (1983). The effects of lead exposure on urban children: The Institute of Child Health/Southampton study. *Developmental Medicine and Child Neurology* 25 (suppl 47), p1 - 54.

\*Smith, F.L II., Rathmell, T.K and Marcil, G.E (1938). The early diagnosis of acute and latent plumbism. *American Journal of Clinical Pathology*, 8, p471 - 508.

\*Sokas, R.K., Simmens, S., Sophar, K., et al. (1997). Lead levels in Maryland construction workers. *American Journal of Industrial Medicine*, 31, p188 - 194.

\*Spivey, G.H., Baloh, R.W., Brown, C.P., et al. (1980). Subclinical effects of chronic increased lead absorption--a prospective study: III. Neurologic findings at follow-up examination. *Journal of Occupational Medicine*, 22, p607 - 612.

\*Staessen, J.A., Buchet, J-P., Ginucchio, G., et al. (1996a). Public health implications of environmental exposure to cadmium and lead: An overview of epidemiological studies in Belgium. *Journal of Cardiovascular Risk*, 3, p26 - 41.

Staessen, J.A., Roels, H and Fagard, R (1996b). Lead exposure and conventional and ambulatory blood pressure: A prospective population study. *Journal of the American Medical Association*, 275 (20), p1563 – 1570.

\*Staessen, J.A., Lauwerys, R.R., Bulpitt, C.J., et al. (1994). Is a positive association between lead exposure and blood pressure supported by animal experiments? *Current Opinion in Nephrology and Hypertension*, 3 (3), p257 - 263.

\*Staessen, J.A., Lauwerys, R.R., Buchet, J.P., et al. (1992). Impairment of renal function with increasing blood lead concentrations in the general population. The cadmibel study group. *New England Journal of Medicine*, 327 (3), p151 - 156.

\*Staessen, J.A., Yeoman, W.B., Fletcher, A.E., et al. (1990). Blood lead concentration, renal function, and blood pressure in London civil servants. *British Journal of Industrial Medicine*, 47, p442 - 447.

\*Staessen, J.A., Bulpitt, C.J., Roels, H., et al. (1984). Urinary cadmium and lead concentrations and their relation to blood pressure in a population with low exposure. *British Journal of Industrial Medicine*, 41, p241 - 248.

\*Staessen, J.A., Dolenc, P., Amery, A., et al. (1983). Environmental lead exposure does not increase blood pressure in the population at large: Evidence from the Cadmibel study. *Journal of Hypertension*, 11 (Suppl 2), S35 - S41.

Standards Australia (1995) Children's Toys (Safety Requirements); Part 3: Toxicological Requirements [AS 1647.3]

Standards Australia (2003) Safety of toys - Migration of certain elements; Part 3: Industrial Applications [AS/NZS ISO 8124.3]

Standards Australia (1995) Guide to Lead Paint Management; Part 1: Industrial Applications [AS no. 4361.1]

Standards Australia (1998) Guide to Lead Paint Management; Part 2: Residential and Commercial Buildings [AS no. 4361.2]

\*Steenland, K and Boffetta, P (2000). Lead and cancer in humans: Where are we now? *American Journal of Industrial Medicine*, 38, p295 - 299.

\*Steenland, K., Selevan, S and Landrigan, P (1992). The mortality of lead smelter workers: An update. *American Journal of Public Health*, 82, p1641 - 1644.

\*Stern, R.M (1983). Assessment of risk of lung cancer for welders. *Archives of Environmental Health*, 38, p148 - 155.

\*Stollery, B.T., Broadbent D.E., Banks H.A., et al. (1991). Short term prospective study of cognitive functioning in lead workers. *British Journal of Industrial Medicine*, 48, p739 - 749.

\*Stollery, B.T., Banks, H.A., Broadbent, D.E., et al. (1989). Cognitive functioning in lead workers. *British Journal of Industrial Medicine*, 46, p698 - 707.

\*Stuik, E.J (1974). Biological response of male and female volunteers to inorganic lead. *Internationales Archiv for Arbeitsmedizin*, 33, p83 - 97.

