NICNAS

National Industrial Chemicals Notification and Assessment Scheme

Alkyl Phosphate Anti-Valve Seat Recession Additive

Priority Existing Chemical Assessment Report No. 25

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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* (Cwlth) (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 carried out in conjunction with Environment Australia which carry out the environmental assessments.

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 focussing on the assessment of chemicals already in use in Australia in response to specific concerns about their health/or environmental effects.

There is an established mechanism within NICNAS for prioritising and assessing the many thousands of existing chemicals in use in Australia. Chemicals selected for assessment are referred to as Priority Existing Chemicals.

This Priority Existing Chemical report has been prepared by the Director (Chemicals Notification and Assessment) in accordance with the Act. Under the Act manufacturers and 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 this chemical as a Priority Existing Chemical, therefore manufacturers and importers wishing to introduce this chemical in the future need not apply for assessment. However, 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 may be inspected by the public at the library of the National Occupational Health and Safety Commission (NOHSC). Summary Reports are published in the *Commonwealth Chemical Gazette*, which are also available to the public at the NOHSC library.

Copies of this and other Priority Existing Chemical reports are available on the NICNAS website. Hard copies are available free of charge from NICNAS either by using the prescribed application form at the back of this report, or directly from the following address:

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Other information about NICNAS (also available on request and on the NICNAS web site) includes:

- NICNAS Service Charter;
- information sheets on NICNAS Company Registration;
- information sheets on the Priority Existing Chemicals and New Chemical 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 web site of the National Occupational Health and Safety Commission:

http://www.nohsc.gov.au

Overview

Anti-valve seat recession (AVSR) fuel additives were declared as Priority Existing Chemicals for full assessment under the *Industrial Chemicals (Notification and Assessment) Act 1989* on 5 December 2000. They were nominated because of health and environmental concerns due to their increasingly widespread use in automotive lead replacement petrol (LRP). This report concerns the alkyl phosphate additive which is one of four AVSR additives which have been notified for assessment: methylcyclopentadienyl manganese tricarbonyl-, phosphorus-, sodium- and potassium-based AVSR additives.

AVSR fuel additives are available for both industrial and consumer use. They are delivered either by oil companies pre-blending them into unleaded petrol (ULP) (known as bulk LRP addition) or by the vehicle owner purchasing and adding them to ULP (known as aftermarket addition).

The alkyl phosphate additive is manufactured in Australia for export and domestic bulk LRP formulation and imported as two aftermarket alkyl phosphate additive products (Valvemaster™).

The scope of the alkyl phosphate additive assessment of toxicity in this report is limited to that of the kerosene-based product DMA-4, which contains the alkyl phosphate additive in the range 70-90% and is synonymous with the alkyl phosphate additive product manufactured in Australia. Environmental and health hazards addressed in this report also relate to phosphoric acid, which is the postulated combustion by-product of the alkyl phosphate additive.

The natural attrition of older cars requiring AVSR additives means a decreasing AVSR additive market and consequently, the use of AVSR additives including the alkyl phosphate additive is likely to decline with time. The production of, and infrastructure support for, LRP will eventually become economically unviable and aftermarket addition of AVSR additives will be the sole method of providing valve seat protection through fuel. This report considered the occupational health and safety, public health and environmental consequences of two separate scenarios for the use of the alkyl phosphate additive – a "Present Use" scenario assuming 100% market share and present delivery modes and levels of demand and a "2004" scenario assuming attrition of the AVSR additive vehicle fleet, with reduced demand and delivery of the alkyl phosphate additive via aftermarket addition only.

Based on limited data, the alkyl phosphate formulation DMA-4 (synonymous with Valvemaster™ Concentrate) has low acute aquatic toxicity. Spill incidents and leaks to water bodies and land should be managed through existing Federal, State and Territory legislative frameworks and protocols to mitigate adverse effects to the environment. Such accidental incidents may potentially occur during shipment into and out of Australia, bulk transport, handling and storage.

Use of the alkyl phosphate in internal combustion engines and subsequent degradation through combustion indicate that aquatic and terrestrial organisms are unlikely to be exposed to the active component (the alkyl phosphate additive) or the proposed exhaust emission of the combustion derivative, phosphoric acid, at or above levels of concern. A low environmental risk is, therefore, predicted.

Phosphorus, which is found in the alkyl phosphate additive products, is naturally occurring and ubiquitous in the environment. It is an essential nutrient of plants and animals. Phosphorus

pollution, leading to eutrophication and nuisance algal blooms in aquatic ecosystems, is a major environmental issue in Australia, and in general, phosphorus reduction in waterways is the current Australian policy direction. Even so, the predicted incremental increase in phosphorus in the environment from use of fuels containing the alkyl phosphate AVSR additive is unlikely to develop to levels of concern to aquatic environments given the current use and declining future use pattern of AVSR additives. The findings of this assessment have not identified any significant risk to the environment given the current use pattern of fuels containing the alkyl phosphate AVSR additive.

Mammalian toxicity studies of DMA-4 indicate low acute toxicity by oral or dermal exposure and possible irritant effects. The inclusion of kerosene in the formulation can not be discounted as a contributing factor to the irritancy effects. There are insufficient toxicology data for classification of DMA-4 in accordance with the NOHSC *Approved Criteria for Classifying Hazardous Substances*. The inclusion of kerosene in the alkyl phosphate additive presents health hazards such as irritation and lung damage (if swallowed).

As the alkyl phosphate additive is combusted to phosphoric acid, the health hazards associated with the use of the alkyl phosphate additive also include those associated with phosphoric acid. The critical effects from exposure to phosphoric acid relate to its acidic nature and hence corrosive/irritancy effects. The severity of the symptoms for the eye, skin or pulmonary irritation depends, however, on the concentration and length of exposure to the acid.

Minimal occupational exposure to the alkyl phosphate additive is likely for workers involved in the manufacture of the alkyl phosphate additive, manufacture and distribution of LRP or aftermarket fuel additives, and automotive maintenance. Overall, the conclusion is that there is a low occupational risk associated with the alkyl phosphate additive. While dermal exposure to the aftermarket product may potentially cause irritation, such an effect is confounded by the presence of kerosene, which is a known irritant. Occupational exposure is expected to be infrequent, minor and of short duration.

Where automotive usage is ubiquitous, chronic inhalation of phosphoric acid may result, e.g. service stations, car parks and workshops, and particularly in work spaces that are enclosed or poorly ventilated. In the absence of Australian occupational exposure data, a worst-case scenario was considered for phosphoric acid exposure of such workers. This exposure was assessed as low and hence, of low risk.

The public risk of acute dermal reactions in consumers to the aftermarket products is considered low given the small amounts of additive to which people are likely to be exposed and the fact that any spill on the skin is unlikely to remain untreated for long periods.

The potential for acute health effects from the alkyl phosphate additive could occur as a result of accidental ingestion of LRP by adults, e.g. when siphoning LRP, or by children. The health risk following acute accidential ingestion of the alkyl phosphate additive in LRP was considered low. This is because of the low level of the alkyl phosphate additive in LRP and low acute oral toxicity of the alkyl phosphate additive. Accidental ingestion of the alkyl phosphate additive via the aftermarket products similarly does not represent a significant acute health risk for children by virtue of the alkyl phosphate additive content. Ingestion of other constituents such as kerosene and solvent naptha, however, may represent specific heath effects.

Chronic inhalation scenarios were considered for phosphoric acid exposure by the public as a result of the use of the alkyl phosphate additive in LRP. Overall, the public health risks associated with phosphoric acid exposure from the alkyl phosphate additive's combustion in LRP were assessed as low.

While there is insufficient toxicology data for classification of the alkyl phosphate additive and its kerosene-based products, the data gaps are not regarded as significant impediments to an adequate assessement of the risks associated with the current use of the alkyl phosphate additive in LRP.

Contents

Acronyms and Abbreviations

ABS	Australian Bureau of Statistics
ACGIH	American Conference of Governmental Industrial Hygienists
ADG Code	Australian Dangerous Goods Code
ADI	acceptable daily intake
AF	air/fuel ratio
ai	active ingredient
AICS	Australian Inventory of Chemical Substances (NICNAS)
AIP	Australian Institute of Petroleum
ALD	acute lethal dose
AMSA	Australian Maritime Safety Authority
ANSI	American National Standards Institute
ANZECC	Australian and New Zealand Environment and Conservation Council
ANZFA	Australia New Zealand Food Authority
ARMCANZ	Agriculture and Resource Management Council of Australia and New Zealand
AS	Australian Standard
AST	above ground storage tank
ATSDR	Agency for Toxic Substances and Disease Registry
AVE	average exposure estimate
AVSR	anti-valve seat recession
bar	barometric pressure
BPP	bioavailable particulate phosphorus
BP	boiling point
bw	bodyweight
C	carbon
°C	Celsius
C^0_p	standard heat capacity at constant pressure

CAA	Clean Air Act
CAS	Chemical Abstracts Service
CEPA	Canadian Environmental Protection Act
CI	confidence interval
CICAD	Concise International Chemical Assessment Document
cm	centimetre
C_{max}	maximum concentration
COWIPP	Clean Our Waterways Industry Partnership Program
CR	compression ratio
CRCFE	Co-operative Research Centre for Freshwater Ecology
DETR	Department of Environment, Transport and Regions (UK)
DNA	deoxyribonucleic acid
DMMP	dimethyl methylphosphonate
DP	dissolved phosphorus
DSL	Domestic Substances List (Canada)
EA	Environment Australia
EC50	median effective concentration
EC	European Community, or European Commission
ECB	European Chemicals Bureau
ECETOC	European Centre for Ecotoxicology and Toxicology of Chemicals
EHD	estimated human dose
EINECS	European Inventory of Existing Commercial Chemical Substances
ELINCS	European List of Notified Chemical Substances
ERMA	Environmental Risk Management Authority (New Zealand)
EU	European Union
FAO	Food and Agriculture Organization (United Nations)
FCL	full container load
FORS	Federal Office of Road Safety
FTIR	Fourier Transform InfraRed

g	gram
g/m^3	gram per cubic metre
GC	gas-chromatography
GC-MS	gas-chromatography/mass spectrometry
GHS	Globally Harmonised System (of Classification and Labelling of Chemicals)
GLC	gas liquid chromatography
GLP	good laboratory practice
$\Delta_f H^0$	standard enthalpy of formation of the substance at 298 K
HAPs	hazardous air pollutants
hr	hour
H_3PO_4	orthophosphoric acid/phosphoric acid
H_3PO_3	phosphorous acid
HPLC	high-performance liquid chromatography
HQ	hazard quotient
IC(NA) Act	*Industrial Chemicals (Notification and Assessment) Act 1989* (Commonwealth)
IDLH	immediately dangerous to life and health
IMO	International Maritime Organisation
IP	intraperitoneal
IPCS	International Programme on Chemical Safety
IR	infrared
IRIS	Integrated Risk Information System (US EPA)
ISO	International Standards Organisation
IUCLID	International Uniform Chemical Information Database
IUPAC	International Union of Pure and Applied Chemistry
iv	intravenous
K	Kelvin
kg	kilogram
kg/ha/a	kilogram per hectare per annum
kJ	kiloJoule

K_{oc}	organic carbon partition coefficient
K_{ow}	octanol/water partition coefficient
km	kilometre
L	litre
LC50	median lethal concentration
LD50	median lethal dose
LEL	lower explosive limit
LEV	local exhaust ventilation
LOAEL	lowest-observed-adverse-effect level
LOEL	lowest-observed-effect level
LRP	lead replacement petrol
µg	microgram
m	metre
m^3	cubic metre
MegaL	megalitre
mg	milligram
mg/dL	milligram per decalitre
mg/kg	milligram per kilogram
mg/kg bw/d	milligram per kilogram bodyweight per day
mg/L	milligram per litre
mg/m^3	milligram per cubic metre
min	minute
MITI	Ministry of International Trade and Industry (Japan)
mL	millilitre
ML	megalitre
mm	millimetre
MMAD	mass median aerodynamic diameter
mmol/L	millimole per litre
MMT	methylcyclopentadienyl manganese tricarbonyl
MOE	margin of exposure

MOE	Ministry of Environment (Korea)
mol	mole
MSDS	Material Safety Data Sheet
MTDI	maximum tolerable daily intake
N	nitrogen
n	number
NA	not applicable
NAPS	National Air Pollution Surveillance (Canada)
NDPSC	National Drugs and Poisons Schedule Committee
NEPM	National Environment Protection Measure
ng	nanogram
ng/m^3	nanogram per cubic metre
ng/mL	nanogram per millilitre
ng/L	nanogram per litre
NHMRC	National Health and Medical Research Council
NICNAS	National Industrial Chemicals Notification and Assessment Scheme
NIOSH	National Institute of Occupational Safety and Health
NOAEL	no-observed-adverse-effect level
NOEC	no-observed-effect concentration
NOEL	no-observed-effect level
NOHSC	National Occupational Health and Safety Commission
NOS	not otherwise specified
NPI	National Pollutant Inventory (NPI)
NSW EPA	New South Wales Environment Protection Authority
O	oxygen
OECD	Organisation for Economic Cooperation and Development
OH	hydroxyl
OSHA	Occupational Safety and Health Administration (USA)
P	pressure

P	phosphorus
P_4	white phosphorus
PH_3	phosphine
P_2O_5 (or P_4O_{10} in its most stable form)	phosphorus pentoxide
PEC	predicted environmental concentration
PEL	permissible exposure limit (US OSHA)
P_{final}	pressure (final)
$P_{initial}$	pressure (initial)
pKa	dissociation constant
PNEC	predicted no-effect concentration
ppb	parts per billion
PPE	personal protective equipment
ppm	parts per million
PULP	premium unleaded petrol
PVC	polyvinyl chloride
QA	quality assurance
QSAR	Quantitative Structure Activity Relationship
RDA	Recommended Daily Allowance
Re	Reynolds number
REM	reasonable maximum exposure estimate
RfC	reference concentration
RNA	ribonucleic acid
RP/BR	red phosphorus/butyl rubber
RTECS	Registry of Toxic Effects of Chemical Substances (US)
s	second
S^0_{298}	standard entropy of the substance at 298 K
sc	subcutaneous
SEM	Scanning Electron Microscope
STEL	short-term exposure limit

STP	sewage treatment plant
SUSDP	Standard for the Uniform Scheduling of Drugs and Poisons
$t_{1/2}$	half life
T	temperature
TGA	Therapeutic Goods Administration
TLV	Threshold Limit Value (ACGIH)
TM	Trademark
TMP	trimethylphosphate
TOTP	tri-o-tolyl phosphate
TP	total phosphorus
TP/L	total phosphorus per litre
TSCA	Toxic Substances Control Act (US)
TWA	time-weighted average (NOHSC)
γ	ratio of specific heats
UK HSE	United Kingdom Health and Safety Executive
UEL	upper explosive limit
ULP	unleaded petrol
UN Number	United Nations (Identifications) Number
US EPA	United States Environmental Protection Agency
USI	Urban Stormwater Initiative
UST	underground storage tank
VSR	valve seat recession
WHO	World Health Organization

1. Introduction

1.1 Declaration

Anti-valve seat recession (AVSR) fuel additives were declared as Priority Existing Chemicals for full assessment under the *Industrial Chemicals (Notification and Assessment) Act 1989* on the 5 December 2000. They were nominated because of their increasingly widespread use in lead replacement petrol (LRP) and potential adverse effects on the environment and human health.

NICNAS received applications for the following AVSR additives in use in Australia:

- Methylcyclopentadienyl manganese tricarbonyl (MMT)-based;
- Phosphorus-based;
- Sodium-based; and
- Potassium-based.

Each AVSR fuel additive has been assessed individually and separate reports are prepared for each. This present report addresses the use of an alkyl phosphate additive as an AVSR additive.

1.2 Objectives

The objectives of this assessment are to:

- Characterise the chemical and physical properties of the alkyl phosphate additive;
- Determine the current and potential occupational, public and environmental exposure to the alkyl phosphate additive as an AVSR additive;
- Characterise the intrinsic capacity of the alkyl phosphate additive to cause adverse effects on persons or the environment;
- Characterise the risk to humans and the environment resulting from exposure to the alkyl phosphate additive as an AVSR additive;
- Determine the extent to which any risk is capable of being reduced and make recommendations for the management of these risks.

1.3 Sources of information

Consistent with these objectives, this report presents a summary and critical evaluation of the relevant information relating to the potential health and environmental hazards caused by exposure to the alkyl phosphate additive. Relevant scientific data were submitted by the applicants listed in Section 3, obtained from published papers identified in a comprehensive literature search of

several online databases up to 3 January 2003, and retrieved from other sources such as reports and resource documents prepared by overseas regulatory bodies.

1.4 Peer review

This report has been subject to peer review by NICNAS and Environmental Australia (EA) during all stages of the preparation. NICNAS sought expert advice on postulated alkyl phosphate additive combustion by-products from Associate Professor John C. Mackie, School of Chemistry, University of Sydney.

2. Background

The phosphorus-based anti-valve seat recession additive, i.e. alkyl phosphate additive, which is the subject of this Priority Existing Chemical assessment report, was originally developed by DuPont in the USA during the 1960s and 1970s and known worldwide as the carburettor detergent DMA-4 (Octel undated;a).

2.1 What is an anti-valve seat recession additive?

Anti-valve seat recession (AVSR) fuel additives are added to petrol to stop excessive valve seat wear and recession of the valve seat into the automotive engine cylinder head (Figure 1).

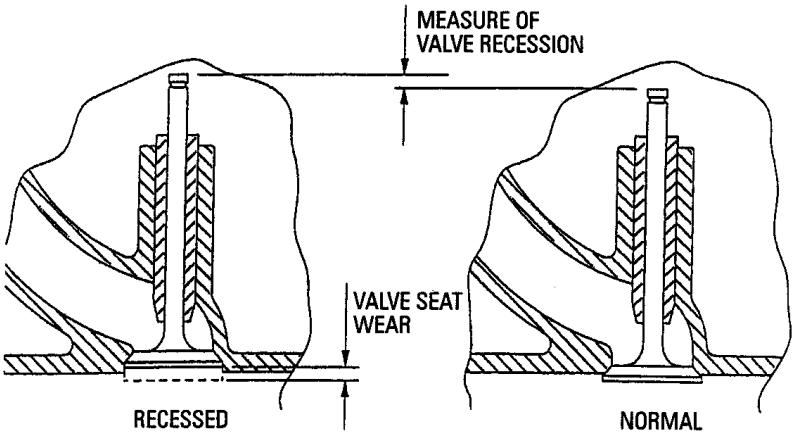


Figure 1. Exhaust valve recession into the cylinder head. From: Barlow (1999)

Although valve seat recession (VSR) occurs as part of the normal wear of an engine, premature erosion of the valve seats are observed as excessive VSR when vehicles with soft exhaust valve seats, normally designed to operate on leaded petrol, are operated on unleaded petrol (ULP).

Valve seats in engines designed for leaded fuel are generally relatively soft. With leaded fuels, lead oxide formed by the combustion of lead alkyls forms a thin layer of lead oxide on the valve and valve seat, so acting as a solid lubricant and preventing erosion of the valve seats in the cylinder head of the engine (Figure 1). VSR can cause valve burning and loss of compression and, if allowed to progress, results in serious loss of performance and ultimately engine failure. Lead replacement petrol (LRP) uses AVSR additives to provide the lubricating qualities previously provided by lead. During fuel combustion, the AVSR additive burns and forms a coating on the exhaust valve seats, providing similar protective lubrication to lead oxide.

Since the early 1970s, increasing environmental and health concerns have resulted in the reduction of lead levels in petrol and the complete removal of leaded petrol in several countries (Lovei, 1998). In 2000, the World Bank reported that 36 countries had already phased out the use of leaded petrol and this was expected to increase to 55 countries by 2005 (Benbarka, 2000). In addition, the use of catalytic converters in

automotive exhaust systems required the introduction of unleaded fuels since lead destroys the capacity of catalytic converters to reduce other pollutants (Lovei, 1998).

A consequence of the removal of lead from petrol is that engine designers have been required to use harder exhaust valve seat materials that maintain integrity without lead lubrication. For existing cars with soft valve seats, the removal of lead has required motorists to use LRP containing an AVSR additive or to modify their engine by fitting hardened exhaust valve seats suitable for ULP with no AVSR fuel additive (Lovei, 1998).

The use of AVSR additives has risen with the demand for LRP petrol resulting from the lead phase out worldwide. The demands for LRP and hence AVSR additives in individual countries have been determined largely by policy decisions regarding the import, sale and retirement of older vehicles, the encouragement of new technology and environmentally cleaner engines and improved petrol standards. Consequently, the population of VSR sensitive cars, and thus demand for AVSR additives in LRP, vary from country to country.

2.2 International perspectives

A variety of petrol additives have been available worldwide to substitute for the lubricating function of lead. These AVSR fuel additives are available for LRP at distribution centres either via the service station pump (bulk supply) or sold in bottles/devices for addition to the petrol tank to treat unleaded petrol (ULP) by the motorist (aftermarket supply).

The gradual worldwide removal of lead from petrol from the early 1970s to the present denotes a diminishing market for AVSR fuel additives - particularly in high-income and middle-income countries (Lovei, 1998). This is based on the observation of older technology petrol vehicles being encouraged to be removed from the world's vehicle population, the gradual attrition/scrappage of vehicles requiring leaded petrol from motor vehicle fleets and the use of increased hardness of exhaust valve seats by auto manufacturers for new vehicles.

The alkyl phosphate additive described in this report has been used in the bulk treatment of petrol in the USA since 1975 (A S Harrison & Co, 2001). Furthermore, the alkyl phosphate additive has been used for bulk treatment of LRP in Argentina, Macau/Hong Kong and the United Kingdom and Ireland since 1994, 1997 and 1999, respectively, and as an aftermarket product in the USA, New Zealand (1996), Singapore (1998), Philippines (1998) and Malaysia (1998) (Octel undated;b).

The chemical listings for the alkyl phosphate additive include the USA (TSCA), Canada (DSL), Europe (EINECS), Japan (MITI) and Korea (MOE). Over 5 billion litres of fuel worldwide is estimated to have been treated with the alkyl phosphate additive (Octel undated;b).

2.3 Australian perspective

In Australia, under the *Fuel Quality Standards Act 2000 (Cwlth)*, lead was removed from automotive fuel from 1 January 2002, requiring the use of alternative additives for valve seat protection. Under this Act, provision is made for the listing of prohibited fuel additives. The alkyl phosphate additive is not listed.

An Australian Standard, AS 4430.1 - 1996 (Standards Australia, 1996), exists for the evaluation of devices and additives which claim to improve vehicle performance. Part 1 of AS 4430.1 – 1996 is noteworthy for the present report as it considers engines designed for leaded fuel to operate on unleaded fuels and includes assessment of valve seat recession.

2.4 Assessment by other national or international bodies

NICNAS has not identified any published hazard or risk assessment for the alkyl phosphate additive although it has been used in LRP on a large scale worldwide for at least the past decade and prior to that as a carburettor detergent known as DMA-4.

Preceding the introduction of the alkyl phosphate additive in New Zealand in 1996, it was subject to assessment by the New Zealand Ministry of Health's Toxic Substance Board and accepted as an AVSR additive at a treatment dose of 600 ppm (mg/kg) or 30 ppm (mg/kg) phosphorus of petrol. Approximately 350 000 vehicles were estimated to require an AVSR additive when leaded petrol supples were withdrawn in New Zealand in 1996 (A S Harrison & Co, 2000). The New Zealand assessment of the alkyl phosphate additive is not publicly available.

3. Applicants

Following the declaration of anti-valve seat recession (AVSR) additives as a Priority Existing Chemical, two organisations applied for assessment of the alkyl phosphate additive. The applicants supplied information on the properties, import quantities and uses of the alkyl phosphate additive. In accordance with the *Industrial Chemicals (Notification and Assessment) Act 1989*, NICNAS provided the applicants with a draft copy of the report for comment during the correction/variation phase of the assessment.

The applicants were as follows:

A S Harrison & Co Pty Limited

PO Box W2

Warringah Mall

NSW 2100

Asia Pacific Speciality Chemicals Limited

15 Park Road

Seven Hills

NSW 2147

4. Chemical Identity and Composition

4.1 Chemical identity

The chemical name, CAS number, molecular and structural formulae, molecular weight, spectral data and details of the chemical composition of the alkyl phosphate additive have been exempted from publication in this report.

The alkyl phosphate additive is not isolated *per se* but manufactured in situ with varying amounts of kerosene used as a hydrocarbon diluent depending on the proprietary product. The chemical composition of the alkyl phosphate additive products described in this report is given in Table 1 (Asia Pacific Specialty Chemicals 2000a, 2000b).

Valvemaster™ Concentrate is synonymous with the carburettor detergent DMA-4 (Octel undated;a). The chemicals contained in the dye mixture used in the described product are listed on the Australian Inventory of Chemical Substances (AICS) and are typically used in hydrocarbon products.

Table 1 - Chemical composition of the alkyl phosphate additive products

Ingredients	CAS No.	Proportion	Use
	Valvemaster™ Concentrate		
kerosene	8008-20-6	10-30%	Bulk LRP (Valvemaster™ Concentrate) & 10 mL single-use aftermarket product
alkyl phosphate additive	confidential	70-90%	
	Valvemaster™ VM11		
kerosene	8008-20-6	1-15%	250 mL multi-use aftermarket product (Valvemaster™ VM11)
solvent naptha	64742-94-5	30-60%	
alkyl phosphate additive	confidential	30-60%	
dye mixture	Mixture	< 0.5% (total)	

5. Physical and Chemical Properties

No physical or chemical properties are available for the alkyl phosphate additive because it is manufactured in situ during the formulation of the proprietary products and never isolated. The physical and chemical properties described below relate to the proprietary products described in Section 4.

5.1 Physical state

Valvemaster™ Concentrate is a pale yellow to colourless liquid. Valvemaster™ VM11 is an orange liquid.

Physical properties of the alkyl phosphate additive products

Property	Value
Valvemaster™ Concentrate [a]	
Autoignition temp.	~220 °C
Boiling point	> 149 °C
Melting point	< –20 °C
Density (15°C)	920 kg/m^3
Flash point (closed cup)	72 °C
Water solubility	Very low (practically insoluble)
Vapour pressure	< 0.1 kPa
LEL Flamm. Limit	1% (kerosene)
UEL Flamm. Limit	6% (kerosene)
Viscosity (40°C; centi posie)	~98
	107 (cST)
Phosphorus % w	5.1
Valvemaster™ VM11 [b]	
Autoignition temp.	~220 °C
Boiling point	> 149 °C
Density (15°C)	920 kg/m^3
Flash point (closed cup)	63 °C
Water solubility	Insoluble in water
Vapour pressure	unknown
LEL Flamm. Limit	1% (kerosene)
UEL Flamm. Limit	7% (kerosene)
Viscosity (40°C; centi poise)	4.5

[a]Details are given in references (Octel, 1999b; Asia Pacific Specialty Chemicals, 2000a; Octel, 2000). [b] Details are given in references (Octel, 1999a; Asia Pacific Specialty Chemicals (2003).

5.2 Chemical properties

Stability: The alkyl phosphate additive products are stable when exposed to light.

Hazardous decomposition: The alkyl phosphate additive products may decompose on exposure to heat. Decomposition products may include toxic and irritant fumes containing phosphorus compounds and oxides of carbon (Asia Pacific Specialty Chemicals, 2000a).

Incompatibilities: The alkyl phosphate additive products are incompatible with strong oxidising agents (Asia Pacific Specialty Chemicals, 2000a).

5.3 Physico-chemical hazards

The products are flammable because the alkyl phosphate additive products contain kerosene.

6. Methods of Detection and Analysis

6.1 Identification

No data are available on the detection and analysis of the alkyl phosphate additive other than the determination of specific parameters for the proprietary products. These parameters include the pH value, phosphorus content and infrared (IR) spectrum of product. Specific characteristics such as the elemental composition of the alkyl phosphate additive formulation are routinely determined for quality assurance purposes during the manufacturing process by a wet chemical titration method and inductively coupled plasma (ICP) instrumental techniques.

6.2 Atmospheric monitoring methods

None available.

6.3 Biological monitoring methods

None available.

7. Manufacture, Importation and Use

7.1 Manufacture and importation

The alkyl phosphate additive is manufactured as Valvemaster™ Concentrate in situ in a closed reaction vessel and diluted with kerosene to produce the final formulated product as one process.

The manufacture is undertaken in a closed non-aerosol forming process with the alkyl phosphate additive remaining within the reactor vessel. At the completion of the manufacture, the alkyl phosphate additive formulation may be held in the manufacturing vessel or transferred from the manufacturing vessel, through dedicated flexible transfer lines via air pumps into either an on-site holding tank (~200 kg closed head steel drums) or 20-22 tonne nominal weight, bottom discharge ISO tanks for distribution to oil companies for addition to LRP or export.

The alkyl phosphate manufacturing site is a licensed Dangerous Goods storage facility, with the manufacture of alkyl phosphate additive undertaken in a Zone 1, Class 1 plant by a single operator (with two operators per manufacturing cycle).

For synthesis, the reaction vessel is charged with the individual chemical components, with the reaction vessel maintained during the procedure at no greater than 85°C. Nitrogen sparging and the application of a vacuum are required during the process. Also during the process, the operators undertake a number of quality assurance tests that require the sampling of approximatly 250 mL of the alkyl phosphate additive product.

Raw materials are loaded either manually through the reactor hatch (solids) or by vacuum pumps (liquids). There is a dust extraction system located in proximity to the hatch to collect dust particles during the loading of raw materials. The reactor is connected to a scrubber system which treats vapours emitted during the manufacturing process. The reaction vessel is located in a bunded building which would facilitate the containment of spills. The reaction vessel is connected via a bursting disc to a catch tank which would contain product in the event that control of the reaction was lost.

Drums of product are stored in appropriate undercover warehouses at the site of manufacture until dispatched to the oil companies for bulk LRP blending. ISO tanks are dispatched directly from the manufacturing site to the oil companies for bulk LRP blending.

Washings and effluent from the drums and manufacturing plant are transferred to a Trade Waste Treatment plant on the manufacturing site for processing prior to any discharge of effluent off-site.

Manufacture of the alkyl phosphate additive aftermarket products does not occur in Australia but the products are imported into Australia by either seafreight or air.

The imported aftermarket products are stored in warehouses located in state capital cities and supplied directly to retail sites in carton quantities. The 10 mL single-use aftermarket applicators are imported in cartons of 160 units while the multi-treat 250 mL bottles are imported in cartons of 24 units.

7.2 Uses

Bulk LRP

The alkyl phosphate additive is used in Australia by major and independent oil companies for bulk LRP production. Bulk LRP is supplied by the oil companies to service stations.

Addition (or treatment) rates in Australia of the alkyl phosphate for bulk LRP blending vary between 180 to 600 ppm (mg/kg) or 9 to 30 ppm (mg/kg) phosphorus of petrol, depending on the oil company. The typical treatment rate of the alkyl phosphate additive in Australia for bulk LRP is 600 mg/kg of petrol.

Alkyl phosphate-based LRP is blended by oil companies by the addition of the alkyl phosphate additive into the ULP while the ULP is being loaded into petrol tankers prior to distibution to service stations. The methods of use and addition mechanisms are described in Sections 7.6 and 9.5.2, respectively.

Aftermarket LRP

The alkyl phosphate 10 mL single-use applicator and 250 mL multi-treat device are used by the vehicle owner for adding the alkyl phosphate additive directly into a vehicle fuel tank containing ULP. This is decribed as aftermarket use of the alkyl phosphate additive.

The 10 mL single-use applicators and 250 mL multi-treat plastic bottles are used to treat 20 L and 250 L, respectively, of ULP.

The typical treatment rate of the alkyl phosphate additive for aftermarket AVSR application in Australia is equivalent to the treatment rate for the bulk LRP market, i.e. 600 mg/kg of petrol.

The aftermarket products are sold by retail service stations across Australia. While the single-use 10 mL applicators have been available for some time, the introduction of the 250 mL multi-treat bottles is recent and primarily for marine and farm users as well as for VSR sensitive motor vehicles.

The composition of alkyl phosphate additive formulation supplied to the oil companies for use in bulk LRP blending is identical to the formulation used in the 10 mL aftermarket applicators (Section 4). The methods of use of the aftermarket products is described in Section 7.6.2.

7.3 Demand for anti-valve seat recession additives

This section describes scenarios for generic AVSR additive use based on the current and anticipated AVSR additive demand in Australia.

AVSR additive fuel additives are available for both oil refinery or terminal use and consumer use. AVSR fuel additives may be delivered either by pre-blending to ULP at the oil refinery or terminal or purchased separately and added to ULP by the vehicle owner. The total Australian AVSR additive market will be referred to as the ‘LRP market’ in this report.

Following the declaration of AVSR fuel additives as a Priority Existing Chemical, importers and manufacturers of various AVSR fuel additives provided information on

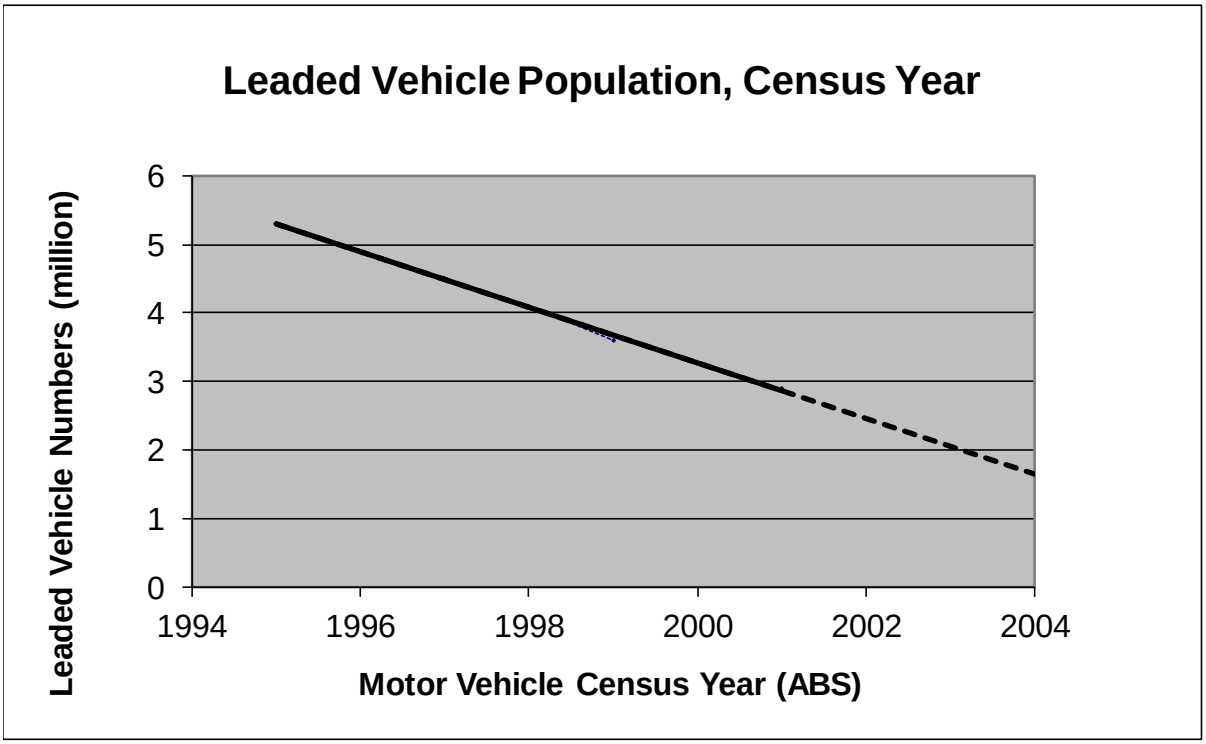
the import and/or manufacturing quantities and uses of their chemicals for the years 2000 and 2001. This information was used to estimate a total LRP market for 2001 of approximately 2500 ML (megalitre). This was calculated using AVSR additive treatment doses for LRP and AVSR additive import/manufacturing volumes as recommended by AVSR additive manufacturers. The calculated figure of 2500 ML is slightly higher than the bulk LRP sales volumes for July 2000 to June 2001 of 1848 ML (Department of Industry, Science and Resources, 2001).

The market share of individual AVSR fuel additives in Australia has not been disclosed in this report due to commercial-in-confidence considerations. An analysis of the import and manufacturing data demonstrated that the aftermarket application of AVSR additives in Australia was less than 10% of the total LRP market in 2001.

In Australia, vehicles requiring leaded petrol are the major consumers of LRP. These vehicles include passenger vehicles, light commercial trucks, rigid trucks, articulated trucks, non-freight carrying trucks, buses and motorcycles (Australian Bureau of Statistics (ABS, 2001). It is likely there are also other VSR sensitive vehicles requiring AVSR additives, e.g. tractors and some plant and equipment engines, not included on the Australian Motor Vehicle Census. These vehicles and engines are not expected, however, to represent a significant component of the AVSR additive market.

There is a declining Australian market for LRP sales (Australian Institute of Petroleum, 1999) and hence AVSR additives. This is due to attrition from the Australian motor fleet of vehicles designed to run on leaded petrol (Figure 2).

Figure 2 - The number of vehicles using leaded petrol 1995-2000 (—) and forecast number **(– – –)** of vehicles using leaded petrol 2001-2004: Australian motor vehicle fleet. Data are taken from the Motor Vehicle Census, Australian Bureau of Statistics (Australian Bureau of Statistics (ABS), 1998, 2001).



By 2004, bulk sales of LRP are expected to decline to less than 5% of total petrol sales (Australian Petroleum Gazette, 1999). This may render the general provision and sale of bulk LRP by the oil refineries and terminals uneconomical. The Australian petroleum industry is yet to announce a phase-out by the oil refineries and terminals of the provision of bulk LRP.

Aftermarket addition of AVSR fuel additives rather than bulk treatment by the oil refineries and terminals is therefore likely to become eventually the only option for motorists with vehicles designed to run on leaded petrol. This may occur as early as 2004 as the supply of LRP from the oil refineries and terminal diminishes significantly. Implementation of any partial or total changeover from bulk to aftermarket supply of LRP would, no doubt, require a broad consensus among stakeholders, entailing consideration of the technical and practical needs of the program and understanding and acceptance by the public.

7.4 AVSR use scenarios

Two use (exposure and emission) scenarios have been assessed in this report – the present state of the market and that likely to occur at 2004. Both scenarios are considered because of anticipated changes in occupational health and safety, public health or environmental exposure as a result of a decreasing supply of bulk LRP as well as the consequent increased use of aftermarket AVSR products and the attrition from the Australia motor vehicle fleet of VSR sensitive vehicles. Details of the AVSR additive use scenarios are presented in Table 2.

Table 2 - A summary of the AVSR additive use scenarios.

Present
Present AVSR additive LRP market: 2500 ML (megalitres) for 2 500 000 vehicles.
10% service station forecourt: 90% bulk AVSR additive market.
2004
AVSR additive LRP market in 2004: 1000 ML (megalitres) for 1 000 000 vehicles.
100% service station forecourt AVSR additive market.

The present use scenario was based on AVSR additive import and manufacturing data provided by industry for the 2001 calendar year. The calculation of 2 500 000 vehicles is based on 2001 calendar year total AVSR additive import and manufacturing data and a petrol fill-up rate of 19.4 L/week/leaded vehicle (Appendix 1).

The calculated figure of 2 500 000 vehicles for the present use scenario (Table 2) is slightly lower than the 2 904 342 vehicles reported in the ABS Motor Vehicle Census 31 March 2001 (Australian Bureau of Statistics (ABS), 2001). This is attributed to the inclusion in the ABS data of all leaded vehicles irrespective of the requirement for or use of an AVSR additive. For example, not all vehicles requiring leaded petrol are VSR susceptible and require an AVSR additive. In 2000, more than 30% of cars built before 1986 were estimated to run efficiently on normal unleaded petrol, with the remaining

70% requiring an AVSR additive (Hill, 2000). The forecast of 1 000 000 vehicles was derived from Australian Bureau Statistics motor vehicle census data (Australian Bureau of Statistics (ABS, 1998, 2001). One million VSR susceptible vehicles equates to a demand for LRP of approximately 1000 ML (megalitres) in 2004. A description of the calculation for LRP demand in 2004 is also given in Appendix 1. The calculated LRP demand of 1000 ML in 2004 is slightly higher than the Australian Institute of Petroleum (AIP) sales forecast made in 1999 of nil to 800 ML (Australian Petroleum Gazette, 1999).

In 2010, a niche market of VSR-sensitive older vehicles and engines requiring leaded petrol is expected to remain (National Heritage Trust, 2000).

For the purposes of commercial-in-confidence and changes in market share, it has been assumed that only one AVSR additive has 100% market share in each use scenario. Across the assessments of all AVSR additives, the same bulk to aftermarket share is assumed for each AVSR additive.

7.5 Use scenarios for the alkyl phosphate additive

Alkyl phosphate additive products currently sold in Australia include Valvemaster™ Concentrate and Valvemaster™ VM11. The chemical composition and addition rates for LRP of these products is described in Section 4 and and Section 7.2, respectively.

The two LRP use scenarios ('present' and '2004') developed in Table 2 have been used to estimate the tonnage of Valvemaster™ Concentrate likely to be used in Australia in these time periods (Table 3).

Due to the commercial-in-confidence nature of the information provided by the Applicants, the calculated tonnage of the alkyl phosphate additive is not disclosed. The estimated tonnage of phosphorus per annum for the two use scenarios is based on 5.1% wt phosphorus in Valvemaster™ Concentrate.

Table 3 - Estimated alkyl phosphate additive use ('present' and '2004')

Scenario 1: Present	
Estimated LRP use per annum (present)	2500 ML (megalitres)
Estimated rate of use of Valvemaster™ Concentrate per annum	10 mL of Valvemaster™ Concentrate per 20 L ULP (or 0.5 mL per L)
Estimated volume of Valvemaster™ Concentrate per annum	1 250 000 L (1.25 ML)
Density of Valvemaster™ Concentrate	0.92 kg/L (at 15°C) (Octel, 1999b)
Estimated tonnage of Valvemaster™ Concentrate per annum	1150 tonnes per annum
Estimated tonnage of phosphorus per annum	57.5 tonnes

Scenario 2: 2004	
Estimated LRP use per annum (2004)	1000 ML (megalitres)
Estimated rate of Valvemaster™ Concentrate per annum	10 mL of Valvemaster™ Concentrate per 20 L ULP (or 0.5 mL per L)
Estimated volume of Valvemaster™ Concentrate per annum	500 000 L (0.5 ML)
Density of Valvemaster™ Concentrate	0.92 kg/L
Estimated tonnage of Valvemaster™ Concentrate per annum	460 tonnes per annum
Estimated tonnage of phosphorus per annum	23 tonnes

7.6 Methods of use

Bulk LRP

The bulk LRP is distributed by oil companies to retail service stations in dedicated petroleum road ISO tankers. The contents of the LRP road tanker are discharged either by gravity or by pump into customer storage tanks. After delivery, the transport contractor conducts an inspection of the interior of the road tankers to ensure the complete discharge of the load into the service station tanks. The road tankers are subsequently cleaned by cleaning contractors using effluent treatment facilities.

Forecourt LRP refueling is typically undertaken by vehicle owners and occurs via a service station forecourt pump or bowser with a direct link to the service station's LRP storage tank. At some service stations, forecourt staff may undertake the refueling activity.

Aftermarket products

Two alkyl phosphate additive aftermarket products are available for consumer use. The aftermarket products are 10 mL and 250 mL formulations that are presented as a single- and multiple-use device, respectively, made from recyclable polyolefin plastic. The alkyl phosphate aftermarket products are available throughout Australia. The chemical composition of the formulations are described in Section 4.

The 10 mL applicators are intended for sale primarily through service station retail sites as a forecourt LRP additive to treat 20 L of ULP. The syringe-like 10 mL plastic applicators are designed to empty completely. As a safety feature, the 10 mL applicator plunger locks in place after use thus preventing refilling with other products. After fueling with ULP the motorist purchases one or more 10 mL applicators and 'injects' the alkyl phosphate additive into the petrol via the fuel tank opening. The motorist disposes of the empty applicator on the service station forecourt in a similar manner to the disposal of empty plastic lubricant bottles.

The 250 mL multi-treat product is designed for treating larger volumes of ULP, e.g. boating or farm applications, as well as for multi-use as a forecourt additive at service

stations. The multi-treat bottle is fitted with a calibrated measuring chamber. To use this device, the customer squeezes the bottle in order to fill the measuring chamber to the level corresponding to the fuel volume to be treated. The contents of the measuring chamber are then dispensed directly into the fuel tanks. The multi-treat bottle is designed to drain completely.

While the 250 mL multi-treat bottles contain a less concentrated form of the alkyl phosphate additive than the 10 mL single-use product, the calibrated measuring chamber is designed to deliver an alkyl phosphate treatment dose equivalent to that of the single-use applicator.

The 10% aftermarket share of the present LRP market (Scenario 1) equates to approximately 13 million single-use alkyl phosphate additive aftermarket applicators Australia-wide (Table 2), assuming all VSR sensitive cars use the single-use product. This number increases in 2004 to 52 million single-use applicators Australia-wide (Scenario 2) when bulk LRP is no longer available (Table 2).

8. Expected Alkyl Phosphate Additive Combustion Emissions

8.1 Introduction

In the absence of consistent data, NICNAS commissioned expert advice on the expected combustion and the final tailpipe emissions by-product(s) of the alkyl phosphate additive as a consequence of the high temperature combustion environment most likely to occur under engine operating conditions.

The advice was sought because of anticipated public and occupational exposure to the combustion by-product(s) of the alkyl phosphate additive. The identification of the thermal combustion products of the alkyl phosphate additive was considered a pre-requisite for determining the risks associated with the use of the chemical.

Choice of the expert advice sought was based on eminence in the field of high temperature kinetics and thermal decomposition studies. Relevant extracts from the advice provided are included in the following sections.

8.1.1 Equilibrium Computational Model

The study on the expected tailpipe emissions from the alkyl phosphate additive is based on an equilibrium computational model and the thermodynamics of the four-stroke spark-ignition engine.

The thermodynamics of the four-stroke spark-ignition engine are well understood (Heywood, 1988) and is described as follows. The intake gases – vaporized fuel, fuel additive and air – enter the cylinder at atmospheric pressure and at a temperature near to 373 K as the cylinder walls are maintained at a constant temperature by pressurised water circulation.

Because the compression stroke is so rapid, there is insufficient time for heat exchange between the air-fuel mixture and the cylinder walls. Hence the compression approximates well to an adiabatic compression. The final pressure, p_{final} after compression is given by:

$$p_{final}/p_{initial} = (CR)^{\gamma} \qquad (1)$$

where CR is the compression ratio and γ is the ratio of specific heats. In the computations used in the model, compression ratios of 8:1 and 11:1 have been considered, representing the two extremes of compression ratios encountered in cars using lead replacement petrol. The typical value of $\gamma = 1.32$ for the intake gases at combustion temperatures has been adopted (Heywood, 1988).

The equilibrium calculations for a 4-stroke spark-ignition engine used in the computational studies were made for adiabatic compressions from an initial temperature of 373 K and initial pressure of 1 bar to a final pressure given by eqn (1), i.e., p_{final} = 15 bar (CR = 8:1) and p_{final} = 23 bar (CR = 11:1). The final combustion temperature was essentially independent of compression ratio but depended on the

air/fuel ratio, *AF*, and computations were made for *AF* values ranging from 12.5:1 to 14:1, corresponding to full throttle and part throttle conditions, respectively.

Equilibrium computations were carried out using the CHEMKIN II (Kee, 1989) and EQUIL (Lutz, 1990) suite of computer codes on UNIX workstations. The exhaust gas temperature range was from 330 K to 1200 K.

The fuel used in the computational studies was iso-octane and the alkyl phosphate additive added at the recommended level of 600 ppm or 600 mg/kg of petrol. The percentage of phosphorus (P) was taken from manufacturers' specifications to be 5.0% giving a level of 30 mg/kg iso-octane or 1.1×10^{-4} moles of phosphorus per mole of iso-octane. Phosphorus-containing species considered at equilibrium were P, P_4 (the molecular formula of elemental phosphorus in its most stable form), PH_3 (phosphine), P_2O_5 (phosphorus pentoxide), PO, PO_2, $P(OH)_2$, $P(OH)_3$, $OP(OH)_2$, HOPO and $HOPO_2$ together with H_3PO_4 (orthophosphoric acid) and H_3PO_3 (phosphorous acid).

Carbon-hydrogen-oxygen-nitrogen species included in the calculations were, in addition to iso-octane, C, H, O, N_2, O_2, OH, CO, CO_2, H_2, H_2O, CH_4, HCO, HO_2, CH_3, C_2H_6, C_2H_4, C_2H_2, NO, CH_2O and H_2O_2. These comprise a comprehensive set of product species known to be present following combustion of iso-octane.

The distribution of products was calculated for the combustion chamber and throughout the exhaust system. The final data on tailpipe emissions is most relevant to the current assessment and is reported here.

8.1.2 Predicted tailpipe emission levels of phosphoric acid

Equilibrium calculations performed for a temperature of 300 K and an air/fuel ratio of 12.5:1 yield a maximum mole fraction of H_3PO_4 (phosphoric acid) of 2.1×10^{-6} in 1 bar of exhaust gases. Treating the exhaust gases as ideal, 1 bar of exhaust gases occupies 0.0249 m^3 mol^{-1} at 300 K. Hence the maximum emission of H_3PO_4 at the tailpipe is calculated to be 8.3 mg m^{-3}. The vapour pressure of H_3PO_4 is stated (MSDS, 2000) to be 0.03 Torr at 20°C corresponding to 160 mg m^{-3} at this temperature. Hence the phosphoric acid emitted from the tailpipe should remain in the vapour phase.

8.1.3 Further consideration of alkyl phosphate by-products in tailpipe emissions

Phosphoric acid tailpipe emission rates

Estimates of the level of phosphoric acid emission from the tailpipe have been made on the basis that all of the phosphorus originally added to the fuel is emitted from the exhaust system. No allowance in these estimates was made for possible heterogeneous reaction between gas phase combustion products containing phosphorus with metal components of the engine or exhaust system. Since the combustion gases reach temperatures exceeding 2000 K in the engine and enter the exhaust system at temperatures around 1200 K, heterogeneous reaction with metal surfaces might take place. This will also depend on the residence time of the combustion gases.

The most likely sites for metal-phosphorus species reaction would appear to be the exhaust valve seating and the top of the exhaust system. It is well known (Corbridge, 1990) that iron and steel and many other heavy metals react with orthophosphoric acid to produce hydrogen phosphates and orthophosphates over a wide range of temperatures. Surface reaction to produce iron phosphates would reduce the level of phosphoric acid in the mainstream (exhaust) gases.

Estimation of the extent of reaction between phosphoric acid in the exhaust gases and the walls of the exhaust system is very complex, depending on the extent of adsorption on the surface, presence of engine oil or other deposits, turbulence of the exhausting gases, rate of surface reaction and the temperatures of surface and gases. Most of the gas will exhaust in the initial blowdown process when the exhaust valve opens (Heywood, 1988). The flow will initially become sonic and hence choked. Pressure pulses will be smoothed out subsequently in the exhaust system and become quasi-steady. Using an average fuel consumption of 8 L/100 km there will be approximately 10 m^3 of exhaust gases per litre of petrol consumed. At an average speed of 100 km hr^{-1} the volumetric flow rate of exhaust gases from the tailpipe is approximately 0.020 $m^3\ s^{-1}$. For a tailpipe of 50 mm internal diameter this corresponds to a linear flow rate of about 10 m s^{-1}. Thus the gases exhaust from the tailpipe significantly less than 1 s after opening of the exhaust valve.

Under these conditions the flow in the exhaust pipe is turbulent (Kanury, 1975) with a Reynolds number, Re ≈ 4000. It is possible to make a maximum estimate of the phosphoric acid that might be able to react with the surface of the exhaust system by assuming turbulent flow with diffusion to the walls of the pipe.

It would be expected that most reaction between phosphoric acid and the metal surfaces of the exhaust would occur in the first 0.1 m when the temperature is high (~1000 K). Approximating the exhaust gases as air and the diffusion coefficient of phosphoric acid in air to be $8 \times 10^{-5}\ m^2\ s^{-1}$ at 1000 K (a value typical of smaller condensible molecules in air) (Kanury, 1975), using the standard formulation (Willeke, 1993) for turbulent diffusional flow through a pipe, it is estimated that the percentage of phosphoric acid molecules diffusing to the walls is 2.6%. If, instead, an average temperature of 700 K is assumed, then the percentage of molecules diffusing to the walls in a distance of 1 m from the exhaust valve is 28%.

If this latter figure is adopted as an upper limit for diffusional losses and if it is assumed that all the phosphoric acid molecules that diffuse to the exhaust pipe walls were capable of reacting and hence removal from the mainstream, this would reduce the emission level of H_3PO_4 from the earlier estimate of 8.3 mg m^{-3} to 6.0 mg m^{-3}. Reaction between phosphoric acid and the metal surfaces of the exhaust system would lead to the production of metal phosphates. From a cast iron exhaust system, iron phosphates would be expected. Part of any metal phosphates produced might deposit on the walls of the exhaust system. However, it is conceivable that some metal phosphates might also be emitted as particulates from the exhaust system.

Reactivity of phosphoric acid

A final consideration concerns the possibility that the combustion process or subsequent passage of the exhaust gases through the exhaust system might lead to the production of the highly toxic gas phosphine, PH_3.