\*Sun, L., Hu, J., Zhao, Z., et al. (2003). Influence of exposure to environmental lead on serum immunoglobulin in preschool children. *Environmental Research*, 92, p124 - 128.

SUSDP (2006) Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) No. 21 Published June 2006, National Drugs and Poisons Schedule Committee (NDPSC)

\*Sweeney, M.H., Beaumont, J.J., Waxweiler, R.J and Halperin, W.E (1986). An investigation of mortality from cancer and other causes of death among workers employed at an East Texas chemical plant. *Archives of Environmental Health*, 41, p23 - 28.

\*Tachi, K., Nishimae, S and Saito, K (1985). Cytogenic effects of lead acetate on rat bone marrow cells. *Archives of Environmental Health*, 40, p144 - 147.

\*Their, R., Bonacker, D., Stoiber, T., et al. (2003). Interaction of metal salts with cytoskeletal motor protein systems. *Toxicology Letters*, 11, p75 - 81.

Torres-Sanchez, L.E., Berkowitz, G., Lopez-Carrillo, L., Torres-Arreola, L., Rios, C and Lopez-Cervantes, M (1999). Intrauterine lead exposure and preterm birth. *Environmental Research*, 81, p297 - 301.

\*Tola, S., Hernberg, S., Asp, S., et al. (1973). Parameters indicative of absorption and biological effect in new lead exposure: A prospective study. *British Journal of Industrial Medicine*, 30, p134 - 141.

- \*Triebig, G., Weitle, D and Valentin, H (1984). Investigations on neurotoxicity of chemical substances at the workplace: V. Determination of the motor and sensory nerve conduction velocity in persons occupationally exposed to lead. *International Archives of Occupational and Environmental Health*, 53, p189 – 204.
- \*Tsaih, S.W., Korrick, S., Schwartz, J., et al. (2004). Lead, diabetes, hypertension, and renal function: The normative aging study. *Environmental Health Perspectives*, 112 (11), p1178 - 1182.
- Vaglenov, A., Creus, A., Laltchev, S., Petkova, V., Pavlova, S and Marcos, R (2001). Occupational exposure to lead and induction of genetic damage. *Environmental Health Perspectives*, 109 (3), p295 – 298.
- Vaglenov, A., Carbonell, E and Marcos, R (1998). Biomonitoring of workers exposed to lead. Genotoxic effects, its modulation by polyvitamin treatment and evaluation of the induced radioresistance. *Mutation Research*, 418, p79 – 92.
- \*Valciukas, J.A., Lilis, R., Eisinger, J., et al. (1978). Behavioral indicators of lead neurotoxicity: Results of a clinical field survey. *International Archives of Occupational and Environmental Health*, 41, p217 - 236.
- \*Valverde, M., Fortoul, T.I., Diaz-Barriga, F., et al. (2002). Genotoxicity induced in CD-1 mice by inhaled lead: Differential organ response. *Mutagenesis*, 17 (1), p55 - 61.
- \*Van Esch, E.J and Kroes, R (1969). The induction of renal tumors by feeding basic lead acetate to mice and hamsters. *British Journal of Cancer*, 23, p765 - 771.
- \*Vaziri, N.D and Sica, D.A (2004). Lead-induced hypertension: Role of oxidative stress. *Current Hypertension Reports*, 6, p314 - 320.
- Venier, P., Montaldi, A., Gava, C., Zentilin, L., Tecchio, G., Bianchi, V., Paglialunga, S and Levis, A.G (1985). Effects of nitrilotriacetic acid on the induction of gene mutations and sister-chromatid exchanges by insoluble chromium compounds. *Mutation Research*, 156, p219 – 228.
- \*Verberk, M.M., Willems, T.E., Verplanke, A.J., et al. (1996). Environmental lead and renal effects in children. *Archives of Environmental Health*, 51 (1), p83 - 87.
- \*Verschoor, M., Wibowo, A., Herber, R., et al. (1987). Influence of occupational low-level lead exposure on renal parameters. *American Journal of Industrial Medicine*, 12, p341 - 351.
- \*Wada, O., Yano, Y., Ono, T., et al. (1973). The diagnosis of different degrees of lead absorption in special references to choice and evaluation of various parameters indicative of an increased lead absorption. *Industrial Health*, 11, p55-67.
- \*Ward, N., Watson, R and Bruce-Smith, D (1987). Placental element levels in relation to fetal development for obstetrically normal births: A study of 37 elements: Evidence for the effects of cadmium, lead, and zinc on fetal growth and for smoking as a source of cadmium. *International Journal of Biosocial Research*, 9, p63 - 81.
- \*Wedeen, R.P., Mallik, D.K and Batuman, V (1979). Detection and treatment of occupational lead nephropathy. *Archives of Internal Medicine*, 139, p53 – 57.
- \*Wedeen, R.P., Maesaka, J.K., Weiner, B., et al. (1975). Occupational lead neuropathy. *American Journal of Medicine*, 59, p630 – 641.