Phosphine was, therefore, included in the phosphorus species considered in the equilibrium computations after combustion of the air-fuel mixture. Its concentration in the combustion products was found to be completely negligible as might be expected owing to its facile oxidation (to H_3PO_4 and H_2O) under combustion conditions. Nevertheless, the possibility that the phosphorus-containing combustion products, especially the oxyacids HOPO, $HOPO_2$, H_3PO_3 or H_3PO_4, might form phosphine in the exhaust system should be discussed. Certainly the equilibrium calculations for the exhaust gases made at cooling temperatures indicate that there are negligible levels of

PH_3, however, the possibility of some catalytic reaction with metal walls leading to PH_3 formation should be considered.

Phosphine is known (Corbridge, 1990) to form by heating dry phosphorous acid, H_3PO_3, at 200°C. Phosphorous acid has the structure $O=P(H)(OH)_2$. It is a tautomer of $P(OH)_3$. $P(OH)_3$ is a species identified by Mackie, Bacskay and Haworth (Mackie, 2002) as having some stability at high temperatures. However, they showed that $P(OH)_3$ rapidly undergoes decomposition to form the more stable $HOPO + H_2O$ rather than tautomerize to phosphorous acid. Hence it is unlikely that there would be any H_3PO_3 (phosphorous acid) of sufficient quantity to make phosphine in the exhaust and this is borne out by the equilibrium calculations which show that it is only present over a limited temperature range and only attains a level of about 0.02 of the orthophosphoric acid at most. The maximum computed mole fraction of phosphorous acid was 1.2×10^{-8} or 0.012 ppm and this maximum is only achieved over a very narrow range of exhaust gas temperatures around 1000 K (727°C). By the time the gases have cooled to 700 K, the computed level of phosphorous acid is only 0.0006 ppm and hence, negligible.

The literature was searched for any reference to the possible formation of PH_3 from H_3PO_4. These were an early Japanese study (Kuwa, 1966) of gases evolved during electrolytic condensation of phosphoric acid in which at high current densities some PH_3 was generated by the reduction of H_3PO_4 and the title only (no abstract) of a Russian publication (Sul'kov, 1975) entitled, "Phosphine Evolution during Production of Furnace-Process Phosphoric Acid". The electrolytic conditions of the first study are irrelevant to a consideration of the exhaust gases; there are no details of the second study available.

The formation of phosphine during phosphoric acid-based metal cleaning is reported in the literature (Patty's 1993). Patty's Industrial Hygeine and Toxicology cites "With regard to this, impurities in the metal may lead to the formation of phosphine". In regard to this claim for phosphoric acid, no evidence to support this can be found in the literature. However, there are several references to the formation of phosphine from phosphorous acid when heated. The most likely explanation for the above claim is that *impure* phosphoric acid containing phosphorous acid, when undergoing exothermic reaction with metals can lead to overheating of the impurity phosphorous acid which could decompose to phosphine.

Orthophosphoric acid is a moderately strong acid and can produce molecular hydrogen when it reacts with many metals (Corbridge, 1990; ChemAlert, 2002). However, there is already hydrogen in the exhaust gases arising from fuel-rich combustion and this level of hydrogen would be significantly higher than any which might be produced from reaction of phosphoric acid with the exhaust pipe.

Certainly conditions in the exhaust gases are reducing – nominally all of the oxygen from air has been burned. However, during the cycling operation there is a period of valve overlap when both intake and exhaust valve are simultaneously open, allowing some air-fuel mixture to flow directly into the exhaust (Heywood, 1988). Additionally there will be some air-fuel mixture in the crevice volume around the edges of the exhaust valve during compression and combustion (Heywood, 1988). Thus oxygen will not be entirely excluded from the exhaust gases. Phosphine is so readily oxidized (Corbridge, 1990) that even if traces were produced, they would most likely be converted into H_3PO_4 and H_2O by residual oxygen present in the exhaust gases.

8.2 Summary of findings

The final equilibrium computations indicate no P (phosphorus), P_4 (white phosphorus) or PH_3 (phosphine) of any significance is expected in the tailpipe emission.

If no account is taken of any reaction between orthophosphoric (phosphoric) acid and the metals of the exhaust valve seat or exhaust system, the level of emission of phosphoric acid (H_3PO_4) at the tailpipe is estimated to be 8.3 mg/m^3 of exhaust gas. Using a model of turbulent flow of the exhaust gases through the exhaust system, an upper limit of losses of H_3PO_4 through reaction with the metals of the exhaust system is estimated to be 28%, leading to a lower limit of 6.0 mg/m^3 emitted from the tailpipe. The orthophosphoric (phosphoric) acid emitted from the tailpipe is expected to remain in the vapour phase, with this level being lowered by atmospheric dilution as it leaves the tailpipe.

NICNAS raised concerns over the hypothetical formulation of phosphine (PH_3) because phosphine forms from phosphorous acid. Maximum levels of phosphorous acid (H_3PO_3) of 0.012 ppm were computed. This level was reached only over a very limited range of exhaust gas temperatures around 1000 K (727°C). When the exhaust gases have cooled to 700 K, the phosphorous acid is computed to drop to negligible levels. Phosphine is readily oxidized so that even if traces were produced, they would most likely be converted into orthophosphoric acid (H_3PO_4) and water (H_2O) by residual oxygen present in the exhaust gases.

9. Exposure

9.1 Environmental exposure to the alkyl phosphate additive

The importation, manufacture, handling, storage, blending and transportation of alkyl phosphate AVSR additives for use in LRP are unlikely to involve large releases into the environment. Such accidental releases are likely to be at managed facilities, or to be of a diffuse nature since LRP is used throughout Australia.

The active alkyl phosphorus-based component is manufactured in situ in a reaction vessel and then diluted into the final formulated Valvemaster™ product as one process. At the completion of manufacture, the manufacturer packs the product into ISO tanks or 200 L closed head steel drums. Product stored in drums is kept in appropriate undercover warehouses at the manufacturing site until dispatched to customers. The manufacturing site is a licensed Dangerous Goods Storage facility and products and raw materials are stored there in accordance with a licence. The full drums are transported around the manufacturing site and loaded and unloaded from delivery vehicles using fork lifts.

Bulk quantities of the alkyl phosphate additive (Valvemaster™ Concentrate) for the production of LRP are supplied to oil companies in 20-22 tonne nominal weight, bottom discharge ISO tanks or in 186 kg steel drums in truckloads of 10-12 drums per load. Since the tanks and drums are transported as sealed containers, there is a low likelihood of environmental release.

After use, empty drums are cleaned and recycled, any residue being removed by the recycling process. A residual amount of Valvemaster™ Concentrate, e.g. 500 mL, may be retained in each drum.

Bulk quantities of Valvemaster™ Concentrate are stored at the manufacturing facility at oil company terminals. Bulk quantities of the alkyl phosphate additive are also transported by road tankers and haul vehicles from storage facilities to customers and retail outlets. Where the alkyl phosphate additive is exported, bulk quantities may be present on ships in Australian waters and at port facilities in ISO tanks and 200 L drums as full container loads (FCLs).

Spillage of Valvemaster™ Concentrate from road tankers may potentially occur during incidents at ISO tank filling, transportation, and emptying operations, although filling and emptying facilities are likely to be located within containment facilities. Potentially, road transportation accidents may result in the loss of concentrated product from ISO tank along transportation routes.

LRP containing diluted Valvemaster™ is not stored in bulk at oil terminals, but is contained in road tankers during transport, at customer and retail service station outlet underground (UST) and above ground (AST) storage tanks.

All pumping and metering at oil terminals during LRP blending is typically conducted under automated control and it is expected that the ISO tanks and the pumping and control equipment associated with the transfer of the alkyl phosphate additive would be installed in bunded areas to contain all leaks or spills. Due to these engineering controls, the release of Valvemaster™ Concentrate or LRP is not expected during routine operations. Spillages resulting from transfer would likely be contained and

diverted to on-site waste treatment facilities and disposed of appropriately in accordance with an approved waste management plan.

Spill incidents involving LRP containing diluted Valvemaster™ may potentially occur from road tankers during transportation to customers and retail services stations and during transfer activities. Minor spillage of LRP may also occur during customer usage from bowsers. Spillage resulting from transfer activities is likely to be managed in accordance with established safety procedures and diverted to site waste containment facilities for collection and off-site waste disposal if present. Such incidents are rare and typically involve small quantities of LRP.

A relatively smaller quantity of the alkyl phosphate additive is currently imported in aftermarket products. Large environmental releases of the alkyl phosphate additive from aftermarket products are unlikely. This may arise due to spillage or residual quantities remaining in the containers following disposal. Most emptied bottles and applicators are disposed of to landfills, although recycling of plastic bottles is also possible. Due to the Australia-wide use of these additives, the associated release of the alkyl phosphate additive from disposal facilities will be diffuse and at low levels.

Physico-chemical properties of the alkyl phosphate additive products are given in Section 4. Valvemaster™ VM11 and Valvemaster Concentrate™ show persistent qualities under typical environmental conditions. Atypical conditions, however, e.g. landfill, increase the rate of degradation. Spills of Valvemaster™ may be persistent in the environment and may enter water and be adsorbed into sediment and soil compartments.

Like other fuels, LRP is typically stored in underground storage tanks (USTs). In general, USTs have a tendency to begin leaking over time, resulting in release of fuel to groundwater. USTs have been installed throughout Australia at terminals and refineries, fuel depots, service stations, and many private facilities and organisations have USTs for fuel storage.

Not all USTs leak. However, many have and have required decommissioning and land remediation. The length of service of the tank is one of a number of factors increasing the risk of UST leakage. Other factors include the type of construction materials, presence of liners, fuel type, fittings/pipes and environmental conditions surrounding the UST. Major fuel suppliers generally have tank decommissioning and replacement programs and install leak detection equipment on their tanks to prevent leaks from occurring and to trigger pollution abatement procedures to minimise risks to the environment where leaks are detected.

Except in the cases of gross spillage of LRP containing the chemical, e.g. leakage from USTs or aboveground spillages, very little release to the soil compartment is likely and apart from areas in the vicinity of such spills and leaks no accumulation of the alkyl phosphate additive is likely in soils.

In the immediate vicinity of leaking USTs, and at LRP spill sites, the alkyl phosphate additive concentration may approximate that of the alkyl phosphate additive in LRP (180-600 mg/L). Site-specific conditions will determine the fate and environmental concentration of the alkyl phosphate additive in groundwater with distance away from leaking UST sources.

9.2 Exhaust release from combustion of the alkyl phosphate additive

The main fate of the alkyl phosphate additive products is likely to be destruction through combustion in internal combustion engines. Most of the alkyl phosphate component of the Valvemaster™ products is destroyed in the cylinders and exhaust trains of motors with the main phosphorus-based compound emitted being phosphoric acid (Section 8).

Based on the current LRP market and assuming an LRP content of 0.5 mL of Valvemaster™ per litre of LRP, the amount of the alkyl phosphate additive in the AVSR additive market in Australia is currently expected to be less than 1150 tonnes per annum as Valvemaster™ Concentrate (Table 3). Corresponding to the expected decline in demand for LRP over time as vehicles requiring AVSR additives are phased out, the amount of alkyl phosphate additive in the AVSR additive market in Australia is expected to decline to 460 tonnes as Valvemaster™ Concentrate in 2004 (Table 3).

Valvemaster™ Concentrate has a total phosphorus content of approximately 4.8% to 5.2%. (5.1%, Octel, 1999b). It is conservative to assume that all the phosphorus is discharged in exhaust emissions. It is indicated that approximately 28% of the phosphoric acid may become entrained in the exhaust system by reaction with metal surfaces (Section 8). Some metal phosphate particulates might also be emitted from exhaust systems (Section 8).

Based on conservative assumptions, i.e. no losses or entrainment of particulate matter in the exhaust system and 5% total phosphorus content, the amount of phosphorus in exhaust emissions may be approximately 57.5 tonnes at present and 23 tonnes in 2004, declining further over time (Table 3). Assuming 28% exhaust entrainment (Section 8), the current and 2004 Australia-wide emission tonnages of phosphorus are reduced to 42.5 and 16.6 tonnes, respectively.

Once emitted, these phosphoric acid vapours and metal phosphate particulates may be transported in air, the fate of which depends on local and regional environmental and climatic conditions. Eventually, precipitation to land or water, or both, is likely.

According to USEPA/USDA (1988), the average vehicle exhaust emissions of CO, HC and NOx from combustion of LRP containing the alkyl phosphate additive were not consistently nor significantly affected by additive use in most cases. Phosphorus emissions will increase as a result of using the alkyl phosphate additive but lead emissions will be less due to the lower lead content of the LRP.

9.3 Fate

This section provides a description of the potential environmental fate of phosphoric acid vapours following exhaust gas emission after combustion of fuels containing the alkyl phosphate additive.

In summary, phosphoric acid vapours are likely to eventually precipitate to land or water, or both, and to integrate with the existing phosphorus biogeochemical cycle in the environment.

This section also describes the fate of the alkyl phosphate addititive in ground water and following disposal to landfill where it forms inorganic phosphate, which will integrate with the existing phosphorus biogeochemical cycle in a similar manner to phosphoric acid.

9.3.1 Phosphorus biogeochemical cycle

The earth's crust contains approximately 0.12% phosphorus, but it does not occur in free form, being found in the form of phosphates in minerals such as chlorapatite, fluorapatite, vivianlite, wavelite and phosphate rock or phosphorite (Merck, 2001).

Phosphorus compounds derived from the combustion of the alkyl phosphate additive are likely to integrate into the existing phosphorus biogeochemical cycle. The phosphorus biogeochemical cycle involves natural and pollutant sources of phosphorus, including biological, organic and inorganic forms. ANZECC and ARMCANZ (2000) indicate that phosphorus is constantly cycled from one form to another within aquatic ecosystems although a fraction may be buried through the process of sedimentation in water bodies or isolated through mineralisation in complexes of low solubility.

Not all phosphorus in the environment is in a bioavailable form. The most bioavailable form of phosphorus is considered to be orthophosphate (PO_4^{3-}). Bioavailable phosphorus is comprised of dissolved phosphorus (DP) and bioavailable particulate phosphorus (BPP). Particulate phosphorus is presumed to be less available, however, for aquatic ecosystems this issue has not been fully resolved (ANZECC and ARMCANZ, 2000).

Biological phosphorus is a key constituent of cellular DNA. The major inorganic phosphorus species include soluble dihydrogen phosphate ion ($H_2PO_4^-$) and monohydrogen phosphate ion (HPO_4^{2-}) and insoluble hydroxyapatite ($Ca_{10}(PO_4)_6(OH)_2$) (Manahan, 1993; ANZECC and ARMCANZ, 2000).

Soil and aquatic processes are very important in the phosphorus cycle. Phosphorus is a common limiting factor in aquatic environments, particularly for the growth of algae. Bacteria are even more effective than algae in taking up phosphate from water and accumulating phosphorus as excess cellular phosphorus that can be released to support additional growth, if the supply of phosphorus becomes limiting in the future (Manahan, 1993).

Phosphorus occurs as organic or inorganic phosphates. Organic phosphates are derived from living plants and animals. Inorganic phosphates may occur naturally and may bond to soil particles. Phosphorus exists in water in both dissolved and particulate forms. Particulate phosphorus includes phosphorus bound up in organic compounds, such as proteins, and phosphorus adsorbed to suspended particulate matter such as clays and detritus (dead and decaying organisms). Dissolved phosphorus includes inorganic orthophosphate ($H_2PO_4^-$, HPO_4^{2-} and PO_4^{3-}), polyphosphates, organic colloids and low molecular weight phosphate esters.

Dead microorganisms release phosphorus that can support additional organisms (Manahan, 1993). Biodegradation of phosphorus compounds is important in the environment for mineralisation (release of inorganic phosphorus from the organic form, thereby providing a source of nutrient orthophosphate) (Manahan, 1993).

Although the percentage of phosphorus in plants is low, it is an essential component. Phosphorus must be present in a simple inorganic soluble form before it can be taken in plants. Utilisable forms for plants include forms of the orthophosphate ion. In the pH range present in most soils, these forms predominantly include $H_2PO_4^-$ and HPO_4^{2-}, with $H_2PO_4^-$ dominant in slightly acidic soils and HPO_4^{2-} dominant in slightly alkaline soils.

In acidic soils, phosphate ions are precipitated or adsorbed by species of aluminium and iron:

$$Al(OH)_3 + H_2PO_{4\ (soluble)}^- \rightarrow Al(OH)_2HPO_{4\ insoluble} \quad (1)$$

In alkaline soils, orthophosphate may react with calcium carbonate to form relatively insoluble hydroxyapatite ($Ca_{10}(PO_4)_6(OH)_2$).

In agricultural systems, phosphorus compounds (including phosphoric acid) are applied to enhance soil nutrition for crop and pasture growth. In general, because of sorption and precipitation, little phosphorus applied as fertilizer leaches from the soil. However, agricultural fertilizer runoff has led to phosphate pollution issues (Manahan, 1993).

In soil, the contribution of phosphorus from fallout from the combustion of the alkyl phosphate additive is expected to be low but bioavailable and to be taken up by opportunistic organisms. A fraction may also complex with metals, e.g., aluminium and iron, and adsorb to soil particles. A portion may partition into runoff, leading to discharge into aquatic receiving environments.

Phosphoric acid will also precipitate from air to water in a bioavailable form for uptake by opportunistic aquatic organisms, e.g. phytoplankton. Sediments can accumulate phosphorus compounds and recycling into the water column can occur through resuspension and diffusion under certain environmental conditions. A fraction may be isolated through the process of sedimentation.

Phosphoric acid is highly miscible in water. Water solubility is estimated at 750 to 850 g/L at 20°C. Phosphoric acid in water is likely to dilute due to mixing and dispersion. The concentration depends on the quantity in the water and the environmental conditions at the time. Natural water hardness minerals, e.g. calcium, magnesium, iron and aluminium, in water bodies are likely to buffer against a reduction in pH (Equation 1). The phosphorus component may persist in the biogeochemical cycle (Environment Canada, 1981, as cited in ECB, 2000).

Sediments (both bottom and suspended) play a significant role in the cycling of nutrients. Phosphorus exists as phosphates, both monomeric and polymeric. In sediments, phosphorus is usually bound with iron (ANZECC and ARMCANZ, 2000). Considerable phosphorus (and nitrogen) can also be bound by bacteria. Under anaerobic conditions, phosphate can be released from sediments into the overlying water, but the general understanding of the processes that drive the cycling of nutrients through sediments is poor (CSIRO, 2000).

Davis et al (1974) introduced radiotracer phosphoric acid into water, which was rapidly accumulated in the sediment, with most becoming bound to iron and aluminium. Bioturbation by sediment-dwelling organisms increased the rate of accumulation and depth of infiltration of phosphorus into the sediment, as did diffusion.

9.3.2 Environmental biodegradation and bioaccumulation

Phosphorus is an essential nutrient of microorganisms, plants and animals. Phosphoric acid is readily utilisable by terrestrial and aquatic plants and microorganisms. Phosphorus compounds are an important component of living matter and all organisms contain a certain quantity; however, excessive bioaccumulation is unlikely due to natural metabolic processes for essential nutrients (Frausto da Silva & Willimans, 1991).

9.3.3 Landfill disposal and fate of the alkyl phosphate additive

Woodward-Clyde (1996) has reviewed the potential likely effects of the disposal of retained Valvemaster™ Concentrate in spent applicators in a typical landfill setting. In summary, the alkyl phosphate additive is not expected to be highly mobile in landfills and is expected to degrade. Cleavage of the phosphate alkyl bond will occur under the mildly acidic conditions typically occurring in landfill and will result in the formation of an aliphatic chain and the remaining alkyl phosphate. Such hydrolysis of the alkyl group is likely to occur rapidly once the alkyl acid phosphate compounds are exposed to water and progressive loss of the alkyl chains to form inorganic phosphate is likely. Of these resulting products, orthophosphate is soluble in water and will exist in the di-protonated form in the pH range of approximately 2.5 to 7. The severed aliphatic chain will remain insoluble in water (Woodward-Clyde, 1996).

The Woodward-Clyde proposal for the biodegradation of the alkyl phosphate additive in mildly acidic conditions is consistent with the general reactivity of organophosphorus compounds. The rate of degradation or reactivity of the alkyl phosphate additive will depend on the nature of the nucleophiles in the landfill and on the leaving groups on the alkyl phosphate additive as well as the acidity of the landfill. Under anaerobic landfill conditions, the aliphatic chains of the alkyl acid phosphate compounds will have a maximum half life of approximately 16 to 20 weeks (Woodward-Clyde, 1996). Aliphatic chains will be readily degraded by heterotrophic bacteria. The presence of the amino group in the active component will assist the microbial degradation of the alkyl phosphate. The more mobile orthophosphate compound will be utilised by landfill microorganisms as a nutrient for growth. Phosphate is also strongly adsorbed to soil particulates such as iron and aluminium oxides and hydroxides and aluminosilicate minerals which will reduce the mobility of this compound in the landfill (Woodward-Clyde, 1996).

The quantities of the alkyl phosphate additive sent to landfills for disposal are unlikely to contribute significantly to the total phosphorus already in landfill leachates from a range of other sources. The Woodward-Clyde (1996) study tries to quantify the amount of Valvemaster™ in landfill for use patterns in New Zealand. A similar negligible contribution to total phosphorus in landfill leachate is expected in Australia. Many landfill systems have leachate collection and treatment systems to capture and treat leachate wastes to prevent environmental contamination.

9.3.4 Groundwater and fate of the alkyl phosphate additive

The processes affecting the fate of the alkyl phosphate additive in groundwater are expected to be similar to those in a landfill leachate environment (refer above). The chemical is not expected to be highly mobile in groundwater and is expected to degrade over time by hydrolysis and biodegradation processes.

9.4 Potential environmental concentrations of phosphoric acid

This section provides an estimation of the potential environmental concentrations of the alkyl phosphate additive and phosphoric acid in the environment following the combustion and exhaust emission of fuels containing the alkyl phosphate additive.

9.4.1 Exposure from use

Following combustion and exhaust emission, the atmospheric concentration of phosphoric acid is expected to be low due to the diffuse patterns of use and release across Australia.

The level of atmospheric phosphorus resulting from the combustion of fuel containing the alkyl phosphate additive is dependent on the amount of fuel used, the quantity entrained in exhaust systems and meteorological conditions in the areas where the fuel is used. There are uncertainties associated with each of these factors and in order to make estimates of likely levels of atmospheric phosphoric acid resulting from use of the alkyl phosphate additive in Australian fuel, it is necessary to make some assumptions based on the following consideration.

All estimates are made for Sydney which, with a population of 3.8 million, comprises 20% of the total Australian population of 19 million and covers an area of approximately 1550 km^2. Since it is reasonable to assume that fuel use would approximate population density, it is assumed that 20% of the petrol usage in Australia would be used in Sydney.

Based on expected LRP demand, two AVSR additive use scenarios (Table 3) are examined corresponding to:

- Scenario 1: where the total current Australian use of Valvemaster™ Concentrate is constant at 1150 tonnes per annum for 90% bulk LRP and 10% aftermarket use; and
- Scenario 2: where the total Australian use of Valvemaster™ Concentrate is constant at 460 tonnes per annum in 2004 for 100% aftermarket use with bulk LRP no longer available for sale.

An atmospheric box model approach has been used. Implicit in the box model approach is that emissions are expected to behave as if they are released into a box with horizontal dimensions of the urban area (selected so that there is no significant influx of emissions into the box). Various assumptions can then be made about phosphoric acid accumulation and dispersion of phosphoric acid from the atmospheric box.

Two predicted environmental exposure concentrations for phosphorus and phosphoric acid in the air have been estimated resulting from the current and future use of the alkyl phosphate additive in Australian fuel. These include an average (AVE) estimate and a reasonable maximum exposure (RME) estimate.

For the calculation of the AVE, representing a long-term average exposure concentration, total yearly alkyl phosphate additive use is used to calculate phosphorus and phosphoric acid emissions over each day with assumed daily clearance of accumulated air from the atmospheric box.

The RME calculation represents the phosphorus and phosphoric acid concentration that may potentially accumulate in the air during weather periods of consecutive windless days. This concentration is unlikely to be attained frequently. Information on consecutive windless days in Australian cities is not readily available as this is not a parameter normally monitored. As such, a conservative estimate of 3 consecutive windless days has been used in this assessment.

Exposure Scenario 1: Present Use

RME concentration for phosphorus and phosphoric acid

The estimated LRP market for 2001 is described in Section 7.5 (Table 3). If 20% of this market was to be used in Sydney, this is equivalent to approximately 500 ML (mega litres) of LRP containing approximately 230 tonnes of Valvemaster™ Concentrate. The phosphorus content of Valvemaster™ Concentrate is approximately 5% (Octel, 1999b). This quantity corresponds to approximately 11.5 tonnes of phosphorus.

Phosphoric acid (H_3PO_4) has a molecular weight of approximately 97.994, and phosphorus contributes about 31.61% of this molecular weight. Oxygen contributes approximately 65.31%, and hydrogen 3.08%. As such, the 11.5 tonnes of phosphorus per annum may equate to approximately 36.4 tonnes as phosphoric acid.

It is readily shown that the effective height of the air column over a particular area is 6.15 km (see for example Connell and Hawker, 1986), and so this 11.5 tonnes of phosphorus (36.4 tonnes of phosphoric acid) would potentially be released into an atmospheric volume of 1550 x 6.15 km^3 or approximately 10^{13} m^3. However, the assumption that the phosphoric acid would be homogeneously distributed throughout a 6.15 km air column is unrealistic. A more realistic assumption is to assume that the particles are only distributed in the lowest 615 metres (i.e. 10^{12} m^3).

In order to go any further it is now necessary to make some simplifying assumptions, and while these are not entirely realistic they nevertheless allow for a first approximation to the atmospheric phosphorus level. If it is assumed that the air column is perfectly static, that the particulate matter is homogeneously distributed through the air volume and that none is precipitated with rain or through other mechanisms, then after one year the atmospheric phosphorus level is estimated as 1.15 x 10^{16} ng/10^{12} m^3 = 11 500 ng/m^3. In terms of phosphoric acid concentration, this equates to 3.64 x 10^{16} ng/10^{12} m^3 = 36 400 ng/m^3 of phosphoric acid.

The assumptions made above are considered unrealistic in that no dispersion through wind or by rain is considered. If it is assumed that all the phosphorus emitted each day remains suspended for 3 days without removal, as may potentially occur occasionally following 3 consecutive windless days, then the atmospheric RME concentration could be as high as 95 ng/m^3 phosphorus or 300 ng/m^3 phosphoric acid.

AVE concentration for phosphorus and phosphoric acid

An AVE phosphorus and phosphoric acid concentration in air at ground-level may be estimated taking into account losses due to wind dispersion out of the urban area. The average concentration at any one time within the atmospheric box may be estimated as the influx rate minus the emission rate from the atmosphere box.

An influx of 1.15 x 10^{16} nanograms P/year (3.15 x 10^{13} ng P/day) has been estimated above. Emitted into an air volume of 10^{12} m^3 each day, an average daily air concentration of 31.5 ng/m^3 has been estimated using this model. This equates to 100 ng/m^3 of phosphoric acid (Table 4).

Exposure Scenario 2: 2004

This scenario assumes bulk sales of LRP have declined to 1000 ML as outlined in Section 7.5 (Table 3) which equates to 460 tonnes of Valvemaster™ Concentrate.

Assuming 20% (92 tonnes of Valvemaster™ Concentrate) is used in Sydney, the calculations and assumptions for this scenario are identical to the above. Therefore, with a 20% release rate, approximately 4.6 tonnes per annum of phosphorus (14.6 tonnes of phosphoric acid per annum) may be emitted into the air column (Table 4).

The estimated phosphorus and phosphoric acid atmospheric AVE and RME values for the current and 2004 use scenarios are given in Table 4.

Table 4 - Estimated average and reasonable maximum potential atmospheric levels in Sydney with the use of the Alkyl Phosphate Additive

Use Scenario	Atmospheric Dispersion of Phosphorus Compounds [a]					
	Nil (ng/m^3) [b]		AVE (ng/m^3)		RME (ng/m^3) [d]	
	P	H_3PO_4	P	H_3PO_4	P	H_3PO_4
Scenario 1: Present	11 500	36 400	31.5	100	95	300
Scenario 2: 2004	4 600	14 560	12.6	40	38	120

a. Air column volume of 10^{12} m^3 (615 m high x $1550x10^6$ m^2); b. Assumes no dispersion throughout year (unrealistic); c. AVE (Long Term Average), assumes wind dispersion with daily clearance of atmospheric box; d. RME (Reasonable Maximum Exposure) – Quiescent conditions for 3 days.

Due to the complexities implied by uncertainties in the use rate of the alkyl phosphate additive and the prevailing atmospheric conditions in particular areas, these estimates of the atmospheric phosphoric acid should be treated as indicative only. The level of phosphoric acid in the atmosphere would be dependent on factors such as rain and wind, and it is likely that ambient and prior weather conditions would impact on any particular daily measurement.

9.4.2 Potential release of phosphorus to water compartment

Based on Scenario 1, the annual use in Sydney of approximately 230 tonnes of Valvemaster™ Concentrate would generate a worst case of 36.4 tonnes of phosphoric acid (11.5 tonnes of phosphorus) emissions (Section 9.4.1).

Eventually, the phosphoric acid will precipitate to the earth's surface (land and water) in rainfall or dust where the soluble nature of the compound means that it may leach into soil and dissolve in water. Reactions forming stable compounds with particulate matter, calcium carbonate or metals, e.g. aluminium, iron, are likely to occur in soils and sediments. The bioavailability of the phosphoric acid means that a fraction may be taken up by biota. Thus, a proportion of the phosphorus fallout will be associated with soils and biota.

Atmospheric dispersion associated with weather patterns is also likely to result in movement of phosphoric acid away from source areas.

If it is assumed that Sydney, with a land area of approximately 1550 km^2, receives an average annual rain fall of one metre, then it is possible to estimate the concentration of phosphorus in storm water (assuming no stabilization of phosphorus compounds in soil,

biological uptake, atmospheric dispersion from source areas) as 11.5×10^6 (grams)/(1550×10^6 (m^2) x 1 m) = 0.0074 g/m^3 or 7.4 μg/L in annual stormwater runoff for the present use scenario, and approximately 2.96 μg/L in the year 2004, assuming lower future LRP use rates. By allowing for atmospheric dispersion due to wind, the annual average phosphorus stormwater run-off concentration is likely to be much lower for the present use and 2004 scenarios.

These concentrations are much less than the concentration range of total phosphorus in estuarine and marine waters in southeast Australia, which is approximately 25 to 30 μg/L (ANZECC and ARMCANZ, 2000).

The assumption of discounting soil attenuation processes following atmospheric precipitation of phosphoric acid is unrealistic because of the sorption and precipitation processes that are likely to occur in both alkaline and acidic soils (Manahan, 1993). Assuming 50% of the land surface is developed, i.e. pavement or hard surface other than soil, the above stormwater concentration estimates could be halved. In addition, some atmospheric dispersion is likely to occur, thus contributing to a reduction in the phosphoric acid in the air column of the Sydney region.

Furthermore, the stormwater concentration estimates do not take into account the dilution, mixing and sedimentation that is likely to occur in receiving waters within and beyond stormwater outlet mixing zones or pollution control measures, e.g. sediment traps, which may reduce stormwater concentrations discharged by several orders of magnitude. As such, the concentration estimates above are unlikely to reflect ambient water quality conditions.

9.5 Occupational exposure to the alkyl phosphate additive

Occupational exposure to the alkyl phosphate additive is possible during manufacture of bulk Valvemaster™ Concentrate, transport and handling of bulk Valvemaster™ Concentrate and aftermarket products and the blending, transport and handling of alkyl phosphate additive-based LRP.

Ideally, the assessment of occupational exposure is based on workplace monitoring data or, where such data is inadequate or unavailable, on knowledge-based models that estimate exposure when the various patterns of use and the physical properties of the substance under investigation are known.

Due to the lack of monitoring data and physico-chemical properties for the alkyl phosphate additive itself, quantitative exposure estimates for the additive could not be undertaken. Consequently, a qualitative description of the potential levels of occupational exposure to the alkyl phophate additive are described in the following section.

Furthermore, a quantitative estimate is made of the potential level of occupational exposure to phosphoric acid, which is identified as the combustion and tailpipe emission by-product of the the alkyl phosphate additive (Section 8).

The major routes for occupational exposure to the alkyl phosphate additive are skin and eye contact with the alkyl phosphate additive products or LRP. Inhalation exposure to the alkyl phosphate additive is not regarded as a major route of exposure due to the low vapour pressure of the alkyl phosphate additive products (Section 5).

9.5.1 Manufacture

Occupational exposure in Australia to the alkyl phosphate additive during formulation is limited to the manufacture and formulation in situ of bulk Valvemaster™ Concentrate.

Valvemaster™ Concentrate is manufactured at one plant in Australia and usually takes place twice a month or a total of approximately four working weeks per year. The number of workers involved in the process is typically two operators (8-hour day) per batch, with one laboratory worker involved in the quality assurance testing of the batch.

The occupational exposure levels to the alkyl phosphate additive during the formulation of Valvemaster™ Concentrate are predicted to be low given the fully enclosed formulation process, low vapour pressue of the product and intermittent (twice monthly) formulation schedule.

9.5.2 Bulk LRP blending

Several oil companies are blenders of bulk LRP and hence handlers of AVSR fuel additives.

ISO tanks and drums transported to oil company terminals and depots by road (or rail) will remain unopened prior to use in fuel blending. Consequently, provided these containers are not accidentially punctured, exposure of transport workers to the alkyl phosphate additive is not expected.

At the oil company terminals and depots, bulk Valvemaster™ Concentrate is typically unloaded in the open air by manually connecting a flexible hose to the lower delivery tank flange of the ISO tank via a closed gravity transfer/delivery system or decanted from drums via a hand pump and a spear or a dedicated pumping system into a bulk storage tank. Oil company terminals are characteristically larger operations than depots.

During LRP blending at oil company terminals and depots, Valvemaster™ Concentrate is typically pumped using an automated vapour recovery system from the storage tank and injected directly (in-line dosing) into the petrol stream of the LRP road tanker. At some depot(s), Valvemaster™ Concentrate is decanted manually into a receptacle for addition to the LRP tanker compartment.

During bulk LRP blending and transfer activities workers wear pvc or rubber gloves, safety goggles, shoes and protective clothing. In addition, a respirator with an organic cartridge is used at depot(s) which decant the alkyl phosphate additive manually into the trunk compartment. Typical exposure durations for workers engaged in bulk LRP blending at terminal and depots decanting Valvemaster™ Concentrate from drums or ISO tankers are shown in Table 5.

Exposure of terminal and depot staff to the alkyl phosphate additive may occur during inadvertent spills and slops resulting from the manual handling of transfer lines containing either LRP or Valvemaster™ Concentrate, the manual addition of the alkyl phosphate additive to road tankers and handling of the final blended fuel in the laboratory.

Table 5 - Handling of the alkyl phosphate additive during LRP blending at typical terminals and depots

Category of worker	No. of workers	Description of Work	Hours/day	Days/year
		Terminal		
Terminal staff	2	Decanting (dedicated pump) of ~200 L drums	6	3
Terminal staff	1	Assisting maintenance staff	1	12
Facility operator		Gravity discharge ISO tanker contents into underground tank	2	1
Fitters	1	Connect ISO tanker to additive pump	5 min/2 weeks	25
Tank dipping operator	1	Stock reconcilation	< 0.5	52
Maintenance worker	1	Maintain additive pump injector	4	2
Transport staff	10	Transport of LRP	1	300
Maintenance worker	2	Maintain despatch systems	2	12
		Depot		
Depot labourers	1	Decanting additive to receptacle and into LRP truck compartment	5 min	260
Yardmen	1	Decanting (dedicated pump) of ~200 L drums to storage tank	2 hr/month	12
Depot staff	1	Automated addition to LRP truck compartment	5 min	variable

The exposure levels during bulk LRP blending are predicted to be low given the typically automated and enclosed transfer activities and the use of personal protective equipment (PPE). Non-automated additions of the alkyl phosphate additive at small depots may potentially result in increased occupational exposure for short (5 min) periods compared to automated enclosed additions of the alkyl phosphate additive to the LRP truck compartment.

9.5.3 Petrol stations and maintenance workshops

Scenario 1: Present use

At petrol stations, bulk LRP, which constitutes 90% of the LRP market, will be transferred from road tankers to underground storage tanks. Transfer requires that tanker drivers manually connect and disconnect flexible tansfer lines between the tanker and storage tank. During this process, skin and eye exposure to the diluted alkyl phosphate additive in LRP is possible from slops and spills.

Potential exposure of transport drivers may occur frequently during the day in metropolitan areas with numerous offloads and less frequently during tanker deliveries to regional areas. There is the potential of mainly dermal exposure for petrol station workers undertaking dip measurement of underground LRP tanks. Typically, dipping occurs for up to 10 minutes once per week.

The potential occupational exposure profile to the aftermarket alkyl phosphate additive products for Scenario 1 will be similar for Scenario 2 and is described below.

Scenario 2: 2004

As bulk LRP blending is replaced by forecourt addition by vehicle owners, no occupational exposure to the alkyl phosphate additive by depot and terminal staff and other personnel associated with bulk LRP blending and distribution will occur.

Service station forecourt workers may be exposed if they assist vehicle owners in filling cars; however, this is likely to be infrequent as most service stations are self service and the majority of cars do not use LRP. The extent of the occupational exposure during this activity is likely to be variable, depending on vehicle throughput, average LRP fill rate per vehicle, the customer's preferred choice for blending LRP, i.e. single- or multi-treat applicators, and the amount of alkyl phosphate additive contaminating spent aftermarket devices.

The potential exposure per vehicle fill-up is greater with the 250 mL multi-treat bottle compared with the 10 mL single-use syringe-type aftermarket product. This is because more contamination from spills and slops may occur when using the multi-treat compared with the single-use applicator. The difference is that the treatment volume is delivered from an open mouthed bottle versus direct injection from a syringe into the fuel tank.

Furthermore, the more viscous alkyl phosphate additive formulation contained in the single use applicators, as compared with the multi-treat bottles, would further limit spills and slops. The more concentrated alkyl phosphate formulation in the 10 mL applicators increases, however, the potential alkyl phosphate dose per exposure. In most cases, exposure will be at most once a week (per fill) for short duration, e.g. 5 minutes, and will not be undertaken by workers.

As for both Scenario 1 and 2, automechanics at petrol stations and maintenance workshops may be exposed to the diluted alkyl phosphate additive in LRP during maintenance of automotive fuel systems. The extent of exposure during these activities is likely to be highly variable and to decrease over time as VSR susceptible vehicles are withdrawn from the Australian motor vehicle fleet.

9.5.4 Importation, transport and handling of the alkyl phosphate products

Aftermarket alkyl phosphate additive products are imported in cartons through a single port of entry prior to central warehousing and dispatching by road (and rail) to distribution centres and retail outlets within the states and territories.

The extent of exposure to the alkyl phosphate additive in the aftermarket devices during these activities is expected to be unlikely except if accidental puncturing of the plastic devices occurs.

9.6 Occupational exposure to phosphoric acid from the alkyl phosphate additive use

The combustion of the alkyl phosphate additive produces phosphoric acid exhaust gas emissions (Section 8). Several classes of workers are exposed potentially to phosphoric acid in occupational settings due to emissions from automotive exhaust of vehicles using fuel containing the alkyl phosphate additive. The extent of this exposure will diminish as VSR susceptible cars are gradually removed via attrition from the Australian motor vehicle fleet.

The exposure of petrol station attendants is likely to be highly variable depending on the required duties (purely retail versus petrol pumping), the level of customer traffic, the separation of retail from service areas and the vehicle fleet, i.e. cars using LRP versus ULP.

Automechanics may be particularly exposed to phosphoric acid exhaust emissions when servicing operating autoengines in poorly ventilated workshops. Attendants, security and other personnel who work in enclosed car parks such as underground parking stations, also have a potential for inhalation exposure to phosphoric acid exhaust emissions during routine duties. Like service station workers, exposure during the working day is likely to be highly variable and dependent on the level of, and proximity to, customer traffic and the effectiveness of ventilation of the enclosed parking station.

Professional drivers such as taxi and truck operators and road maintenance workers may also be exposed to phosphoric acid from inhalation of automotive exhaust emissions. Again, the level of exposure is likely to be highly variable and dependent on traffic volume and, in the case of road workers, whether work is on new, uncommissioned or light duty roads or on heavily trafficked arterial roads repaired while in use.

The major routes for occupational exposure to phosphoric acid in exhaust emissions are eye and inhalation. No quantitative data are available regarding personal exposure levels to phosphoric acid as a result of the use of the alkyl phosphate additive based LRP within workplaces.

It is anticipated that occupational exposure of garage mechanics/autorepairers may potentially be high depending on the degree of ventiliation and whether or not the workshop doors are open or closed.

A tailpipe phosphoric acid exhaust emission concentration of 6.0 to 8.3 mg/m^3 is calculated (Section 8) dependent on reaction between phosphoric acid and the metals of the exhaust valve seat or exhaust system.

Atmospheric dilution of tailpipe phosphoric acid exhaust emissions will occur. An estimated atmospheric phosphoric concentration is calculated assuming an exhaust gas dilution factor of 100 (Dolan et. al, 1979) which may occur within two metres of the end of a tailpipe on an idling vehicle. This estimated atmospheric phosphoric concentration of 0.06 to 0.083 mg/m^3 is potentailly reduced by a factor of 3 to 5 as the distance from the tailpipe increases to 4 metres (Dolan et. al, 1979) to give atmospheric phosphoric acid concentrations of 0.02 mg/m^3 to 0.028 mg/m^3 (for a dilution factor of 300) and 0.012 mg/m^3 to 0.017 mg/m^3 (for a dilution factor of 500).

9.7 Public exposure and level of exposure to the alkyl phosphate additive

9.7.1 Consumer exposure

Consumer exposure to the alkyl phosphate additive is likely to occur as a result of contact during refuelling vehicles with LRP, adding aftermarket product to petrol tanks, using LRP as a solvent or cleaner, or substance abuse (petrol sniffing). Direct exposure of skin and eyes to the additive is, therefore, probable.

Due to lack of information, it is difficult to quantify the level of exposure likely to be encountered during consumer end-use of bulk LRP and the aftermarket alkyl phosphate products. Nonetheless, it is expected that consumer exposure is likely to be a regular occurrence for vehicle owners, e.g. weekly, take place under open conditions and involve small quantities per event, as evidenced by the small size of the aftermarket devices and low concentration of the alkyl phosphate additive in LRP.

The low vapour pressure of the concentrated alkyl phosphate additive product (< 0.1 kPa) indicates that the extent of inhalation exposure to the alkyl phosphate additive during filling with LRP is limited. Exposure to the petroleum constituents of LRP is considered to be of greater potential concern than exposure to the alkyl phosphate additive. Further, exposure to alkyl phosphate additive via the aftermarket products is likely to be of greater potential concern than exposure via LRP. This is because the aftermarket products contain higher concentrations of the alkyl phosphate additive than LRP.

Accidental skin and possibly eye exposure to the alkyl phophate additive may occur when refuelling vehicles with LRP and adding aftermarket products to fuel tanks.

Consumer dermal exposure to the alkyl phosphate additive will be highly variable yet regular depending on a vehicle's or an engine's LRP use. Accidental ocular exposure as a result of splashes of LRP and/or aftermarket products is likely to occur only infrequently and involve very small amounts of the alkyl phosphate additive.

In young children, ingestion exposure is generally unlikely, although accidental ingestion may occur if aftermarket products are stored in or around the home. Children aged between one and a quarter and three and a half years can swallow approximately 4.5 mL of liquid (Gosselin et al., 1976). A child (10 kg) ingesting one mL of the

Valvemaster™ Concentrate (5.1% wt phosphorus) single use or multi-treat Valvemaster™ VM11 (2.5% wt phosphorus) aftermarket product would receive an oral dose of 4.7 mg/kg bw phosphorus (equivalent to 14.4 mg/kg bw PO_4^{3-}) and 2.35 mg/kg bw phosphorus (equivalent to 7.2 mg/kg bw PO_4^{3-}), respectively. The ingestion by a child (10 kg) of 1 to 4.5 mL of Valvemaster™ Concentrate represents 9.4% to 42.2% of the recommended daily allowance (RDA) of phosphorus of ~500 mg (National Academies, 2003), for a child aged between 1 and 10 years of age (Section 11). This ingested phosphate dose is halved if the alkyl phosphate additive is obtained from the multi-treat aftermarket device. Aftermarket products are more likely to be stored in garages than homes. Furthermore, the single-use applicator is primarily purchased and immediately used as required at the service station forecourt by the vehicle owner and the multi-treat aftermarket product is fitted with a child resistant closure.

Accidental ingestion of the alkyl phosphate additive in LRP could occur when syphoning petrol. Accidental ingestion by a child could occur also if LRP containing the alkyl phosphate additive is stored in inappropriate containers in or around the home environment. Australian National Hospital Morbidity Data shows approximately 133 hospital discharges annually between 1998 and 2000 were associated with the toxic effects of petroleum products (AIHW, 2002). Victorian data show that there were 75 hospital admissions between 1987 to 1994 involving children below five years of age who were poisoned by petroleum fuels and cleaners including kerosene. Data from a selection of Victorian hospitals showed that there were 16 emergency department presentations between 1989 and 1995 involving children below 5 years of age ingesting petrol. Three of the 16 had syphoned petrol from a car or lawn mower and two had drunk petrol from drink bottles (Ashby and Routely, 1996).

Although no data was available on the amounts of petrol ingested in these cases, it is likely that only small amounts of LRP would be accidentally ingested. Data collected by Watson et. al (1983) shows that the average volume of a swallow (of tap water) for a child up to 5 years of age is between approximately 1 and 7 mL and for a person between 5 and 18 years of age is between 2 and about 30 mL. Given these low amounts of LRP and the low concentrations of the alkyl phosphate (600 mg/kg or 30 mg/kg phosphorus) in LRP, accidential ingestion would involve potentially only very small amounts of the alkyl phosphate additive. With the solvent nature of petroleum products, repeated ingestion or ingestion of larger amounts of LRP, e.g. 100 mL or more, is considered unlikely.

9.7.2 Indirect exposure via environment

As outlined in Section 9.1, exposure to the alkyl phosphate via the environment would be due to incidental or accidental environmental release of the alkyl phosphate additive, which will differ only slightly according to the nature of the application, e.g. bulk application by the oil companies for the production of bulk LRP or aftermarket applications by the general public.

The importation, manufacture, handling, storage and transportation of alkyl phosphate AVSR additives for use in LRP are unlikely to involve large releases of the alkyl phosphate additive into the environment (Section 9.1). Accidental releases are likely to be at managed facilities or of a diffuse nature since LRP is used throughout Australia. Therefore, it is likely that public exposure to the alkyl phosphate additive as a result of atmospheric, soil, water or food contamination would be very low.

9.7.3 Public exposure and level of exposure to phosphoric acid via air

The combustion by-product of the alkyl phosphate additive is phosphoric acid (Section 8), which thus has the potential to increase public exposure to airborne phosphoric acid.

Using atmospheric phosphoric acid concentrations from the most realistic atmospheric dispersion model (Table 4) for Sydney, an estimate can be made for the potential public inhalation exposure to phosphoric acid according to the two use scenarios described in (Table 2). Scenario 1 assumes the LRP market share is maintained at present levels and patterns of use. Scenario 2 assumes the LRP market is reduced in the year 2004 to an aftermarket supply.

No data is currently available on ambient phosphoric acid levels in Australia. Given no significant sources of airborne phosphoric acid can be identifed in Sydney, a negligible mean air concentration of phosphoric acid is assumed as a background level for Sydney. Furthermore, it is assumed that the total estimated phosphoric acid emissions due to the combustion of the alkyl phosphate additive in LRP is respirable phosphoric acid. The estimated atmospheric phosphoric acid levels given for Scenario 1 and 2 (Table 6) represent the estimated increase in air phosphoric acid concentrations and exposures attributable to combustion of the alkyl phsophate additive in LRP.

As a worst case, it could be assumed that indoor and outdoor air concentrations of respirable phosphoric acid are the same and individuals will be exposed to ambient air phosphoric acid for 24 hours per day. The phosphoric acid exposure estimates also assume an average respiration rate of 20 m^3/day for a 70 kg adult and the calculation of the human dose assumes 100% absorption of the respirable phosphoric acid.

Table 6 - Lifetime average estimated human exposure to phosphoric acid in ambient air

Scenario	Average ambient air concentration (H_3PO_4 ng/m^3)	Human exposure (ng/day)	Human dose [a] (ng/kg bw/day)
Baseline	negligible	negligible	negligible
Scenario 1: Increase due to combustion of the alkyl phosphate additive	100	2000	28.6
Scenario 2 : Increase due to combustion of the alkyl phosphate additive	40	800	11.4

[a] Dose for a 70 kg person.

People living in rural areas would be expected to have lower exposure than people living in cities. Thus, given that the estimated total phosphoric acid concentration in Sydney due to the use of the alkyl phosphate-based LRP is 100 ng/m^3 (Table 4), exposures of Australians living in rural and remote regions are expected to be even lower.

People living in areas that have industries emitting phosphoric acid could be expected to have the highest levels of exposure. No Australian ambient air phosphoric acid concentration data are available and no personal monitoring studies have been completed in Australia.

It should be noted that ambient air concentrations may not always reflect the actual exposure of individuals living in a given area. This is because typical human activity patterns include time spent in microenvironments with higher or lower concentrations of a pollutant and for which there is generally no monitoring data. Hence, a measurement of personal exposure to a compound is preferable to ambient air data. Furthermore, a measurement of personal exposure should be representative of the population of interest throughout the time period of interest. The above worst case estimate of public inhalation exposure to phosphoric acid as a result of the combustion of the alkyl phosphate additive cannot be refined and can be considered only as a general exposure level.

Exposure to phosphate via food, water and soil

It is conceivable that phosphorus levels in foodstuff may be increased as a result of environmental contamination with the combustion products of the alkyl phosphate additive. There are no Australian studies on the possible contribution of the alkyl phosphate additive combustion product to phosphorus levels in food.

It is considered that the contribution of the combustion of the alkyl phosphate additive to phosphorus intake from foodstuff in Australia is likely to be very low. This is because of the expected low atmospheric (Table 4), water (Section 9.4.2), and soil (Section 14.1.1) levels of phosphoric acid or phosphate due to the combustion of the alkyl phosphate additive in LRP, especially in rural areas, compared with the recommended dietary intake of phosphate (Section 11).

Other possible sources of phosphate exposure

Another possible source of phosphate exposure is soil ingestion. Phosphate exposure as a result of soil ingestion is not expected to increase significantly from current exposure levels (Section 14.1.1). This is because of the estimated low fallout value of phosphoric acid as a result of the combustion of the alkyl phosphate additive in LRP.

10. Animal and Human Health Effects of the Alkyl Phosphate Additive

The aim of this section is to describe the health effects of the alkyl phosphate additive. The scope of this section is limited to describing the health effects of the proprietary product DMA-4 in animals (mammals and avian). This is because of the lack of data for the alkyl phosphate additive itself and the equivalence of DMA-4 to Valvemaster™ Concentrate (Section 4) (Octel, 1999b).

In the case of end-points studied in animals (mainly irritation and acute toxicity studies), this assessment report is based on a limited number of individual animal studies. Some of the animal studies do not comply with Good Laboratory Practices (GLP) or international standards such as the Organisation for Economic Co-operation and Development (OECD) Test Guidelines. In view of the small number of studies, all existing studies are included in this section irrespective of their compliance with formal quality criteria. Studies that provide insufficient scientific detail to permit a critical appraisal of their conclusions are acknowledged.

There are no published case reports, epidemiology or other studies addressing the human health effects of the alkyl phosphate additive or its proprietary products Valvemaster™ Concentrate (including DMA-4) and Valvemaster™ VM11. No subchronic, chronic, reproductive or developmental toxicity or genotoxicity studies were available for DMA-4.

No work-related injuries or health conditions due to exposure to alkyl phosphate additive were reported in industry submissions.

10.1 Acute toxicity

The acute oral toxicity study of DMA-4 in male rats is based on the OECD 401 "Acute Oral Toxicity" Guideline (Haskell Laboratory, 1964). A single animal was dosed at 670, 2250, 3400, 5000, 7500, 11 000, 17 000 or 25 000 mg/kg bw by intragastric intubation, with single doses undiluted, or as a solution in peanut oil. Survival was recorded over a 14-day timeframe. Generalized clinical observations of weight loss, digging motion, salivation after dosing, pallor and diarrhoea were observed at doses of 5000 mg/kg bw and above. Animals died at the top two doses (17 000 and 25 000 mg/kg bw) at Day 2. Under the conditions of the study, the oral acute lethal dose (ALD) for DMA-4 in male rats was 17 000 mg/kg bw.

In an acute oral study in male rats of DMA-4 in rats, a single oral dose (1 animal per dose) of 710, 2300, 3400, 5100, 7500 or 11 000 mg/kg bw was administered in corn oil by intragastric intubation (Haskell Laboratory, 1986). No deaths occurred over the 14-day study period. The clinical signs of toxicity most commonly observed included diarrhoea, discharge from the nose and mouth, wet perineum and weight losses (6-12% of body weight) up to three days after dosing. Ruffled fur was observed in rats dosed at 7500 and 11 000 mg/kg bw. Weakness, yellow stained perineum, ocular discharge and hunched posture were also observed in the rat dosed at 11 000 mg/kg bw. Under the conditions of the study, the oral ALD was greater than 11 000 mg/kg bw.