- \*Weiss, S.T., Munoz, A., Stein, A., et al. (1988). The relationship of blood lead to systolic blood pressure in a longitudinal study of policemen. *Environmental Health Perspectives*, 78, p53 - 56.
- \*Weiss, S.T., Munoz, A., Stein, A., et al. (1986). The relationship of blood lead to blood pressure in longitudinal study of working men. *American Journal of Epidemiology*, 123, p800 - 808.
- \*Weisskopf, M.G., Wright, R.O., Schwartz, J., et al. (2004). Cumulative lead exposure and prospective change in cognition among elderly men: The VA Normative Aging Study. *American Journal of Epidemiology*, 160(12), p1184 - 1193.
- WHO (1995). *IPCS Environmental Health Criteria 165 – Inorganic lead*. World Health Organisation, Geneva
- \*Wildt K., Berlin, M and Isberg, P.E (1987). Monitoring of zinc protoporphyrin levels in blood following occupational lead exposure. *American Journal of Industrial Medicine*, 12, p385 - 398.
- \*Willems, M.I., Deschepper, G.G., Wibowo, A.A.E., et al. (1982). Absence of an effect of lead acetate on sperm morphology, sister chromatid exchange or on micronuclei formation in rabbits. *Archives of Toxicology*, 50, p149 - 157.
- \*Wong, O and Harris, F (2000). Cancer mortality study of employees at lead battery plants and lead smelters, 1947-1995. *American Journal of Industrial Medicine*, 38, p255 - 270.
- \*Woźniak, K and Blasiak, J (2003). In vitro genotoxicity of lead acetate: Induction of single and double DNA strand breaks and DNA-protein cross-links. *Mutation Research*, 535, p127 - 139.
- Wright, R.O., Tsaih, S.W., Schwartz, J., Spiro III, A, McDonald, K, Weiss, S.T and Hu H (2003). Lead exposure biomarkers and mini-mental status exam scores in older men. *Epidemiology*, 14 (6), p713 – 718.
- Wu, F-Y., Chang, P-W., Wu, C-C and Kuo, H-W (2002). Correlations of blood lead with DNA-protein cross-links and sister chromatid exchanges in lead workers. *Cancer Epidemiology, Biomarkers and Prevention*, 11, p287 – 290.
- Xie, H., Wise, S.S, Holmes, A.L., Xu, B., Wakeman, T.P., Peluse, S.C., Singh, N.P and Wise, J.P, Sr., (2005). Carcinogenic lead chromate induces DNA double-strand breaks in human lung cells. *Mutation Research*, 586, p160 – 172.
- Xu, J., Wise, J.P and Patierno, S (1992). DNA damage induced by carcinogenic lead chromate particles in cultured mammalian cells. *Mutation Research*, 280, p129 – 136.
- \*Yeh, J.H., Chang, Y.C and Wang, J.D (1995). Combined electroneurographic and electromyographic studies in lead workers. *Occupational and Environmental Medicine*, 52(6), p415 - 419.
- Zelikoff, J.T., Li, J.H., Hartwig, A., Wang, X.W., Costa, M and Rossman, T.G (1988). Genetic toxicology of lead compounds. *Carcinogenesis*, 9 (10), p1727 – 1732.