In the acute dermal lethality study of DMA-4 in rabbits, a single dose was applied to clipped, intact skin of male New Zealand White (Haskell Laboratory, 1987). The treatment dose included one animal treated at 2250 mg/kg bw and 6 animals treated at 5000 mg/kg bw; under occlusion for 24 hours. The rabbits were observed for 14 days post-application and no animals died. No details are given of the method used to describe the symptoms of skin irritation nor observations given for the individual animals. While the test parameters do not comply with a standard testing guideline, they are considered to be scientifically reliable with regard to the measured endpoint, i.e. mortality.

Approximately 5-7% weight loss occurred within 24-hours in all the rabbits dosed at 5000 mg/kg bw, with weight recovery by the end of the study. Superficial necrosis and dry cracked skin were observed at the termination of the study in 5 out of the 6 animals dosed at 5000 mg/kg bw. The single rabbit treated at 2250 mg/kg bw displayed only mild erythema and oedema at Day 3 and no skin irritation was observed by the sixth day. Under the conditions of the study, the ALD by skin absorption for DMA-4 was greater than 5000 mg/kg bw in male White New Zealand Rabbits.

The results of a first phase study to investigate delayed neurotoxic response in mature female white leghorn chickens (n = 4), reports a LD50 for DMA-4 to be in excess of 5000 mg/kg bw following a single oral dose of 5000 mg/kg bw administered to each bird. Further details of this study are given in Section 10.4. Clinical signs of toxicity noted in the four birds included slight lethargy and anorexia at Day 2 and Day 3, respectively, which resolved at Day 3. There were no deaths associated with the doses given (Du Pont De Nemours & Co., 1986) .

10.2 Irritation and corrosivity

Eye irritation

The effect of direct contact of DMA-4 with the eye was examined in New Zealand white rabbits using the Draize scoring method. Study methodology was similar to OECD Guideline for Acute Eye Irritation/Corrosion 405 (Haskell Laboratory for Toxicilogy and Industrial Medicine, 1986a). The study involved two animals and the instillation of 0.01 mL of DMA-4 (rather than 0.1 mL as recommended by OECD Guideline for Acute Eye Irritation/Corrosion 405) into the lower conjunctival sac of the right eye, with the left eye used as a control. The treatment and control eyes of one animal remained unwashed while the eyes of the other animals were rinsed for one minute with water approximately 20 seconds after the administration of the test material. Approximately one and four hours and one, two and three days after treatment, the rabbits were examined for evidence of eye irritation. No cornea/iris irritation was observed in both washed and unwashed eyes in both animals. Slight redness/chemosis of the conjuctiva (Score 1) was observed only at one hour in both washed and unwashed eyes for both animals after exposure to DMA-4. No reactions of the cornea or iris were observed in either animal.

Skin irritation

Skin irritation in 6 female rabbits was assessed using a Draize scale (Draize et. al, 1944) for scoring primary skin irritation in a modified OECD Guideline 404 for assessing Acute Dermal Irritation/Corrosion (Haskell Laboratory for Toxicology and Industrial Medicine, 1986b). The dermal application of 0.5 mL of DMA-4 was administered at each test site, 4 test sites per animal for 24 hours under an occlusive

(rubber) wrap Test sites were evaluated at 25, 48 and 72 hours after application. The test material caused moderate to severe inflammation of the skin under the conditions of the study (Table 7). Test results show delayed oedema following the initial observed erythema. The peak oedema time point may be beyond the 72-hour end-point used in the study.

Table 7 - Results of a skin irritation study in rabbits

Lesion	Mean Score	Maximum Value
Erythema/Eschar (25 hr)	2	3
Oedema (25 hr)	0.33	2
Erythema/Eschar (48 hr)	3	3
Oedema (48 hr)	2.7	4
Erythema/Eschar (72 hr)	3	3
Oedema (72 hr)	2.8	4

In an additional study of skin irritation, an unknown volume of 0.5, 1, 5, 10 and 25% of DMA-4 was applied to intact and abraded skin for 24 hours to an unspecified number of test sites per animal in 9 male guinea pigs (described non-specifically as young and old) (Haskell Laboratory for Toxicology and Industrial Medicine, 1964). The study method is non-standard and involved the application of the test material in a 1:1 solution of acetone:dioxan with 13% guinea pig fat. No grading scale is described other than nil, mild, or strong erythema. No information is given as to whether or not an occlusive dressing was used. The study gives only a limited narrative description of the observed erythema (Table 8).

Table 8 - Results of a skin irritation study in guinea pigs

DMA-4 (%)	Observation
25	Strong erythema on intact skin
10	Strong erythema on intact skin; moderate to mild erythema on abraded skin
5	Mild erythema on intact and abraded skin (young guinea pig); strong erythema and strong to mild erythema on intact and abraded skin, respectively, in older guinea pigs.
1	Occasional mild erythema on intact skin
0.5	No erythema

Under the study conditions, the application of DMA-4 was reported to be irritating to guinea pig skin in concentrations as low as 10%. No information is available from the study of the resolution time for the induced erythema. The study authors noted that the test material appears to be more irritating to older than younger guinea pigs. No data or details are available or given for individual animals. There is insufficient documentation and experimental detail to reliably accept the adequacy of the results and conclusions.

10.3 Sensitisation

A single study is reported on the potential skin sensitisation of DMA-4 using an unknown method in 9 male albino guinea pigs (Haskell Laboratory for Toxicology and Industrial Medicine, 1964). The study was undertaken concurrently with the primary irritation skin test in guinea pigs that is reported in Section 10.2. A 10% solution of DMA-4 was applied topically to abraded skin in guinea pig fat 9 times over a three week period. No further experimental details were reported.

None of the 9 treated animals are reported to show allergic skin sensitisation. The data generated, however, is not in accordance with an acceptable methodology and there is insufficient experimental detail and documentation for assessment.

10.4 Neurotoxicity

A 21-day neurotoxicity study of H-16,407 (also described as DMA-4 in the study) in mature female white leghorn chickens (19-20 months) is reported (Du Pont De Nemours & Co., 1986). While not stated, the study appears similar to the OECD Delayed Neurotoxicity of Organophosphorous Substances Following Acute Exposure Guideline 418 and involved a first phase (range finding) study to determine an LD50 value and a subsequent second phase 21-day neurotoxicity study. The objective of the study was to establish whether or not the compound produces a delayed neurotoxic response.

The first phase (range finding study) involved four birds orally dosed at 5000 mg/kg bw administered as a single dose by gavage. All birds were observed on a daily basis for seven days post-dosing and sacrificed at the end of the study period. No mortality nor signs of delayed neuropathy were observed. Clinical signs of toxicity noted included greasy cage droppings at Day 1 and Day 2; slight lethargy Day 2 and anorexia at Day 3. All these observed clinical signs resolved after Day 3. The results of the first phase showed the acute oral LD50 for H-16,407 to be in excess of 5000 mg/kg bw.

In the second phase of the investigation, 40 birds were selected for the 21-day neurotoxicity study. The birds were divided into a treatment control group of 10 birds (receiving a single oral dose of corn oil), a positive neurotoxicity control group of 10 birds (receiving a single oral dose of tri-o-tolyl phosphate (TOTP) 500 mg/kg bw at 0 hour on test Day 1), and a H-16,407 treatment group of 20 birds (receiving a single oral dose of H-16,407 of 5000mg/kgbw at 0 hour on test Day 1 following an approximate 19 hour fast with water permitted). The study contains no details as to whether or not the positive or treatment control animals were also fasted. The treatment control birds received corn oil in an amount equal to the largest dosage of test material administered in the H-16,407 treatment group, i.e. 10.7 mL. The selected dosage level for the H-16,407 treatment group in the 21-day neurotoxicity was derived from the first phase of the study, i.e. 5000 mg/kg bw. All animals were sacrificed at the end of the study on Day 21. Gross pathology investigations including a complete necroscopic and microscopic examination of the brain, spinal cord and sciatic nerves, in addition to a microscopic examination for neoplasms, were undertaken on the carcasses at the end of the study period.

The birds were evaluated daily for signs of neurotoxicity and graded with a score of 0 to 5 according to criteria as follows: no neurological signs (Grade 0); generalised weakness with or without slight intermittent ataxia (Grade 1); slight continuous ataxia (Grade 2); moderate to severe ataxia (Grade 3); bird unable to stand, sits on haunches

(Grade 4); bird unable to stand, paralysis of legs and wings (Grade 5). Individual body weights were recorded at 0 hour Day 1 of the study and on test days Day 3, 6, 9, 12, 15, 18 and 21. Food consumption was determined at each weighing interval.

No clinical signs of delayed neurotoxicity or death were noted in either the treatment control or H-16,407 treatment group over the 21-day study period. By comparison, the positive control birds exhibited behavioural signs of neurotoxicity by Day 9 post-treatment; with all animals showing signs of delayed neurotoxicity by Day 12 and progressively worsening signs of neurotoxicity by Day 16. All positive control birds were sacrificed at Day 16 post-treatment due to severe neurological signs.

Signs of lethargy, anorexia and abnormal cage droppings were present following dosing with H-16,407, with recovery by Day 8. Greasy droppings were noted in the H-16,407 group within 21 hours post-dosing and were still evident 24 hours later along with concomitant lethargy and anorexia (Day 2). A statistically significant reduction in body weight was noted at Day 3 (9.7% reduction; 99% CI) and Day 5 (8.2% reduction; 95% CI) that resolved by Day 9. All birds (including cage droppings) appeared normal on Day 7-8 of the study.

No visible lesions or gross pathology were revealed in the 10 treatment control birds sacrificed on Day 21 nor in 17 of the 20 birds treated with H-16,407. Of the remaining three birds in the H-16,407 treatment group, one bird revealed a slightly emaciated carcass, another displayed ruptured egg yolks in the oviduct and the third bird revealed reabsorbed egg yolk material in the abdominal cavity in addition to a slightly emaciated carcass. In the 10 positive control birds, no abnormal tissue alterations were noted in seven birds. One displayed an enlarged caecum (or distension due to gas), another emaciation and one bird revealed reabsorbed eggs in the visceral cavity.

The results of the above studies indicate H-16,407 to be of low toxicity in chickens via the oral route; with no signs of delayed neurotoxicity noted under the conditions of the study. Furthermore, gross pathological examinations of the sciatic nerves, brain and vertebral column of all birds sacrificed on Day 21 revealed no abnormal tissue changes in the H-16,407 treatment group.

11. Pharmacokinetics and Toxicity of Phosphoric Acid

11.1 Introduction

The toxicology of phosphoric acid (orthophosphoric acid) is addressed in this section because it is postulated to be the significant combustion by-product of the alkyl phosphate additive in LRP (Section 8). A brief description of the physico-chemical properties of phosphoric acid is given in Appendix 2.

The mammalian toxicity data and information presented in this section builds on the review of phosphoric acid undertaken in 1993 by the Working Group on the Assessment of Toxic Substances (WATCH Committee) of the UK Health and Safety Commission's Advisory Committee on Toxic Substances (Payne et al., 1993) and the U.S. EPA's Integrated Risk Information System (IRIS) summary of phosphoric acid that was last updated on 8 January 2003 (IRIS, 2003). Any additional relevant data identified in recent literature searches are also included.

The toxicity studies in this section do not represent a review of the complete toxicology of phosphoric acid. The main focus is chronic inhalation and acute exposure to an aqueous mist of phosphoric acid. Such exposures are likely to be the most relevant scenarios as a result of the combustion of the alkyl phosphate additive in LRP.

The reported inhalation toxicity of phosphoric acid characteristically relate to the effects of phosphorus pentoxide (P_2O_5), which is the anhydride form of phosphoric acid. It is generated from the combustion of phosphorus under oxygen rich conditions. This is because there is little or no data generated in humans or animals on the effect of the inhalation of phosphoric acid which would allow a more accurate assessment of the threshold level of irritation or evaluation of the effects of prolonged low-level inhalation exposure. The treatment doses used in these inhalation studies may be expressed as phosphorus pentoxide, phosphorus or phosphoric acid equivalents.

Phosphorus pentoxide is relevant because it hydrolyses with moisture in the air and lungs to produce phosphoric acid. Phosphorus pentoxide is a powerful dehydrating agent that, when combined with moisture in the atmosphere or in the respiratory tract, produces phosphoric acid in an exothermic reaction. This reaction generates heat and is likely to desiccate tissues with which phosphorus pentoxide comes into it contact, thereby causing more tissue damage than pre-formed phosphoric acid. The application of the results of inhalation toxicity studies on phosphorus pentoxide to an assessment of the hazards of phosphoric acid will possibly overestimate the inhalation hazard of phosphoric acid.

11.2 The phosphate body load

Most of the phosphorus in the human body exists as phosphates or esters, with phosphate playing an important role in the maintenance of the body's acid-base balance (Haslett et al., 1999).

Phosphoric acid dissociates in water to hydrogen and phosphate ions (Appendix 2). Phosphoric acid, in the form of the phosphate anion, is an essential component (not

synthesised by the body) of the normal body and skeleton of practically all life forms and is a natural component of the diet (Fauci et al., 1998). The phosphate body load/content is often expressed in terms of elemental phosphorus. Of the average 700 g of phosphorus in the human body, 85% is in the skeleton (Fauci et al., 1998).

Dietary phosphate is found in highest concentrations (0.1-0.5% or more, in terms of phosphorus) in such foods as milk, cheese, nuts, fish, meat, poultry, eggs (yolk) and certain cereals (WHO, 1965). Phosphoric acid is also used in a range of foods as additives (ANZFA, 2001). For example, phosphoric acid is added as an acidifier in soft drinks, as a nutrient in the preparation of yeast and in the refining of cane sugar, oil and fats (Commission of the European Communities, 1992). Dietary phosphorus occurs in both inorganic and organic forms such as phospholipids, phospho-sugars and phospho-proteins (Lee et al., 1981). Most organic phosphates are hydrolysed in the gut to release the inorganic phosphate that is absorbed, although some phospholipids may be absorbed in the organic form (Lee et al., 1981).

The daily human intake of phosphorus is estimated to range between 1 and 2 g (Haslett et al., 1999, WHO, 1965). The World Health Organization (WHO) states the value of 70 mg/kg bw as representing the maximum tolerable daily intake (MTDI) of phosphates expressed as phosphorus when the diet contains adequate calcium (WHO, 1982). A daily phosphorus intake of 70 mg/kg bw equates to 4.2 g of phosphorus or 12.9 g of the phosphate anion (PO_4^{3-}) for a 60 kg adult.

The US National Academies, Institute of Medicine has published Recommended Dietary Allowance (RDA) values for phosphorus ranging from from 100 mg for infants (0.0 to 0.5 years old), ~500 mg for children (1 to 10 years of age) and up to 1250 mg for adolescents (11 to 18 years of age) and adults (National Academies, 2003). A maximum level of daily phosphorus intake of 4000 mg is specified by the US National Academies, Institute of Medicine as a level that is likely to pose no risk in adults of adverse effects (National Academies, 2003).

11.3 Toxicokinetics of phosphoric acid

There is no information on the toxicokinetics of phosphoric acid *per se* . However, the mechanisms controlling the acid-base homeostasis in the body (including the interrelationships of phosphate and calcium) and the potential of exogenous phosphorus and hence, phosphate loads, to disrupt the body's acid-base balance and cause hypocalcaemia and hyperphosphatemia, is well understood. These mechanisms are not described here because they are well explained in publications on phosphorus and phosphate metabolism and distribution (Lee et al., 1981; Fauci et al., 1998; Haslett et al., 1999).

In humans, hyperphosphataemia is defined in adults as an elevation of serum phosphorus above 1.67 mmol/L (5 mg/dL) with decreased renal excretion of phosphorus the most common cause (Fauci et al., 1998). High phosphate loads may lead to the deposition of calcium phosphate complexes in soft tissue and a range of physiological disturbances such as kidney damage, extra-skeletal calcification and bone demineralisation (Lee et al., 1981; Fauci et al., 1998) (Haslett et al., 1999).

In general, when dietary phosphate intake is increased or decreased, the body's homeostatic mechanisms are activated to maintain the phosphate balance in the body; with serum phosphate levels tending to remain within normal ranges (0.8 to 1.4 mmol/L fasting (Haslett et al., 1999).

11.4 Acute phosphoric acid toxicity in animals and humans

11.4.1 Acute toxicity

Humans

The limited human inhalation exposure data on phosphoric acid relates to accidental exposure. Weakness, dry cough, chest pain and shortness of breath/dyspnea were reported as developing 7 to 8 hours after exposure to high concentrations of phosphoric acid in a 37 year old mechanic aboard a ship transporting 4000 tonnes of phosphoric acid. Investigating a leak, the mechanic developed symptoms following exposure to phosphoric acid vapours on three occasions, each for approximately 20 minutes/exposure (Boutoux et al., 1995). The mechanic was a non-smoker with no history of allergies or previous respiratory problems. Symptoms of reactive airways dysfunction were reported in the worker one year after the incident. A colleague similarly exposed to the phosphoric acid vapours was taken to hospital and died in a state of severe respiratory distress. Medical staff initially suspected Legionella to be the cause of the fatality, but blood tests for the organism proved negative.

In a poorly reported Russian study of 15 non-smoking healthy adults aged between 18 and 36 years, "momentary" inhalation of hydro-aerosols of phosphoric acid expressed as 1.2, 5.2 or 8.0 mg P_2O_5/m^3 (equivalent to 1.6, 7.2, or 11.0 mg H_3PO_4/m^3) reportedly produced irritation in 18% of the subjects at exposure level of 5.2 mg P_2O_5/m^3 and 82% at exposure level 8.0 mg P_2O_5/m^3 (Sigova, 1983, as cited in Payne et al., 1993). The lowest concentration reportedly did not provoke irritation in any of the volunteers. A copy of the original reference was unavailable to NICNAS and the reliability of the data is uncertain.

Animal

Oral and dermal

Phosphoric acid has low acute oral and dermal toxicity as demonstrated by an oral rat LD50 of 1530 mg/kg bw and dermal rabbit LD50 of 2740 mg/kg, respectively (Payne et. al, 1993).

A study of the administration to groups of 5 Sprague-Dawley rats of commercial preparations of 85%, 80% and 75% phosphoric acid by oral gavage, report oral LD50 values of 3500, 4200 and 4400 mg of test material/kg bw, respectively (Randall & Robinson, 1990).

Inhalation

A rat LC50 (1-hr) value of > 850 mg/m^3 is reported for phosphoric acid (BIOFAX Industrial Bio-Test Laboratories, as cited in RTECS, 1998) but the individual data is unavailable to NICNAS and the reliability of the study cannot be validated. The WATCH Committee report notes the availability of a phosphoric acid mouse inhalation LD50 of 25.5 mg/m^3 indicates serious doubts about the validity of the study (Payne et al., 1993).

A range of LC50 (1-hr) values for phosphoric acid (and phosphorus equivalents) in male rabbits, mice and guinea pigs was comprehensively reported by Ballantyne in 1998 (Ballantyne, 1998). This followed an earlier short communication on the study

that was described as an acute inhalation toxicity study of phosphorus pentoxide smoke (Ballantyne, 1981).

The LC50 (1-hr) values, expressed as phosphorus and phosphoric acid equivalents, relate to inhalation toxicity following the complete oxidation of unformulated red phosphorus and exposure to a range of phosphorus concentrations from 35 to 2000 mg/m^3 (equivalent to 111 to 6327 mg/m^3 phosphoric acid) (Ballantyne, 1998). The use of unformulated red phosphorus under complete oxidation conditions was cited by the investigator in order to result in phosphoric acid as the principal combustion product and to avoid the presence of formulation by-products in the generated smoke.

Expressed as phosphorus, the LC50 (1-hr) values were 1689 mg/m^3 (rabbit), 1217 mg/m^3 (rat), 271 mg/m^3 (mouse), and 61 mg/m^3 (guinea pig); expressed by Ballantyne as phosphoric acid equivalents, the values are 5337 (rabbit), 3846 (rat), 856 (mouse), and 193 (guinea pig) mg/m^3, respectively (Ballantyne, 1981; Ballantyne, 1998). Most of the animals that died did so during exposure although a small proportion survived a few hours or days post-exposure. Concentrations of phosphorus pentoxide not associated with respiratory tract pathology in 14-day survivors were 450 mg/m^3 (rat and rabbit), 111 mg/m^3 (mouse) and 36 mg/m^3 (guinea pig). This is equivalent to phosphoric acid concentrations of 621 (rat and rabbit), 153 (mouse) and 50 (guinea pig) mg/m^3.

11.4.2 Irritation and corrosivity

Human

Gastrointestinal

Widespread corrosion of the human gastrointestinal tract is reported in a fatal case report of oral ingestion of phosphoric acid (unspecified quantity) (Hawkins et al., 1980).

Eye

Phosphoric acid in concentrated solution is reported as corrosive to the human eye and splashes have been reported to result in permanent injury in severe cases (Proctor & Hughes, 1978, as cited by Payne et al., 1993). A single drop into the human eye of approximately 1.5% phosphoric acid buffered at pH 2.5 (volume unspecified) is reported as causing a moderate brief stinging but no injury, with the same solution at pH 3.4 reportedly having no discomfort (Grant, 1974).

Dermal

Stinging sensations following the facial application to humans of phosphoric acid during profuse sweating is reported (Frosch & Klingman, 1977). The study reports an immediate yet transient stinging response within the first 10 seconds of exposure to 5% phosphoric acid; followed by a severe stinging sensation developing after 2-8 minutes. The severe delayed response was also experienced on the application of 3.3% (pH 1.9) phosphoric acid solutions, whereas 1% (pH 2.1) phosphoric acid solution produced only a slight response. The study (on several acids) did not find a correlation between stinging and irritancy.

Animal

Eye

Based on an unknown scoring methodology in a rabbit test model, severe eye irritation is reported with phosphoric acid at a dosage level of 119 mg (no further details are available) (RTECS, 1998).

In a testing protocol equivalent to OECD Guideline 405: Acute Eye Irritation/Corrosion, mild conjunctivitis, chemosis and opacity of the cornea is reported following the instillation of a 100 µL of a 17% solution (pH not given) into rabbit eye (n = 6) (Jacobs, 1992). In the same study, a reduction in the mean Draize scores was observed after the instillation of a 100 µL of a 10% solution (pH not given). Mild conjunctiviti was the only adverse effect observed at the study end-point at 96 hours.

Irrigation of a rabbit eye for 5 minutes with a solution of phosphoric acid at pH 3.8 (unspecified concentration) reportedly caused slight transient redness and swelling, with the eye normal by the next day (Grant, 1974).

Instillation of an unspecified volume of 4% phosphoric acid (pH not given) into the rabbit eye produced significant conjunctival swelling one hour later, as measured by a 50% increase in moisture content of the conjunctiva in comparison with the untreated eye (Larson et al., 1956).

Dermal

Based on an unknown scoring methodology in a rabbit test model, severe skin irritation is reported with phosphoric acid at a dosage level of 595 mg/24-hour (no further details are available) (Payne et al., 1993).

A range of phosphoric acid solutions is reported to be skin irritants in animals; with the length of exposure, degree of occlusion and concentration of the acid significant factors. For example, phosphoric acid solutions (0.5 mL; 75- 85% applied to an unspecified number of animals) are reported as corrosive in primary skin irritation tests in rabbits following 24-hour exposure under semi-occlusion (Randall & Robinson, 1990). In the same study, 75% and 80% phosphoric acid solutions were non-corrosive to rabbit skin after 4-hour exposure. In a modified test protocol involving only one rabbit, 0.5 mL of a 75% phosphoric acid solution applied for 4 hours under occlusion was not found to be irritating (Weiner et al., 1990). Phosphoric acid concentrations of 17.5% and above (pH 0.6-0.2) under occlusion for 4 hours, however, are reported to be corrosive in rabbit skin as evidenced by the formation of scar tissue (Loden et al., 1985) as cited by (Payne et al., 1993). One of three rabbits in the study reportedly developed severe erythema of the skin with mild to moderate swelling 48-72-hours after exposure to 2.5% phosphoric acid at pH 2.1.

A 5% phosphoric acid solution (pH < 2) is reported as moderately to severely corrosive in rat and mice skin irritation tests following the application of 1mL/kg (50 mg/kg) for 7 days without occlusion (Sekizawa et al., 1994).

11.4.3 Repeat exposure

Oral

In a poorly described study, rats were fed a diet containing 0, 4000 or 7500 ppm phosphoric acid from weaning up to 15 months of age. Offspring were similarly fed for

up to 6 months of age (Bonting & Jansen, 1956, as cited by Payne et al., 1993). No adverse effects on body weight, blood clinical chemistry or substance-related macroscopic or microscopic abnormalitites of the liver, spleen, adrenals, testicles, skeletal muscles or femur were observed.

It is suggested by the World Health Organization (WHO) that an important and sensitive criterion of orally ingested phosphate overload is the appearance of metastatic calcification in the soft tissues, especially the kidney, i.e. nephrocalcinosis, as well as in the stomach and aorta (Joint FAO/WHO Expert Committee on Food Additives, 1974). Phosphoric acid has been shown to cause nephrocalcinosis in rats when administered at relatively high concentrations in the diet (FAO, 1970; USEPA, 1990) (Joint FAO/WHO Expert Committee on Food Additives, 1974). The WHO has determined that the lowest level that produces nephrocalcinosis in the rat is 1% phosphorus in the diet (Joint FAO/WHO Expert Committe on Food Additives, 1974). This figure is the basis for an extrapolation by the WHO to a dose of 6.6 g of phosphorus daily (based on a daily food intake of 2800 calories) as an estimate of the lowest possible level that may cause nephrocalcinosis in humans (Joint FAO/WHO Expert Committe on Food Additives, 1974). This is equivalent to 20.2 g of the phosphate anion (PO_4^{3-}).

Inhalation

Phosphoric acid

Human

High prevalence of chronic bronchitis (45.7%) and a significant incidence of impaired respiratory performance has been reported for workers at one plant producing phosphoric acid (Fabbri et al., 1977). Concomittant exposure to volatile fluorides (hydrofluoric acid, hexafluorosilicic acid and tetrafluoride) was considered to be very significant, however, the direct contribution of phosphoric acid to the observed effects is not known.

In another study, the health of workers was investigated in the phosphoric acid production division (type of process unspecified) of a phosphate fertiliser plant (Renke et al., 1987). The incidence of bronchial and peribronchial changes in the phosphoric acid division was 13% compared to no greater than 11.8% for non-exposed office staff at the same factory.

No conclusions can be drawn about the toxic effects of phosphoric acid from these occupational studies because exposure to emissions other than phosphoric acid cannot be discounted. In addition, in neither study was the exposure concentration or duration reported.

Animal

Studies of the effects of chronic inhalation of phosphoric acid in animals (using pre-prepared phosphoric acid treatments) could not be found in the literature.

Phosphorus Pentoxide

Human

An occupational study is reported on lung function in a cohort of 131 workers involved in refining phosphorus rock for 3 to 46 years to obtain elementary phosphorus (Dutton et al., 1993).

Typical exposures included phosphoric-acid, phosphorus oxides (including phosphorus pentoxide), fluorides, and coal-tar-pitch volatiles. While protective equipment was available to the workers, observation revealed they rarely made use of it. The maximum concentrations of phosphorus-pentoxide, fluorides, and coal-tar-pitch volatiles in the refinery air were 2.23, 4.21 and 1.04 mg/m^3, respectively. Fifty-five subjects were current smokers, 39 were former smoker and 37 were nonsmokers. The mean duration of exposure was 11.4 years for all workers, 11.0 years for smokers, 15.1 years for former smokers and 7.8 years for nonsmokers.

Pulmonary function tests (forced vital capacity, forced expiratory volume in one second and forced expiratory flow) were conducted annually over an 8-year period in all workers to assess any effects of occupational exposure. These data were analysed longitudinally over 3 to 7 years and cross-sectionally. Neither analysis revealed any significant effect as measured by spirometry measures of pulmonary function after adjusting for age and smoking. The authors concluded that occupational exposures in the refinery did not contribute to annual changes in pulmonary function in the workers as measured by spirometry measures. The observed decline in pulmonary function in smoking workers was attributed to the effects of smoking.

Animal

In a poorly reported Russian study (a copy of which could not be sourced), rats were exposed for an unspecified time, described as chronic, to a hydro-aerosol of phosphoric acid expressed as phosphorus pentoxide 10.6 mg/m^3 (which is equivalent of 14.6 mg/m^3 phosphoric acid) (Sigova, 1983, as cited by Commission of the European Communities, 1992). At post mortem, increased relative weights of the lungs, kidneys and spleen were reported. Inflammation of the bronchi, cell damage in the convoluted tubules and enlargement of the glomeruli in the kidney, an increase in the amount of lymphatic tissue in the spleen and liver damage is cited. A "threshold value" for these effects was determined to be 2.5 mg/m^3 for phosphorus pentoxide (which is equivalent to 3.45 mg/m^3 phosphoric acid). Nevertheless, it is unclear from the report if this dose represents the lowest dose at at which effects were seen or the highest dose at which effects were not seen (Commission of the European Communities, 1992).

A chronic inhalation toxicity study reports pulmonary toxicity in rats, mice and guinea pigs following exposure to phosphorus smoke generated by the combustion of 95% amorphous oiled red phosphorus and 5% polyvinyl butyral BL18 at phosphorus concentrations of 0, 16 or 128 mg/m^3 for 1-hour/day, 5 days/week for 180 to 200 days (Marrs et al., 1989). While the smoke is reported to contain mostly phosphoric acid (derived from phosphorus pentoxide), phosphine (which is produced from phosphorus trioxide), cyclotetraphosphoric and other polyphosphoric acids are proposed as additional components of the smoke.

Marrs et al reports marked interspecies differences in tolerance to the smoke (Marrs et al., 1989). Rats were more tolerant, guinea pigs least and mice intermediate. There was a high death rate in all groups (including controls). With the exception of the high

dose guinea pig group, decedents mostly failed to show statistically significant pathological changes either in the respiratory tract or elsewhere. Pulmonary congestion (accompanied in some cases by haemorrhage and collapse) was observed, however, in the high dose guinea pig group and in all the animals that died during or just after the first exposure. The investigators attributed these observations to exposure to the smoke.

A study in male Sprague-Dawley rats (sample sizes unspecified) investigated the effects of exposure to smoke generated by burning 95% of red phosphorus 5% butyl rubber (RP/BR). Exposure was to a single inhalation of 1000 mg/m^3 for 3.5 hours or subchronic smoke concentrations ranging from 300 to 1200 mg/m^3 for 2.25 h/day for 4 consecutive days and 4 and 13 weeks (Aranyi et al., 1988a; Aranyi et al., 1988b). Total phosphoric acids levels were measured in the smoke and reported to contribute to around 70% of the smoke.

The respiratory tract was identified as the target organ. Mild or moderate-to-severe terminal bronchiolar fibrosis was reported in all rats after 4- and 13-week exposures to ≥ 750 mg/m^3 of RP/BR. Of those animals exposed to 300 mg/m^3 for 13 weeks, 13% had terminal bronchiolar fibrosis. The severity of the lesions increased with the severity of the exposure conditions. The investigators state that the incidence of fibrosis in those rats sacrificed 8 weeks after the 13 week exposure to 750 or 1200 mg/m^3 of the smoke, suggested that the severity of the lesion did not increase following the cessation of exposure nor resolve with time. Decreased pulmonary bactericidal activity was depressed after acute and 13-week exposures but not after 4-week exposures.

The paper states that an additional subchronic study has identified the no-effect level of the smoke for fibrosis of 50 mg/m^3; however, no details are provided. This result is also cited by the US EPA in a description of two parallel 13-week inhalation studies but full copies of the studies were not available and could not be reviewed for this assessment report.

The two parallel 13-week inhalation studies reported by the US EPA involve exposure of Sprague-Dawley rats for 2.25 hours/day on 4 consecutive days/week to either filtered air (controls) or an aerosol of combustion products from burning 95% red phosphorus and 5% butyl rubber (Aranyi et al., 1988a; Aranyi et al., 1988b) as cited by (US EPA).

In the first study, male Sprague-Dawley rats were exposed to filtered air or 300, 750, or 1200 mg/m^3 of combustion products (Aranyi et al., 1988a; Aranyi et al., 1988b). In the second study, male Sprague-Dawley rats (40 per group) were exposed to either filtered air or 50, 180, or 300 mg/m^3 of these same combustion products.

The percentage of the aerosols that were phosphorous acids ranged from 71-79% (apparently based on gravimetric analysis). The duration-adjusted values for the second, lower concentration study were 2.7, 9.6, and 16.7 mg/m^3.

In the first study, the number of rats in the control and high- and mid-exposure groups was 176 and 84 rats were in the group exposed to 300 mg/m^3. All major organs and respiratory tract tissues were examined histologically in a portion of the animals (n = 12) from each exposure group. Neurobehavioural studies also were performed in the first of the study, although details are not reported by the US EPA.

The focus of the second, lower concentration study was the respiratory tract. Tissues examined in this study included the turbinates (two sections), trachea and five lobes of

the lung from 20 animals in each exposure group and controls. Concentration-related decreases in pulmonary bactericidal activity were observed for all exposure groups only in the first study.

Agent-related mortality was observed among the animals exposed to the two highest concentrations: 19/176 at 1200 mg/m^3 and 1/176 at 750 mg/m^3. Decreases in body weight gain also were observed in these two groups. The number of deaths in the controls is not reported by the US EPA. No deaths were noted in the second study.

The US EPA states that both studies indicated the target organ to be the respiratory tract, specifically the terminal bronchioles. Pathological examination of a portion of those animals that died revealed extensive involvement of bronchiolar and laryngeal mucosa, the latter probably contributing to death. Terminal bronchiolar fibrosis (minimal to severe) with no or minimal involvement of pulmonary tissues was the only concentration-dependent lesion noted in the respiratory tract of animals surviving repeated exposures. This lesion was present in all animals examined that had been exposed to 750 or 1200 mg/m^3, including those necropsied after an 8-week recovery period, and was judged predominately as moderate and severe. In the second study, this lesion was present with minimal severity in 9/20 animals exposed to 300 mg/m^3, 4/20 animals exposed to 180 mg/m^3 and 0/20 animals exposed to 50 mg/m^3. Based on the histologic lesions in the tracheobronchiolar region for smoke generated from burning 95% red phosphorus and 5% butyl rubber, 180 mg/m^3 is the LOAEL, and 50 mg/m^3 is the NOAEL.

The smokes generated in the above studies are complex and uncertainty has been expressed over the exact chemical identities generated (Payne et al., 1993). For example, temperature and oxygen conditions can affect the type of phosphorus oxide formed during combustion of the phosphorus. Phosphorus pentoxide is produced in an oxygen-rich environment while phosphorus trioxide is produced if the oxygen levels are limited (Corbridge, 1985). In an excess of cold or hot water, the hydrolysis product of phosphorus trioxide is phosphorous acid or a mixture of products (phosphoric acid, phosphine and phosphorus), respectively (Corbridge, 1985). Furthermore, a low concentration of phosphine along with phosphorus pentoxide has been reported in phosphorus smoke aerosols used in some animal inhalation studies (Burton et al., 1982; Marrs et al., 1989).

11.5 Mutagenicity, genotoxicity and carcinogenicity

Phosphoric acid yields negative results in bacterial mutagenicity assays, the only genotoxicity data available to date on phosphoric acid (Demerec et al., 1951; Cipollaro et al., 1986; Al-ani & Al-Lami, 1988). No human data are available on phosphoric acid's potential to cause genotoxicity.

There is no reliable information on the carcinogenic potential of phosphoric acid in animals or humans. Workers in the phosphate industries such as mining and processing or fertiliser production reportedly appear to have a slight increase in lung cancer (Commission of the European Communities, 1992). The evidence for the slight increase in lung cancer in such workers is regarded as weak due to the workers' exposure to a wide variety of chemicals. These chemicals include sulphuric acid, arsenic, chromium and soluble fluorine compounds, some of which are known to be associated with lung cancer.

In a population-based case-control study, an increased risk for renal cancer in relation to occupational exposure to phosphoric acid is reported (Parent et al., 2000). The low precision of the odds ratio estimates (3.4 (1.3-9.2; 95% CI)) and the lack of detail on the industry environment to which the workers were exposed points to further investigations being required to confirm any proposed correlation.

11.6 Reproductive toxicity

There are no adequate reproductive toxicity investigations of phosphoric acid in humans and animals. In a poorly described animal oral toxicity study, the effect of a prolonged intake of phosphoric acid is reported in rats fed diets containing nil or 4000 ppm phosphoric acid since weaning and mated at 32 weeks and again at 43 weeks of age (with the offspring fed the same diet). (Bonting & Jansen, 1956). No adverse effects on reproduction were reported in the study as judged by the weight of the mothers and offspring at birth, the number of live and stillborn young and the survival of young up to weaning.

No information is available on the teratogenicity, neurotoxicity, sensitisation or immunotoxicity of phosphoric acid in mammals.

12. Hazard Classification

12.1 Health hazards

There is no or insufficient toxicity data to enable the alkyl phosphate additive to be classified according to the NOHSC *Approved Criteria for Classifying Hazardous Substances* (the Approved Criteria) (NOHSC, 1999a) or the OECD *Globally Harmonised System of Classification and Labelling of Chemicals* (GHS) (OECD, 2002).

The Approved Criteria are cited in the NOHSC *National Model Regulations for the Control of Workplace Hazardous Substances* (NOHSC, 1994a) and provide the mandatory criteria for determining whether a workplace chemical is hazardous or not. Classifications under the GHS will come into force when it is adopted by the Australian Government and promulgated into Commonwealth legislation.

There are no data for the alkyl phosphate additive itself due to its manufacture and formulation in situ. Toxicology data supplied by the Applicants relate to the alkyl phosphate formulation DMA-4 which is equivalent to Valvemaster™ Concentrate.

Mammalian DMA-4 toxicology data indicate, low acute oral and dermal toxicity and possibly skin irritant effects, most likely related to or confounded by the kerosene content of DMA-4.

Kerosene, which is the hydrocarbon diluent used in the manufacture of the alkyl phosphate formulations (Section 4), is contained in the Valvemaster™ products at and above a concentration of 20% which can be considered a skin irritant according to the NOHSC *List of Designated Hazardous Substances* (NOHSC, 1999b). The alkyl phosphate additive is not listed in the NOHSC *List of Designated Hazardous Substances* (NOHSC, 1999b). Solvent naptha at concentrations greater or equal to 10% is classified according to the NOHSC *List of Designated Hazardous Substances* (NOHSC, 1999b) as Harmful (Xn) and may cause lung damage if swallowed (R65).

12.2 Physico-chemical hazards

There are no physio-chemical data for the alkyl phosphate additive itself due to its manufacture and formulation in situ. The data supplied by the Applicants relate to the alkyl phosphate formulations Valvemaster™ Concentrate and Valvemaster™ VM11 described in Section 5.

Valvemaster™ Concentrate has a low volatility (vapour pressure < 0.1 kPa at 20°C), a boiling point greater than 149°C, and a flash point of 72°C (open cup). The autoignition temperature is ~220°C. Valvemaster™ VM11 has an unknown vapour pressure with a boiling point greater than 149 °C and flash point of 63 °C (open cup). The autoignition temperature is ~220°C.

Regarding the ADG Code (FORS 1998), Valvemaster™ Concentrate and Valvemaster™ VM11 meet the criterion for C1 combustible liquids based on their flash points, which need to range between 60.5 and 150 °C to meet this criterion. Regarding the ADG Code (FORS 1998), the alkyl phosphate aftermarket products do not meet the criteria for classification as a dangerous good on the basis of physicochemical hazards.

13. Effects on Organisms in the Environment

This section provides information on the effects of the alkyl phosphate additive on animals and plants. In addition, information is provided on phosphoric acid (H_3PO_4), the predicted main phosphorus-based combustion product (Section 8) and phosphorus.

Based on the alkyl phosphate additive use pattern and environmental fate, the review of effects has included the potential effects to organisms typically inhabiting terrestrial and aquatic environments.

Issues associated with the use of the alkyl phosphate additive to the environment include:

- ecotoxicological issues associated with accidental spills and leaks to waters and land of alkyl phosphate additive as a concentrate or diluted solution in LRP;
- ecotoxicological issues associated with exhaust emissions of phosphoric acid; and
- contribution to nutrient pollution and eutrophication in aquatic environments due to the emission of a nutrient, i.e. phosphorus, that is often limiting in the environment.

13.1 Terrestrial organisms

13.1.1 Alkyl phosphate additive

No toxicity data were available on the effects of the alkyl phosphate additive on terrestrial plants. Toxicity data for the alkyl phosphate additive product DMA-4 which is synonymous with Valvemaster™ Concentrate in mammals is presented in Section 10.

These DMA-4 animal studies show that the alkyl phosphate additive formulation is not acutely toxic by oral or dermal exposure. The oral ALD in male rats was cited as 17 000 mg/kg bw and ALD by skin absorption greater than 5000 mg/kg bw in male white New Zealand rabbits. While the test guidelines were not standard, the DMA-4 animal toxicity studies indicate the potential for DMA-4 to cause skin and eye irritation.

The results of a 21-day delayed neurotoxicty study using mature white leghorn chickens, indicate DMA-4 to be of low toxicity in chickens via the oral route (LD50 greater than 5000 mg/kg body weight), with no signs of delayed neurotoxicity noted under the conditions of the study.

13.1.2 Phosphoric acid

Animals

Kinetics, metabolism and toxicity of phosphoric acid to mammals is presented in Section 11.

Plants

Environment Canada (1981), as cited by the European Chemicals Bureau (ECB, 2000), describes phytotoxicity test data for concentrated phosphoric acid applied to foliage of peas, beans, beets, rapeseed and weeds. A 15% to 20% solution of phosphoric acid (estimated pH 0.8 to 0.9) applied to foliage-resulted in the death of the exposed plants.

Phosphoric acid, in low concentrations, is a natural component of many fruits and their juices (Furia, 1972).

13.1.3 Phosphorus

Phosphorus is a macronutrient of plants and is also an essential element for microorganisms and animals. It is a structural component of nucleic acids, which are basic building blocks of DNA and RNA. Phosphorus is necessary for cell division and the regulation of all cell processes (Ohio State University, 1996).

In plants, the addition of phosphorus stimulates early root formation, rapid and vigorous growth, hastens maturity and blooming and gives winter hardiness.

Phosphorus deficiency symptoms in terrestrial plants include reddish-purple leaves with the older leaves affected first, short thin leaves, stunted plant growth and defoliation starting at the lower leaves.

Phosphorus toxicity in plants, such as that caused by the addition of excessive amounts of fertilizer to crops, may result in zinc or iron deficiency (Rosen and Eliason, 2002) and necrosis and death in extreme cases.

13.2 Aquatic organisms

13.2.1 Alkyl phosphate additive

Acute aquatic toxicity of DMA-4 was tested by Haskell (1986c) using juvenile fathead minnows (*Pimephales promelas*), a warm freshwater fish, under static, unaerated test conditions. DMA-4 contains the alkyl phosphate compound at similar concentrations to Valvemaster™ Concentrate. Fish (~2.1 cm length, ~0.085 g weight) were laboratory bred and acclimated for 141 days prior to testing. Nominal concentrations tested were 1000, 750, 562.5, 422, 316, 237, 178, 133, 100 and 0 mg/L. Fish were not fed 48 hours prior to, or during, the tests. Test solutions were not aerated but dissolved oxygen was monitored. Temperature was maintained between 21.8 and 22.1°C. Day/night simulation was maintained at 16 hours light:8 hours dark. Mortality counts were made each 24 hours during the 96-hour period and pH was monitored. The 96-hour LC50, derived by probit analysis, was 230 mg/L (95% CI of 210 to 270 mg/L).

Based on this study, DMA-4 does not meet the classification criteria for acute aquatic toxicity using the Globally Harmonised System of Classification and Labelling of Chemicals (GHS; OECD, 2002).

13.2.2 Phosphoric acid

This section provides a brief description of toxicity data for phosphoric acid.

Aquatic toxicity data for phosphoric acid has been obtained from an IUCLID dataset of the European Chemicals Bureau (ECB, 2000). Environment Australia has not reviewed all of the publications referenced (see citation for specific reference sources).

Although the pH of phosphoric acid is approximately pH 1 to 1.5 (ECB, 2000), it is unlikely that pH changes to water bodies would result from the emission of phosphoric acid from the combustion of LRP containing Valvemaster™. Nevertheless, following is a brief account of the potential adverse effects of low pH on aquatic organisms.

Low pH can cause direct adverse effects on fish and aquatic insects, and pH changes (particularly reduced pH) can cause the toxicity of several pollutants (e.g. ammonia, cyanide, aluminium) to significantly increase (ANZECC and ARMCANZ, 2000).

Following is a summary of aquatic toxicity data for aquatic animals exposed to concentrated phosphoric acid. No toxicity data were available for aquatic plants.

Table 9 - Summary of aquatic toxicity data for phosphoric acid

Taxa	Habitat	Acute LC(EC)50 Reported as pH value
Invertebrates	Freshwater	≤4.6
	Marine	---
Fish	Freshwater	≤3.5
	Marine	---

--- = No data available.

ECB (2000) has summarised aquatic toxicity data for two species of freshwater invertebrates (*Daphnia magna* and *D. pulex* waterfleas), with derived 12-hour LC50 values of pH 4.6 and 4.1, respectively. The survival rate was found to depend on the pH value generated by the acid. In addition, a 12-hour LC50 for amphipods (*Gammarus pulex* and *G. fossarum*) of pH 3.4 has been derived.

The acute (96-hour) toxicity of concentrated phosphoric acid has been studied in two species of freshwater fish including Bluegill sunfish (Lepomis macrochirus) and mosquito fish (Gambusia affinis) under static test conditions. ECB (2000) reported aquatic toxicity data for phosphoric acid, with a 96-hour LC50s of pH 3.0 to 3.5 for both species. At sublethal concentrations of acids, L. macrochirus become hypoactive with respect to their swimming behavior (Ellgaard and Gilmore, 1984, as cited by ECB, 2000). The LC50 range of pH 3.0 to 3.5 for fish is common to phosphoric acid and several other acids including sulfuric acid, nitric acid and hydrochloric acid. The contribution of the anions of these acids to fish toxicity is similar.

Most natural freshwaters have a pH in the range 6.5 to 8.0, while the pH of most marine waters is close to 8.2. The pH in most waters is controlled by the carbonate-bicarbonate buffer system, which is particularly strong in marine waters ANZECC/ARMCANZ (2000).

13.2.3 Phosphorus and aquatic ecosystems

Phosphorus occurs naturally in waters of aquatic ecosystems, typically at concentrations that limit plant and algae growth (Cullen, 1986, Donnelly et al., 1992; cited by NSW EPA, 2000). Nevertheless, excess phosphorus can lead to excessive growth of algae (blooms) and other aquatic plants. Eutrophication is the over-enrichment of a body of water with nutrients, primarily nitrogen and phosphorus,

resulting in excessive growth of organisms and depletion of the oxygen concentration, often affecting other aquatic organisms. NSW EPA (2000) indicates that in most instances, algal blooms are harmless; however, cyanobacteria can cause environmental problems either by giving rise to odours and taints, or by producing toxins. A number of cyanobacteria species may produce one or more potent toxins with up to four modes of action including hepatotoxins (liver and internal organs), neurotoxins (neuromuscular blocking agents), endotoxins (dermatitis and conjunctivitis) and cytotoxins (internal organs) (Water Directorate, 2001). These toxins may potentially pose risks to aquatic organisms, wildlife and people. Other negative effects of excessive algal growth and decay include reduced light penetration into water, unpleasant smells from decomposing algae, restriction of fish migration and impairment of recreational use of water (e.g. boating, fishing, swimming).

In marine waters (including estuaries), blooms of diatoms and dinoflagellates may occur. Many dinoflagellates, whose blooms are known as 'red tides', can produce toxins that lead to skin irritations and illness in exposed humans. They can also enter the food chain where they are particularly hazardous to human consumers of seafood, who may contract an illness known as paralytic shellfish poisoning (NSW EPA, 2000).

Nutrient pollution of waters is a major environmental issue in Australia. The NSW EPA highlighted important anthropogenic sources of nutrients in ground water, fresh surface waters, estuarine waters and marine waters including:

- run-off from urban and rural residential areas (including road run-off);
- discharges from sewage treatment plants (STPs) and septic systems;
- erosion and run-off from grazing and cultivated land;
- run-off from intensive animal industries and forestry;
- tailwater from irrigation areas; and
- increasing riverbank and streambank erosion arising from the removal of vegetation.

The significance of diffuse sources in any given situation depends on the yield of nutrient generated by the particular land-use activity and the area or proportion of the catchment devoted to that activity, i.e., kilograms of nutrient per hectare per annum. Generally, the highest yields of nutrients are from urban areas, with lower yields from agricultural and forested catchments (Campbell & Doeg, 1989, as cited in ANZECC and ARMCANZ, 2000).

In NSW in 2000, the NSW EPA indicated that most inland rivers had concentrations of phosphorus that exceeded management objectives for the control of eutrophication. Generally, phosphorus concentrations increased with distance downstream; however, phosphorus concentrations in coastal rivers were generally low (NSW EPA, 2000). NSW EPA (2000) phosphorus management objectives for phosphorus for the management of eutrophication is a scale:

- <0.02 mg Total Phosphorus (TP)/Litre - Good
- 0.02 to 0.05 mg TP/Litre - Fair
- >0.05 mg TP/Litre - Poor

A value of >0.05 mg TP/Litre would exceed the phosphorus management objective. As indicated later in Section 15.5.2, caution is advised when trying to correlate total

phosphorus with prediction of eutrophication and algae bloom (type, magnitude or duration), as phosphorus may not be linked due to other important factors.

Regarding coastal rivers, NSW EPA (2000) indicates that between 1997 and 1999 two algal blooms potentially harmful to marine organisms and seven blooms potentially toxic to humans were recorded. These bloom frequencies were similar to those recorded between 1994 and 1996. The high nutrient (nitrogen and phosphorus) levels in stormwater and sewer overflows affect many of the urban estuaries in Sydney.

Polluted stormwater in urban areas is a major non-point-source contributor to poor water quality. During periods of high rainfall, significant volumes of water enter rivers and streams, transporting nutrients into waterways. Stormwater in urban areas is a particular problem because of the large area of paved surfaces and complex drainage systems (NSW EPA, 2000).

Depending on the level of treatment, the hundreds of licensed sewage treatment plants in NSW discharge effluent that can affect water quality, particularly during periods of little rainfall and low river flow. Sewage treatment plants are a significant contributor of nutrients in all NSW waterways (NSW EPA, 2000).

There are no definitive guidelines to assess the risks posed by marine and estuarine algal blooms, primarily because of the number of species that are potentially toxic and the variability in toxicity of algal blooms. The degree of toxicity depends on environmental and physiological factors (NSW EPA, 2000).

The National Land and Water Resources Audit (AMU, 2002) has identified nutrients (total phosphorus and total nitrogen) as a major surface water quality issue in Australia affecting most of the more intensively developed basins in the North-East Coast, Murray-Darling, South-East Coast and South-West Coast drainage divisions. Basins assessed to have nutrient levels within acceptable levels include the relatively well vegetated and less developed basins in areas such as north Queensland, north eastern Victoria and south western Australia.

14. Risk Characterisation

The information on the biological effects of the alky phosphate additive and related vehicle emissions presented in Section 10 is integrated in this section with the environmental, public and occupational exposure estimates developed in Section 9. An overall estimation is provided of the incidence and severity of the adverse effects the chemical may have on people and the environment in Australia. This process provides the basis for identifying areas of concern and evaluating risk management strategies.

14.1 Environmental risk

A hazard quotient (HQ) approach has been used to predict the hazard to terrestrial and aquatic organisms. The HQ is the ratio of the predicted environmental concentration (PEC) to the predicted no effect concentration (PNEC). To predict a low environmental hazard, the HQ value needs to be 1 or less (i.e. HQ $\leq$ 1).

No PNECs for phosphorus in waters for eutrophication and algal bloom end-points are available. ANZECC and ARMCANZ (2000) provide guidance levels only due to the complexity of correlating phosphorus concentration to algal bloom occurrence (Harris, 1994).

Leakages of LRP containing the alkyl phosphate additive from underground storage tanks (USTs) poses specific risks at local scales; however, the risk to the environment will depend on site-specific environmental conditions and in most situations it is expected to pose a negligible risk.

While emergency spill incidents and accidents and leakages from USTs may lead to the alkyl phosphate additive in the environment, terrestrial and aquatic wildlife are unlikely to be exposed at or above levels of concern under current use patterns.

14.1.1 Terrestrial risk

Animals

Most of the alkyl phosphate additive used each year will be destroyed during combustion within internal combustion engine cylinders with the emission of phosphoric acid and other exhaust gases. Animals may be exposed to phosphoric acid in air through inhalation.

Section 11 describes a NOAEL (in rats) of 50 mg/m^3 from two parallel 13-week inhalation studies of an aerosol of 95% red phosphorus and 5% butyl rubber combustion products containing ~70% phsophoric acids. A phosphoric acid $PNEC_{mammals}$ of 0.5 mg/m^3 is derived by dividing the NOAEL by an assessment factor of 100. Conservative estimation of potential phosphoric acid levels in air indicates PECs of 0.1 ng/L (current) and 0.040 ng/L (2004) (Table 4) These PECs are several orders of magnitude lower than the estimated phosphoric acid $PNEC_{mammals}$ of 500 ng/L.

Plants

No published phytotoxicity benchmark or data were available on the acceptable concentration of phosphoric acid in air for terrestrial plants. No records of adverse

effects on plants were identified in the literature in existing alkyl phosphate additive use areas. Phosphorus is an essential nutrient for plants and of low toxicity, only becoming a problem to terrestrial plants when exposed to excessive soil or foliar concentrations.

An estimate of the concentration of phosphoric acid deposition from air to land can be made using the Sydney use pattern as an example and the alkyl phosphate additive use scenarios developed in Section 9.3.1.

Research by Handreck (1997) suggests that Australian plants from a range of genre can tolerate phosphate concentrations of 0.9 kg/m^3 soil (at least 1400 mg PO_4^{3-}/kg soil assuming soil density of 1.6 kg/m^3). Sydney land area is 1550 km^2 or 1.55 x 10^9 m^2. With a biologically-active soil depth of approximately 0.1 m, this equates to 1.55 x 10^8 m^3 soil. Assuming conservatively that 50% of the soil surface is exposed, this equates to 7.75 x 10^7 m^3 of soil in the biologically-active zone in the Sydney region. Assuming soil density of 1.6 kg/m^3, the soil volume equates to 1.24 x 10^8 t (or 1.24 x 10^{11} kg) soil.

For Sydney, the estimated worst case (Scenario 1) atmospheric fallout of phosphorus is equivalent to about 11.5 tonnes of P or 35.3 tonnes when presented as PO_4^{3-} per annum (or 3.53 x 10^{10} mg)/annum. By assimilating the available soil mass (1.24 x 10^{11} kg) with the estimated atmospheric fallout (3.53 x 10^{10} mg/annum), the incremental amount of PO_4^{3-} per kg surface soil is estimated to be approximately 0.28 mg PO_4^{3-}/kg soil/annum.

Biological uptake by soil fauna and plants of an undefined portion of this phosphate would likely occur resulting in soil concentrations being lowered simultaneously. The proportion of land area is probably under-stated. It is doubtful whether soil phosphate concentrations would ever exceed phytotoxicologically-significant levels, even in Sydney soils.

14.1.2 Aquatic risk

The alkyl phosphate formulation DMA-4 (synonymous with Valvemaster™ Concentrate) has a median lethal concentration (LC50) for fish of 230 mg/L (95% CI 210-270 mg/L).

Concentrated phosphoric acid is acutely toxic to terrestrial and aquatic organisms, with LC50 values of pH 4.6 or less ; however, the likelihood of environmental pH values reaching levels of concern is unlikely due to low input and natural buffering capacity.

Phosphorus is a nutrient of plants and animals, but typically limits growth in the environment due to its low concentrations and/or low bioavailability. Excessive amounts of phosphorus can increase the risk of eutrophication and nuisance plant and algae growth in aquatic environments.

ANZECC and ARMCANZ (2000) indicate that a wide range of nutrient concentrations have been reported for Australian rivers and streams. Total phosphorus concentrations have been recorded in the range of 10 µg/L or less in near pristine mountain streams to approximately 1000 µg/L in polluted rivers. There are few published surveys of nutrient concentrations in estuarine waters of Australia and those available indicate considerable spatial variation in the nutrient concentrations in Australian waters. While no trigger values are available for phosphoric acid, ANZECC and ARMCANZ (2000) provide guidance ranges for total phosphorus in Australian aquatic ecosystems for use in preliminary assessment of risks of eutrophication and algal blooms to aquatic

ecosystems. Guidance levels for freshwater and marine ecosystems for total phosphorus are 10-50 and 25-30 μg/L, respectively (ANZECC and ARMCANZ, 2000).

The basis of the default trigger values is that phosphorus may potentially promote nuisance aquatic plant/algal growth in waterways. These indicative values are not standards that should never be exceeded. This is because other environmental conditions may enhance or limit aquatic plant or algal growth.

As indicated in Section 9.3.3, the predicted environment concentration (PEC) for total phosphorus in Sydney's stormwater derived from urban runoff using conservative assumptions may approximate only a fraction of the estimated background concentrations.

In general, effects from multiple natural and anthropogenic sources of phosphorus in the Australian aquatic environment will be cumulative. Together, they increase the risk to the aquatic environment of eutrophication and nuisance algal blooms. Nevertheless, comparison with typical background concentrations of total phosphorus in marine areas supports a conclusion of a negligible contribution, or low expected incremental risk, from use of LRP containing alkyl phosphate-based AVSR additives for the uses prescribed and the volumetric use rates estimated.

The above-mentioned PEC values are based on conservative assumptions including limited atmospheric dispersion, limited soil attenuation or other losses of phosphorus. Furthermore, the effects of dilution, mixing and sedimentation that are likely to occur in receiving waters have not been taken into account. No atmospheric data were available to verify the PEC estimates made in this assessment.

The two use scenarios developed in this assessment report represent the worst case examples for AVSR petrol additives. Elsewhere in Australia the amount of LRP used would be less than the worst-case example calculated in this assessment.

14.2 Occupational risk

There is no documentation on adverse human health effects due to the alkyl phosphate additive. Therefore, the risks characterisation to human health of the alkyl phosphate additive is assessed with regard to the proprietary products Valvemaster™ Concentrate and Valvemaster™ VM11 and the combustion by-product phosphoric acid.

The margin of exposure methodology is commonly adopted in international assessments for determining risk to to human health from exposure to chemicals (EC, 1994; OECD, 1994). For health effects caused by repeated or prolonged exposure, risk(s) have been characterised as follows:

- Identification of critical effect(s)
- Identification of the most appropriate/reliable NOAEL (if available) for the critical effect(s)
- Where appropriate, comparison of the NOAEL with the estimated human dose (EHD) or exposure, to provide a margin of exposure (MOE) that is defined as:

$$MOE = NOAEL/EHD$$

- Characterisation of risk by evaluating whether the MOE indicates a concern for the human population under consideration.

The MOE provides a measure of the likelihood that a particular adverse effect will occur under the conditions of exposure. As the MOE increases, the risk of potential adverse effects decreases. In deciding whether the MOE is of sufficient magnitude, expert judgement is required. Such judgements are usually made on a case-by-case basis and should take into account uncertainties arising in the risk assessment process, such as the completeness and quality of the database, the nature and severity of effect(s) and variability between and within species.

14.2.1 Critical health effects

The critical health effects described in this section are derived from toxicity studies of the alkyl phosphate formulation DMA-4 (see Section 10) and it's predicted combustion by-product, phosphoric acid, which is described in Section 8.

Alkyl phosphate additive

Animal (mammalian) studies of DMA-4 indicate the alkyl phosphate additive is not acutely toxic by oral or dermal exposure, although irritant effects are possible. No repeated dose data are available. While there are no toxicokinetic data available for the alkyl phosphate additive, exposure has the potential to add to the total body phosphate/phosphorus load.

In poorly reported studies, the acute lethal doses were reported as greater than 11 000 and as 17 000 mg/kg bw for oral exposure in male rats. The acute lethal dose via the dermal route in rabbits was greater than 5000 mg/kg bw.

Animal (mammalian) studies indicate moderate to severe dermal irritation following prolonged exposure (24-72 hours). The inclusion of kerosene in the alkyl phosphate additive formulation can not be discounted as a contributing factor to the observed irritant response observed in these animal studies.

Neither surrogate data nor Quantitative Structure Activity Relationships (QSAR) are used in this assessment report to predict irritancy. This is because even with simple inorganic phosphates, a wide range of eye and skin irritancy is reported (Weiner, 2001).

The inclusion of kerosene in the formulation presents additional adverse health effects such as irritancy and lung damage (if swallowed) (NOHSC, 1999b).

Phosphoric acid

The present assessment also considers the health effects of phosphoric acid which is identified as the combustion by-product of the alkyl posphate additive.

In animals (mammalian) and humans, the critical effects following acute exposure to phosphoric acid relate to the acidic nature and hence the corrosive effects of the acid. The corrosive effects of phosphoric acid include eye, skin, pulmonary and gastrointestinal irritation with the severity of the symptoms depending on the concentration and length of exposure to the acid.

Corrosion of the gastrointestinal tract and permanent eye damage are reported in humans following accidental oral ingestion and splashes to the eye, respectively, of phosphoric acid. Animal data confirm the eye and dermal irritancy of phosphoric acid with concentration, occlusion and length of exposure determining the severity of the adverse effect. A single drop into the human eye of 1.5% phosphoric acid reportedly

causes moderate stinging but no injury. Concentrations as low as 2.5-5% phosphoric acid causes skin irritation in animals.

There are limited human inhalation exposure data for phosphoric acid. Weakness, dry cough, chest pain and shortness of breath or dyspnea are reported following accidental occupational inhalation of high (unspecified) concentrations of phosphoric acid vapour. Symptoms of reactive airways dysfunction were evident one year after exposure.

In animals (mammalian), based on limited data, phosphoric acid has low acute oral, dermal and inhalation toxicity. Phosphoric acid does not appear to have been well studied with respect to repeated dose toxicity in animals or humans. No reports have been found in the literature of systemic poisoning following repeated inhalation exposure to phosphoric acid. Phosphorus pentoxide which hydrolyses to phosphoric acid also has low acute inhalation toxicity although there appears to be differences between species. Repeated dose toxicity data for phosphoric acid rely on phosphorus smokes generating phosphorus pentoxide which is hydrolysed to phosphoric acid. These studies show the nature of the effect is dependent on the concentration, duration of exposure and the hydroscopic character of the acid with interspecies differences.

Concentration dependent terminal bronchiolar fibrosis in male Sprague-Dawley rats is reported in two parallel 13-week inhalation studies. Both involved exposure to the combustion products of 95% red phosphorus and 5% butyl rubber, with a reported NOAEL of 50 mg/m^3. The study proposed 71-79% phosphoric acid in the inhaled aerosols.

Phosphorus

The WHO cites a maximum tolerable daily intake of phosphorus of 70 mg/kg bw, which, if calcium intake is adequate, equates to 4.2 g of phosphorus or 12.9 g of phosphate (an ionic form of phosphoric acid) for a 60 kg person.

14.2.2 Occupational health and safety risks

Manufacture

Exposure during the manufacture of the alkyl phosphate additive is predicted to be low given the fully enclosed manufacturing method, enclosed tranfer systems and use of personal protective equipment.

Exposure during manufacture to the alkyl phosphate additive via dermal and ocular routes is possible from spills and slops while manually connecting transfer lines, e.g., from the reaction vessels or on-site holding tanks to steel drums or ISO tanks.

Dermal and ocular expousure is also possible during manual sampling of the alkyl phosphate additive formulation from the reaction vessel for quality assurance (QA) testing and during laboratory testing of the QA sample.

The oral and dermal ALD values of 11 000-17 000 mg/kg bw and greater than 5000 mg/kg bw, respectively, for DMA-4 indicate the overall risk of acute health effects is low given the small amounts to which workers are likely to be exposed.

Exposure to the alkyl phosphate additive is expected to be infrequent, minimal and of short duration.

Oil company terminals and depots

Exposure levels during bulk LRP blending are predicted to be low given the typically automated and enclosed transfer activities and use of personal protective equipment (PPE). Non-automated additions of the alkyl phosphate additive at some depots may potentially result in increased occupational exposure for short (5 min) periods daily.

Exposure via the dermal and ocular routes is possible from slops, spills and residue during decanting of Valvemaster™ Concentrate from either drums or ISO tanks into storage tanks. Such exposure is expected to be infrequent, minimal and of short duration, although staff decanting from drums via a hand pump and a spear or a dedicated pumping system into a bulk tank may be exposed for a longer period of time, e.g. six hours, but infrequently, e.g., three times per year at most.

In general, as for manufacturing workers, the risk to the terminal's workers from handling the alkyl phosphate additive is assessed as low because direct handling of the alkyl phosphate additive is miminal or infrequent, or both.

Occupations associated with vehicles

Petrol stations, maintenance workshops and car parks

Tanker drivers, petrol station attendants and auto mechanics may be exposed occupationally to the alkyl phosphate additive through contact with LRP fuels and less frequently with aftermarket products containing the the alkyl phosphate additive. Although exposure to fuel vapours may be expected during a typical working day, the low vapour pressure of the alkyl phosphate additive formulations and its high dilution in fuel renders inhalation exposure to the alkyl phosphate additive unlikely.

Exposure to the alkyl phosphate additive via dermal and ocular routes is expected to be infrequent, minor and of short duration and also limited due to its dilution with solvents and other additives in the fuel and fuel additives. Therefore, the risk to petrol station and maintenance workshop workers from handling the alkyl phosphate additive is assessed as low. Car park personnel are not expected to handle the alkyl phosphate additive.

Auto mechanics at petrol stations and at dedicated maintenance workshops and car park personnel have the potential for repeated exposure to phosphoric acid associated with auto exhaust. Such exposure is likely to be highly variable depending on the level of separation from the exhaust sources and traffic densities. No personal exposure data for such workers are reported.

The level of emission of phosphoric acid at the tailpipe of vehicles using alkyl phosphate additive-based LRP is estimated in this assessment report to range from 6 to 8.3 mg/m^3 (equivalent to 600 to 830 ng/L) of exhaust gas depending on whether or not entrapment of phosphoric acid occurs in the exhaust system (Section 8).

Atmospheric dilution of the exhaust gas will occur. Dilution rates used in this report to predict atmospheric phosphoric concentrations around vehicles using the alkyl phosphate additive rely on the work of Dolan et al (Dolan et. al, 1979). The estimated atmospheric phosphoric acid concentration at 2 metres from the exhaust tailpipe is 0.06 to 0.083 mg/m^3 based on an exhaust gas dilution rate of 100. This is significantly lower than the phosphoric acid LC50 values of 111 to 6327 mg/m^3. There is the potential for higher occupational exposures to atmospheric phosphoric concentrations found in enclosed or poorly ventilated workshops or car parks and at busy petrol station forecourts.

For repeated occupational exposure, margins of exposure can be calculated from estimated atmospheric phosphoric acid concentration at 2 metres from the exhaust tailpipe by applying the NOAEL of 50 mg/m^3 for pulmonary fibrosis in rats (Ballantyne, 1998).

Applying the NOAEL of 50 mg/m^3, assuming 100% phosphoric acid in the smoke and converting intermittent exposures (4 days/week, 2.25 hours/day) to a working week (5 days/week, 8-hr day) exposure, the following margins of exposure are calculated:

At 2 m from the tailpipe, assuming no entrapment of exhaust phosphoric acid emissions (8.3 mg/m^3) and an exhaust gas dilution rate of 100:

Margin of Exposure = (50 mg/m^3 x 4/5 x 2.25/8)/0.083 mg/m^3 = 135 (3)

Using the same calculation above, the MOE at 2 metres is increased to 187 if entrapment of exhaust phosphoric acid emissions (6.0 mg/m^3) occurs.

The risk to petrol stations and maintenance workshops workers and car park personnel from phosphoric acid associated with auto exhaust is, therefore, considered low. Furthermore, the typical use of open workshops and limited forecourt service at service stations in Australia will decrease the exposure of maintenance and petrol station workers, respectively, to exhaust gas phosphoric acid. Exposure may be higher for car park personnel if they are confined to enclosed or poorly ventilated car parks.

Professional drivers and road maintenance workers

Like petrol station, maintenance workshop and car park personnel, professional drivers and road maintenance workers may be occupationally exposed to phosphoric acid exhaust emissions. No personal exposure data for professional drivers and road maintenance workers are reported.

Calculations based on Dolan's work (Dolan et. al, 1979) may be made of atmospheric phosphoric acid concentrations from the tailpipes of moving vehicles. An exhaust gas dilution rate is reported as typically 1000 in less than one second, with the dilution rates ranging from approximately 630 at idle to 5000 at 80 km/hour vehicle speeds (Dolan et. al, 1979). Consequently, the phosphoric acid exhaust gas levels of 68.3 mg/m^3 will be diluted to 9.52 x 10^{-3} mg/m^{3}to 0.0132 mg/m^3 (idle speed) and 1.2 x 10^{-3} mg/m^3 to 1.66 x 10^{-3} mg/m^3 (80 km/hr), respectively. These phosphoric acid concentrations are several magnitudes lower than the LC50 values for acute exposure and the NOAEL for repeated exposure.

Given that the exposure of professional drivers and road maintainence workers is lower than for petrol station, maintenance workshop and car park personnel, the risk of this group to phosphoric acid exhaust emissions in Australia is likewise considered to be low.

14.2.3 Uncertainties

Uncertainties exist in the assessment of risk to local workers from the use of the phosphorus AVSR additive. No Australian personal exposure data exist regarding exposures to the alkyl phosphate additive or phosphoric acid in the workplace.

There are no data on the potential health effects of the alkyl phosphate additive because it is never isolated; instead, it is manufactured in situ into a formulated proprietary product at a concentation range of 70-90%.

The limited number of toxicity studies relied on in this report relate to the kerosene-based alkyl phosphate additive formulation DMA-4. The kerosene in DMA-4 will be a confounding factor in determining the toxicology profile of the alkyl phosphate additive.

There are limitations in the DMA-4 toxicological data. For example, few of the DMA-4 studies meet standard test guidelines. No repeated dose toxicity, reproductive effects, genotoxicity or carcinogenicity data in humans and animals are available for the alkyl phosphate additive or DMA-4. Also, there is insufficient data to determine specifically the irritancy potential of the alkyl phosphate additive.

There are limited data on the inhalation toxicity of the phosphoric acid. Animal (mammalian) studies rely on smokes generated from the oxidation of phosphorus and may contain combustion products other than phosphoric acid. Furthermore, there appears to be interspecies differences in the toxic effects of phosphoric acid inhalation that makes the extrapolation of such data to humans problematic.

There is also uncertainty associated with the estimation of potential doses that may be received and the extrapolation from animal data to humans.

Uncertainities exist in estimating the atmospheric phosphoric acid concentrations because exhaust gas dilution rates will vary depending on distance from the tailpipe, wind speeds and ventilation rates, different volume flow rates and velocities of the exhaust gases as well as the speed and type of vehicle.

Adding to these uncertainties in the assessment of occupational risk in this report are:

- climatic conditions (which may affect the extent or otherwise of the use of enclosed workplaces and dissipation of the the AVSR additive and phosphoric acid)
- lack of ambient alkyl phosphate additive and phosphoric acid levels and exhaust gas analyses on the alkyl phosphate additive and
- differences in local use patterns for AVSR additives.

14.3 Public health risk

14.3.1 Acute effects

Direct public exposure to the alkyl phosphate additive is likely to occur primarily via the skin as a result of spills and splashes of LRP and aftermarket products. The alkyl phosphate additive is not expected to be a skin or eye irritant at the concentrations present in LRP. There is a low risk of acute systemic health effects in the general public as a result of skin exposure to the alkyl phosphate additive.

Skin irritation is possible following exposure to aftermarket products containing the alkyl phosphate additive, due in part to the presence of kerosene.

Acute health effects could occur as a result of accidental ingestion by a child. Accidental ingestion might occur in young children if aftermarket products are stored in or around the home. Children between one and a quarter and three and a half years of age can swallow approximately 4.5 mL of liquid (Gosselin et al., 1976).

Given the RDA of phosphorus for infants 1 to 8 years of age is approximately 500 mg (equivalent to 50 mg/kg bw for a 10 kg child), ingestion of half the contents of a 10 mL

single use aftermarket product corresponds to 23.5 mg/kg bw phosphorus for a 10 kg child or approximately half of the phosphorus RDA (Section 9.7.1). This ingested alkyl phosphate additive dose corresponds to 368 mg/kg bw for a 10 kg child compared with the DMA-4 oral acute lethal dose (in rats) of 11 000-17 000 mg/kg bw.

The multi-treat product contains a less concentrated alkyl phosphate additive formulation compared with the single use product and, therefore, the risk is lower.

Small ingested volumes of the alkyl phosphate additive may potentially occur in children, but will not significantly add to the phosphorus body load nor represent a significant acute health risk. Ingestion of the kerosene contained in the product, however, may present an additional risk due to in advertent inhalation and the potential lung damage that can occur with kerosene.

In general, public exposure to the alkyl phosphate additive is expected to be minimal and of short duration. Consequently, the risk to the public from handling the alkyl phosphate additive is assessed as low.

The public's inhalation exposure to phosphoric acid from tailpipe emissions is likely to be lower than for occupational exposure. This is because of the rapid dissipation of phosphoric acid from the tailpipe of moving vehicles (Dolan et. al., 1979).

14.3.2 Chronic effects

Total phosphorus exposures from the combination of all sources is unlikely to be changed significantly by the use of the alkyl phosphate additive. This is because the ingestion of food represents by far the greatest proportion of the total daily phosphorus dose (Section 11.2).

Data in Table 4 shows that the use of the alkyl phosphate additive according to the present use scenario of maintained LRP market share and the 2004 scenario of diminished LRP market share will increase the phosphoric acid dose inhaled to levels above assumed negligible ambient air concentrations.

Applying the NOAEL of 50 mg/m^3 (50 x10^6 ng/m^3), assuming 100% phosphoric acid in the smoke and converting intermittent exposures (4 days/week, 2.25 hours per 24 hours/day) to continuous exposures, the following margins of exposure are calculated:

For Present Use scenario, where current LRP market share is maintained:

Margin of Exposure = (50 x10^6 ng/m^3 x 4/7 x 2.25/24)/100 ng/m^3 = 26 786

For 2004 scenario, where the LRP market share declines:

Margin of Exposure = (50 x10^6 ng/m^3 x 4/7 x 2.25/24)/40 ng/m^3 = 66 964

These margins of exposure are considered satisfactory and the risk is considered low.

The US EPA have derived a chronic inhalation reference concentration (RfC) that is based on bronchiolar fibrosis of the respiratory tract in rats for phosphoric acid of 10 μg/m^3 (IRIS, 2003). In general, the RfC is an estimate of a daily inhalation exposure of the human population (including sensitive subgroups) which is proposed as likely to be without an appreciable risk of deleterious effects during a lifetime. The RfC value assumes that thresholds exist for certain toxic effects such as cellular necrosis.

The RfC determined by the US EPA uses uncertainty factors to account for intraspecies and interspecies variability. It is extrapolated from subchronic to chronic exposure, specific data on bronchiolar fibrosis in the tracheobronchial region in Sprague-Dawley rats, estimates of the diameter of the hydrated aerosol particles at the site of the pulmonary lesions and an average weight of the rats.

There is currently no Australian ambient air standard for phosphoric acid. Comparing the estimated ambient air concentrations in Table 4 (up to 100 ng/m^3) with the RfC value set in the US (10 $\mu g/m^3$), it is apparent that the estimated phosphoric acid air concentrations for Scenarios 1 and 2 are much lower than the US EPA RfC value.

14.3.3 Uncertainties

Like uncertainties associated with occupational risk assessment, uncertainties involved in the chronic public health risk assessment are derived in part from significant database limitations. There is a general lack of public exposure data and Australian baseline ambient air phosphoric acid data on which to base a realistic exposure assessment.

15. Risk Management

This section discusses measures currently employed in the management of human and environmental risks from exposure to the alkyl phosphate additive.

The key elements on the management of risks discussed in this section are:

- Workplace control measures
- Hazard communication and
- Regulatory controls.

15.1 Assessment of current control measures

According to the NOHSC *National Model Regulations for the Control of Workplace Hazardous Substances* (NOHSC 1994a), exposure to hazardous substances should be prevented or, when 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 1994b) provides further guidance in the form of a hierarchy of control strategies, namely, elimination, substitution, isolation, engineering controls, safe work practices and personal protective equipment (PPE).

15.1.1 Elimination and substitution

Elimination is the removal of a chemical from a process and should be the first option considered when minimising risks to health. In situations where it is not feasible or practical to eliminate the use of a chemical, substitution should be considered. Substitution includes replacing the chemical with a less hazardous substance or the same substance in a less hazardous form.

As indicated in Section 7.3.1, there is a declining market for LRP sales and hence AVSR additives due to attrition from the Australian motor fleet of vehicles designed to run on leaded petrol.

By 2004, bulk sales of LRP are expected to decline to less than 5% of total petrol sales (Australian Petroleum Gazette, 1999). The general provision and sale of bulk LRP by the oil companies will become uneconomical at some point. This will eliminate the requirement for the addition by oil companies of the alkyl phosphate additive to fuel.

When bulk LRP is phased out, aftermarket addition of AVSR fuel additives will become the only option for those motorists with vehicles designed to run on leaded petrol. Given the likelihood that mechanical alteration of engine components to run on ULP may be prohibitive for some vehicles, e.g. vintage vehicles, the total elimination of AVSR additives from the Australian fuel market is unlikely and the use of the alkyl phosphate additive as an aftermarket additive may continue indefinitely.

Several AVSR additives that are potential substitutes for the alkyl phosphate additive are available on the Australian market (Section 1.1). Users will need to consider the efficacy, cost, health, safety and environmental effects of each as alternatives to the alkyl phosphate additive.

15.1.2 Isolation and engineering controls

Isolation as a control measure aims to separate employees, as far as practicable, from the chemical hazard. This can be achieved by distance, use of barriers or enclosure. Engineering controls are plant or processes which minimise the generation and release of hazardous substances. They include total or partial enclosure, local exhaust ventilation and automation of processes.

Manufacturing plant

At the manufacturing plant, the alkyl phosphate manufacturing vessel is isolated in a bunded building. Engineering controls include a fully enclosed manufacturing process with the reaction vessel connected to a scrubber system that treats any vapours emitted during the manufacturing process. The finished manufactured product is transferred from the reaction vessel to a storage tank, ISO tanks or drums using manually controlled enclosed and dedicated transfer lines. The reaction vessel is connected via a bursting disc to a catch tank which would contain product in the unlikely event that control of the reaction was lost.

Oil company terminals and depots

Oil company terminals use a number of different control measures when receiving the bulk alkyl phosphate additive from the manufacturing plant and when blending bulk LRP for distribution to service stations. These control measures generally rely on closed transfer systems with the nature of the control measure dependent on the type of container received from the manufacturing plant, e.g. ISO tanks or drum lots of the alkyl phosphate additive.

Decanting of drummed alkyl phosphate additive at the oil terminals is carried out using a sealed and dedicated transfer system into the bulk storage tank. While full drums of the alkyl phosphate additive are stored undercover prior to decanting (in an approved flammable drum storage area), the decanting occurs on an open air concrete bunded bay with workers manually transferring a suction pipe from drum to drum during the process. Drum decanting is limited to three times per year. On occasions when it is necessary to discharge drums of the alkyl phosphate additive, a hand pump and a spear into the underground tank is reported to be used.

For bulk LRP blending, the terminals use an automated closed in-line dosing system for adding the alkyl phosphate additive to road tankers. The alkyl phosphate additive is pumped directly from the bulk storage tank and injected into the unleaded petrol stream as the LRP road tanker is loaded with fuel.

During pipeline maintenance, drainage into containers for return to storage tanks is used at terminals to limit accidential release of the alkyl phosphate additive.

Once of the ISO tankers of the alkyl phosphate additive are received at the terminal, discharge by gravity feed into a closed underground tank via a manually controlled, closed delivery hose system is used.

No engineering controls are reported at some oil company depots, i.e. small terminals, which use manual addition by workers of the alkyl phosphate additive to road tankers. The manual addition (undertaken by workers wearing PPE) involves decanting the alkyl phosphate additive into a receptacle prior to pouring it into the road tanker compartment.

Petrol stations

All oil companies report that transportation of the alkyl phosphate additive-based LRP to service stations is carried out using dedicated petroleum road tankers. Isolation and engineering controls are achieved at petrol stations by using enclosed transfer hoses for transferring LRP containing the alkyl phosphate additive from the road tankers and storage in underground tanks.

With regards to USTs, there are currently no existing leak prevention or detection requirements for operators of underground fuel storage tanks in all states to detect and control leakages from UST facilities. UST leak detection systems are implemented on a voluntary basis by industry, particularly by major petroleum suppliers

Aftermarket product use

Engineering controls for public and occupational users of aftermarket products containing the alkyl phosphate additive consist presently of single use injection devices, which completely empty when the contents are injected into the petrol tank and multi-treat bottles, which have childproof screw caps and measuring chambers designed to drain completely into the petrol tank. The viscous nature of alkyl phosphate additive formulation contained in the single use aftermarket product lends itself to minimisation of any leakage or dripping of the formulation when the device is handled.

15.1.3 Safe work practices

Safe work practices are administrative practices that require people to work in ways that are safe.

Manufacturing plant

At the alkyl phosphate additive manufacturing plant, all work procedures relating to the manufacture and handling of the alkyl phosphate additive are documented. This documentation includes instructions regarding temperature and vacuum controls as well as PPE requirements, categories of exposure to be avoided and saftey precautions to be taken due to the properties of the chemicals handled.

Oil company terminals and depots

At oil company terminals and depots, all work procedures relating to the handling and storage of the the alkyl phosphate additive are documented as are the PPE requirements for handling of the fuel additive. Procedures for maintenance and calibration of oil terminal in-line dosing systems are reported. Terminals have written procedures for decanting drums and transferring product containing the alkyl phosphate additive. Areas are designated for this task and operators trained in the correct handling procedures for the product and task, with full access to the Material Safety Data Sheet (MSDS).

15.1.4 Personal protective equipment

Where other measures are not practicable or adequate to control exposure, personal protective equipment (PPE) should be used. The PPE requirements recommended by the manufacturers for the handling the alkyl phosphate additive are based on avoidance of the potential irritant effect of the kerosene-based product and hence the prevention of skin and eye contact.

Manufacture

Specific safety requirements are provided to operators via the safety instructions on the manufacturing batch sheet. During the manufacture of the alkyl phosphate additive, operators wear protective overalls, impermeable gloves, safety footwear and safety glasses at all times. In addition, the wearing of a face shield, a protective apron and respiratory protection such as a particle filter respirator is recommended during the handling of components during the manufacturing process. During analysis of the alkyl phosphate additive samples for quality control tests, the analyst wears protective clothing including a lab coat, safety footwear, and safety gloves and glasses.

Oil company terminals and depots

The practice at oil company terminals is to use gloves, protective clothing and safety shoes and glasses during the discharge of the ISO tankers and the decanting of the drums containing the alkyl phosphate additive. Similar PPE is also worn during in-line dosing of the road tanker with the alkyl phosphate additive and during maintenance of equipment used in the various processes.

Typically, protective clothing such as PVC gloves, safety glasses, wrist to ankle clothing and safety boots are worn by the terminal workers during drum decanting.

Depots where the alkyl phosphate additive is added manually to the road tanker, recommend that an organic vapour cartridge respirator be worn along with rubber gloves and safety goggles.

Petrol stations and maintenance workshops

At petrol stations during the unloading of road tankers and the dipping of underground tanks, workers typically wear protective clothing, footwear and gloves. Auto mechanics with potential exposure to the alkyl phosphate additive in fuel also wear protective clothing and footwear.

15.2 Hazard communication

15.2.1 Labels

Labels for two aftermarket alkyl phosphate additive products and one label for alkyl phosphate additive used in bulk LRP blending were available for assessment.

Labels submitted for assessment were assessed for requirements under the NOHSC *National Code of Practice for the Labelling of Workplace Substances* (NOHSC, 1994c). The assessment took the form of a qualitative appraisal of the following categories of information:

Substance identification;

- hazard category/Signal word
- ADG Code classification/packaging group
- details of manufacturer or supplier
- risk information (or phrase)
- safety information (or phrase)

- information on spills, leaks or fires and
- reference to MSDS.

There are insufficient toxicity data for the alkyl phosphate additive to be classified in accordance with the NOHSC *Approved Criteria for Classifying Hazardous Substances* (NOHSC 1999a). The limited data for the alkyl phosphate additive products suggest the alkyl phosphate additive has a low acute oral and dermal toxicity and may cause dermal irritation.

Kerosene is a major ingredient of the Valvemaster™ products (Section 4) and is classified as a hazardous substance (NOHSC 1999a). Given the kerosene content of the alkyl phosphate additive products, the labels should contain the following hazard classification, risk and safety phrases with regard to the NOHSC *Approved Criteria for Classifying Hazardous Substances* (NOHSC 1999a):

Products containing kerosene ≥ 10% should be classified as Xn (Harmful) with the risk phrase R65: Harmful: May cause lung damage if swallowed. The most appropriate safety phrases are S2: Keep out of reach of children, S23: Do not breathe vapour, S24: Avoid contact with skin and S62: If swallowed, do not induce vomiting; seek medical advice immediately and show this container or label.

An additional classification for kerosene is Xi (Irritant) with the risk phrase R38: Irritating to skin, in products where the kerosene content is ≥ 20%. This additional classification for kerosene (unspecified nature) is sourced from the Australian Institute of Petroleum AIP (NOHSC 1999a).

Solvent naptha at concentrations greater than or equal to 10% is classified as Xn (Harmful) with the risk phrase R65: Harmful: May cause lung damage if swallowed. The most appropriate safety phrases are S2: Keep out of reach of children, S23: Do not breathe vapour, S24: Avoid contact with skin and S62: If swallowed, do not induce vomiting; seek medical advice immediately and show this container or label (NOHSC 1999a).

Bulk alkyl phosphate additive

One label for the alkyl phosphate additive formulation (Valvemaster™ Concentrate) used in bulk LRP blending was available for assessment.

The label contained identification information and the signal words and risk phrases "Hazardous", "Harmful: may cause lung damage if swallowed", and "irritating to eyes, respiratory tract and skin". The label contained appropriate signal words and safety phrases, first aid and emergency procedures and included details of the Australian manufacturer and references to the MSDS.

Aftermarket products

In the case of labelling of hazardous substances of 500 mL capacity or less and where space on the containers is especially limited, the NOHSC Labelling Code describes the required minimum information as:

- signal words
- product name and
- details of manufacturer or importer.

Products sold to the public should meet the requirements of the *Standard for the Uniform Scheduling of Drugs and Poisons* (SUSDP, 2002) which describe labelling and packaging requirements.

10 mL aftermarket products

The 10 mL aftermarket consumer product does not need to meet the requirements of the SUSDP (SUSDP, 2002). This is because the formulation's container has a nominal capacity less than 15 mL (SUSDP, 2002).

The 10 mL label contains the product name and appropriate signal words, e.g. 'harmful', 'irritating' and discloses the exact kerosene and phosphate additive content (volume/volume concentration) of the product. The label contains details of the overseas packaging company (but not the local distibutor), and describes the recommended use and the directions for use, with advice to refer to the brochure for full instructions.

The label contains the safety phrases S2: Keep out of reach of children, S23: Do not breathe vapour and S24: Avoid contact with skin, but not S62: If swallowed, The label notes the product may be harmful if swallowed and includes the phrase "Caution".

250mL aftermarket product

The 250 mL label contains the product name and the appropriate signal words, harmful and irritating, details of the local distributor, and disclosure of the exact kerosene content of the product but not the concentration (nor concentration range) of the alkyl phosphate additive. The label identifies the product's recommended use and gives directions for use. The label contains the safety phrase S2: Keep out of reach of children, and the phrase "Caution".

The 250 mL aftermarket product contains the safety phrase S62: If swallowed, do not induce vomiting; seek medical advice immediately and show this container or label. The label includes advice that the product is "irritating to skin", "harmful, may cause lung damage if swallowed". First aid advice is grouped together and prefaced by the words "FIRST AID" and the contact number of the Poisons Information Centre is included. The 250 mL plastic container is embossed with the word "POISON".

15.2.2 MSDS

Material Safety Data Sheets (MSDSs) are the primary source of information for workers involved in the handling of chemicals. Under the NOHSC *National Model Regulations for the Control of Workplace Hazardous Substances* (NOHSC 1994a) and the corresponding state and territory legislation, suppliers of a hazardous chemical for use at work are obliged to provide a current MSDS to their customers and employers must ensure that an MSDS is readily accessible to those employees with potential for exposure to the chemical.

A total of two MSDSs, one for the alkyl phosphate additive formulation used in bulk LRP blending and the single-use aftermarket product, and one for the multi-treat aftermarket product, were available for assessment against the NOHSC *National Code of Practice for the Preparation of Material Safety Data Sheets* (NOHSC 1994e). The results of the MSDS assessment are presented in Appendix 3.

In general, the MSDSs comply well with the NOHSC *National Code of Practice for the Preparation of Material Safety Data Sheets* (NOHSC 1994e). The Valvemaster™ Concentrate MSDS, however, did not identify the recommended use.

The MSDS for the multi-treat aftermarket product Valvemaster™ VM11 assigns a Class 9 Dangerous Good classification (UN Number 3082 Environmentally Hazardous Substance, Liquid, NOS) with regards the ADG Code (FORS, 1998). This is consistent with the inclusion of Solvent Naptha in the formulation.

Although an assessment of the toxicology of kerosene and solvent naptha have not been undertaken in this assessment report, the inclusion of such information in the MSDS is regarded as pertinent.

15.2.3 Education and training

Guidelines for the induction and training of workers exposed to hazardous substances are provided in the NOHSC *National Model Regulations for the Control of Workplace Hazardous Substances* (NOHSC 1994a). Under these regulations, employers are obliged to provide training and education to workers handling hazardous substances. These regulations stipulate that training and induction should be appropriate for the workers concerned.

Oil company terminals and depots that use the alkyl phosphate additive specify procedures for handling the fuel additive including PPE requirements.

15.3 Occupational monitoring and regulatory controls

15.3.1 Atmospheric monitoring

Under the NOHSC Model Regulations (NOHSC, 1994a), employers are required to carry out an assessment of the workplace for all hazardous substances. The NOHSC *Guidance Note for the Assessment of Health Risks Arising from the Use of Hazardous Substances in the Workplace* (NOHSC, 1994d) provides the methodology for this assessment. When the assessment indicates that the risk of exposure via inhalation is significant, atmospheric monitoring should be conducted to measure levels of the hazardous substances in the workplace before the implementing of suitable control measures to reduce exposure. Subsequent monitoring is also required to ensure that such measures are effective.

No atmospheric monitoring programs for the alkyl phosphate additive in workplaces have been identified and none is considered necessary.

15.3.2 Occupational exposure standards

While Australia as well as other countries have not set occupational exposure standards for the alkyl phosphate additive, it has set occupational exposure limits for phosphoric acid.

According to the NOHSC *Exposure Standards for Atmospheric Contaminants in the Occupational Environment*, the current Australian national occupational exposure standard for phosphoric acid is 1 mg/m^3, expressed as an 8-hr time-weighted average (TWA) airborne concentration (NOHSC, 1995). In Australia, the short-term exposure limit (STEL) for phosphoric acid is 3 mg/m^3. The Australian occupational exposure limits for phosphoric acid are comparable to those set by the majority of overseas

countries (ACGIH, 2000). Some countries, however, have set an 8-hr TWA but not a STEL value or vise versa (ACGIH, 2000). Both Ireland and the Netherlands have set a STEL value of 2 mg/m^3 (ACGIH, 2000).

NIOSH cite a revised Immediately Dangerous to Life or Health (IDLH) concentration for phosphoric acid of 1000 mg/m^3 based on acute oral toxicity data in animals (NIOSH, 1996). This IDHL concentration for phosphoric acid is cited by NIOSH as a conservative value due to the lack of relevant acute toxicity data for workers.

According to the NOHSC Exposure Standards (NOHSC, 1995) a process is not considered to be under reasonable control if short-term exposures exceed three times the TWA exposure standard for more than 30 minutes per 8 h working day, or if a single short-term value exceeds five times the TWA exposure standard.

15.3.3 Health surveillance

In accordance with NOHSC Model Regulations (NOHSC, 1994a), employers have a responsibility to provide health surveillance in those workplaces where the workplace assessment indicates that exposure to a hazardous substance may lead to an identifiable substance-related disease or adverse health effect. The alkyl phosphate additive is not listed in Schedule 3 (list of substances requiring health surveillance) and as such there are no formal requirements for health surveillance programs for exposed workers.

No personal air monitoring or health surveillance programs for the alkyl phosphate additive have been reported in Australia.

15.3.4 National transportation regulation

The alkyl phosphate additive is not listed in the *Australian Code for the Transport of Dangerous Goods by Road and Rail* (ADG) Code, however Valvemaster™ and Valvemaster™ VM11 meet the criteria for C1 combustible liquids (Section 12.2).

It is noted with respect to the ADG Code Section 2.1.10 (FORS 1988) sub-clause (1) that combustible liquids are taken to be dangerous goods of Class 3 if:

(a) the combustible liquids are transported in a bulk container or a tank which is part of a vehicle; and

(b) the combustible liquids are transported on the same vehicle with:

(i) dangerous goods of Class 3 in bulk; or

(ii) packaged dangerous goods of Class 3 in an aggregate quantity of more than 1000 L.

Sub-clause (2) of the ADG Code Section 2.1.10 (FORS 1988) does not apply to the transport of combustible liquids and dangerous goods of Class 3 on a rail wagon if the combustible liquids and the dangerous goods are in different bulk or freight containers which are separated by at least 12 metres. .

The *National Standard for the Storage and Handling of Workplace Dangerous Goods* (NOHSC, 2001) and *National Code of Practice for the Storage and Handling of*

Dangerous Goods (NOHSC, 2001b) complement the *National Model Regulations for the Control of Workplace Hazardous Substances* (NOHSC 1994a).

The *National Standard for the Storage and Handling of Workplace Dangerous Goods* sets out requirements to ensure the effective control of the storage and handling of dangerous goods in order to protect the safety and health of workers and the public and to protect property and the environment (NOHSC 2001a). The accompanying *National Code of Practice for the Storage and Handling of Dangerous Goods* (National Code) provides practical advice on compliance for those who have duties under the National Standard. In addition, the National Code provides information and guidance for storage and handling of dangerous goods as minor quantities and as consumer packages supplied by retailers.

The National Standard and National Code for the storage and handling of dangerous goods apply only to hazardous substances in the workplace that meet the classification requirements for dangerous goods. Where dangerous goods are stored or handled in private residences or other premises which are not workplaces, the National Standard and National Code may provide useful guidelines for ensuring health and safety.

The *National Standard for* the *Storage and Handling of Workplace Dangerous Goods* specifies the trigger levels (based on the quantities in Schedule 1) for the following requirements for C1 combustible liquids:

- **Placarding** — **Placard Level - Schedule 1**
- **Develop a manifest and site plan for the premises** — **Manifest Level - Schedule 1**
- **Develop of an Emergency Plan** — **Manifest Level - Schedule 1**
- **Notify the Appropriate Authority** — **Manifest Level - Schedule 1**

For C1 combustible liquids, a placard as specified in Schedule 2 of the National Standard is required for C1 combustible liquid in quantities exceeding 10 000 L in bulk containers, 50 000 L in packages, and 50 000 L combined bulk and packaged (provided the quantity of C1 combustible liquids in bulk does not exceed 10 000 L). Similarly, an occupier of a premise must ensure that the above requirements triggered at the Manifest Level of Schedule 1 are complied with in instances where the total quantity of a C1 combustible liquid exceeds 100 000 L bulk or packaged.

15.3.5 Control of major hazard facilities

According to the NOHSC *National Standard for the Control of Major Hazard Facilities* (NOHSC, 1996), the alkyl phosphate additive is not one of the specifically identified chemicals that must be considered when determining whether a site is a major hazard facility.

15.4 Public health regulatory controls

The alkyl phosphate additive is not currently included in a Schedule of the *Standard for the Uniform Scheduling of Drugs and Poisons* (SUSDP, 2002).

15.5 Environmental regulatory controls

This section provides information on the environmental regulatory controls governing Valvemaster™ and phosphorus compounds in Australia with reference to international

control initiatives. In brief, the management of environmental pollution and waste in Australia is regulated through individual state and territory regulatory systems rather than at a national level. All states and territories have general environment protection legislation pertaining to pollution and contaminated land.

However, there are currently no existing leak prevention or leak detection requirements for operators of underground fuel storage tanks in NSW, and probably other states and territories, to detect and control leakages from UST facilities. Leakages from USTs have historically resulted in contamination of the environment. In Australia, UST leak detection systems are implemented on a voluntary basis by industry, particularly by major petroleum suppliers.

15.5.1 Air quality management

Australia

Emissions of 'air toxics' (defined below) in Australia are regulated through individual state and territory regulatory systems rather than at a national level. Each state and territory has established legislative frameworks and strategies for monitoring and managing air quality. National strategies are or have been developed to allow consistent management of ambient air quality throughout Australia (refer below).

Air toxics are gaseous, aerosol or particulate pollutants that are present in the air in low concentrations. Characteristics such as toxicity or persistence make them hazardous to human, plant or animal life. The terms 'air toxics' and 'hazardous air pollutants' (HAPs) are used interchangeably. Air toxics include volatile and semi-volatile organic compounds, polycyclic aromatic hydrocarbons, metals and aldehydes (NEPC, 2002). Specific emission limitations and maximum ground level concentrations for individual sources are used in some states to control emissions from industrial sources (NEPC, 2002).

Emissions of air toxics from new motor vehicles are controlled through Australian Design Rules that set emission standards for a range of pollutants. These are set at a national level rather than state or territory level. Recently the Australian Government introduced National fuel quality standards that will reduce the level of some air toxics in ambient air. Currently, no AVSR is listed in the register of prohibited fuel additives.

At a national level, at least two National Environment Protection Measures (NEPMs) apply to air quality including the National Pollutant Inventory (NPI) NEPM (NEPC, 1998a) and the Ambient Air Quality NEPM (NEPC, 1998b). An additional NEPM (Ambient Air Toxics) is also being developed (NEPC, 2002). The Ambient Air Quality National Environment Protection Measure (NEPM) (NEPC, 1998b) sets national standards for six air pollutants. Phosphorus compounds are not specifically included in either of the two Ambient Air Toxics NEPMs being developed (refer NEPC, 2002) or the Ambient Air Quality NEPM (NEPC, 1998b).

An inventory of emissions of phosphoric acid from facilities with significant emission are included in the National Pollutant Inventory (NPI) NEPM (NEPC, 1998a). NPI data are obtained from registered facilities. In 2000-2001, reportable emissions of phosphoric acid into air were derived from three industry sources in Australia, with a total emission of 3.7 tonnes to air (NPI, 2001). Use of Valvemaster™ in LRP (Scenario 1) is estimated to result in the emission of approximately 182 tonnes per annum of phosphoric acid (57.6 tonnes of phosphorus per annum) Australia wide and 36.4 tonnes phosphoric acid for the Sydney region, but to decline over time as demand for LRP

declines. The decline in emissions of phosphoric acid from vehicles reliant on LRP to 2004 (Scenario 2) is estimated to be 60%.

International air quality management

Several international organisations have introduced regulations or policies that aim to limit the exposure of the general public to air toxics. The Organisation for Economic Cooperation and Development (OECD, 1999) has implemented the Advanced Air Quality Indicators and Reporting Project in OECD member countries, including Australia. The project focuses on six major urban air pollutants. Phosphorus compounds are not among them (Environment Australia, 2001).

In the United States, air quality is managed and regulated under the Clean Air Act (CAA) 1970. The National Air Toxics Program: The Integrated Urban Air Strategy (US EPA 1999) outlines the US EPA's plan for addressing cumulative health risks from 189 identified HAPs, including phosphorus compounds, in urban areas of the United States. Phosphorus compounds in air are not of such concern in the United States as to be listed as one of the 33 priority urban air toxics (Environment Australia, 2001).

In Canada, a range of air toxics is measured and analysed within the National Air Pollution Surveillance (NAPS) Network, which was established in 1969 to monitor and assess the quality of ambient air in Canadian urban areas. Canada does not identify phosphorus compounds as priority air pollutants (Environment Australia, 2001).

Air quality in the United Kingdom (UK) is managed by the Department of Environment, Transport and Regions (UK DETR). The UK also does not identify phosphorus compounds in air as priority air pollutants (Environment Australia, 2001).

In 1994, the New Zealand (NZ) Ministry for the Environment published Ambient Air Quality Guidelines. Guideline values were established for 8 common air pollutants. Phosphorus compounds are not considered a priority air pollutants in New Zealand (NZME, 1994).

15.5.2 Aquatic ecosystem management

Shipping safety and pollution from ships is managed at a national level by the Australian Maritime Safety Authority (AMSA). Australia is a member of the International Maritime Organisation (IMO) and is a signatory to the International Convention for the Prevention of Pollution from Ships 1973/78 (MARPOL 73/78; Commonwealth of Australia, 1988). This convention controls operational pollution and introduces measures to mitigate the effects of marine pollution.

The Commonwealth Department of Transport and Regional Services manages chemical transportation in Australia at a national level. The *Australian Code for the Transport of Dangerous Goods by Road and Rail* and its supplements (ADG code) (FORS, 1998) is designed to apply to all surface land transport in Australia.

Significant facility discharges of phosphoric acid to the Australian aquatic environment are monitored by a National Pollutant Inventory (NEPC, 1998a), with one registered facility discharging approximately 16 tonnes per annum to water.

The Australian water quality guidelines (ANZECC and ARMCANZ, 2000), established under the National Water Quality Management Strategy, provide assessment guidance and water and sediment quality assessment values for freshwater and marine ecosystems throughout Australian states and territories.

There has been considerable investment in the development and implementation of catchment based nutrient reduction strategies across Australia in response to algal management issues (CRCFE, 2000). These strategies involve national, state and territory and local governments, environment protection agencies, catchment management committees, industry and the community. Examples at the national level include the National Water Quality Management Strategy, the Urban Stormwater Initiative (USI), which aims to enhance water quality in the waterways of major coastal cities by improving stormwater management, and the Cleaning Our Waterways Industry Partnership Program (COWIPP), which encourages local government and industry to enhance long-term economic performance with cleaner production techniques that benefit the natural environment.

The NSW Algal Management Strategy has been established to address the immediate management of bloom effects, short- to medium-term management measures (such as biological control methods), and management of bloom causes (such as nutrient control strategies and waterway management). Responses to the occurrence of blooms are coordinated through regional algal coordinating committees. Each committee has prepared an algal contingency plan. Algal bloom management is also being improved by better environmental flows in rivers throughout NSW. In addition, the NSW Marine Algal Biotoxin Group has been established. Sydney Water Corporation and Sydney Catchment Authority each have nutrient pollution reduction programs underway or planned. Similar nutrient reduction programs operate in other states and territories in Australia through government and non-government stakeholders.

As indicated above, algal blooms can be a surrogate indicator of eutrophication or high nutrient load. High nutrient concentrations in fresh waters in NSW have been linked to algal blooms and poor water quality (ANZECC and ARMCANZ, 2000). Conversely, ANZECC and ARMCANZ (2000) provide guidance levels for total phosphorus and other nutrients throughout Australia, with the intention of providing preliminary water quality assessment values for assessing risks of eutrophication and algal blooms.

Quantifying relationships of nutrient load, availability and algal growth is complex and has rarely been achieved in Australia (Harris, 1994; CRCFE, 2000). This is a key data gap for algal management strategies (CRCFE, 2000). Factors affecting the survival and growth of algae do not only include phosphorus concentration, but also nutrient sources (internal and external), transformation and bioavailability of nutrients for uptake by algae, influences of light, temperature and mixing conditions, and processes such as grazing by herbivores. The modelling of any or all of these components is complex, even for a single species or in a single reservoir, and the availability of acceptable data will be critical to the assessment (CRCFE, 2000).

15.5.3 Disposal and waste treatment

Each Australian state and territory provides statutory controls on waste generation and management. In general, non-recyclable materials should be sent to licenced waste disposal contractors in accordance with state and territory requirements. Care should be exercised in disposing of contaminated wastes to avoid pollution of the environment.

15.5.4 Emergency procedures

The availability of an emergency response plan to deal with unexpected releases of the aklyl phosphate additive products, such as large spills, is good practice. All employees should be trained in accident and emergency procedures. All emergency plans and

procedures should be documented fully and made available to all workers. Local emergency services should be consulted on the appropriateness of the emergency procedures developed.

Emergency and first aid measures in place at oil company terminals are generally comprehensive. They include access to safety showers and eye wash stations, staff trained in first-aid and access to a trained emergency response team. Companies do not have emergency plans specific to the alkyl phosphate additive products.

Fire and spill responses for products containing the alkyl phosphate additive are included in MSDSs and are as follows:

Spills

For spills and leaks, wear suitable protective clothing, gloves and eye/face protection. Shut off all ignition sources. Absorb spillage with suitable inert material. Ventilate the area and the wash spill site after material pick-up is complete. Do not allow the spill to enter public sewers and watercourses. Disposal of the spilled material should be in accordance with local, state, territory or national legislation.

Fire response

Wear a chemical protection suit and a positive-pressure breathing apparatus. Use water, alcohol resistant foam, carbon dioxide or a dry agent to extinguish the fire. Do not use water jets. In the advent of an adjacent fire, cool the containers containing the alkyl phosphate additive with water spray.

16. Discussion and Conclusions

Alkyl phosphate additive is used in Australia in lead replacement petrol (LRP) as an anti-valve seat recession (AVSR) additive. As no data is available for the alkyl phosophate chemical because it is manufactured in situ into a kerosene product, the report relates to two alkyl phosphate AVSR additive formulations currently available on the Australian market.

AVSR fuel additives are added to petrol to prevent excessive valve seat wear and consequent recession of automotive engine valves into the engine head. Until its phase out in lead petrol, tetraethyl lead was the most commonly used AVSR additive in Australia.

There are currently four types of AVSR additives marketed in Australia. Methylcyclopentadienyl manganese tricarbonyl (MMT)-, phosphorus- and sodium-based AVSR additives are presently being assessed by NICNAS as Priority Existing Chemicals. A potassium-based AVSR additive has been assessed by NICNAS as a New Chemical. These four AVSR fuel additives are either pre-blended into bulk LRP supplies by oil companies for distribution to service stations or sold as aftermarket products for addition by consumers to unleaded petrol (ULP).

The exposure and risk assessments for the different types of AVSR additive assessed as Priority Existing Chemicals assume a 100% LRP market share for the individual AVSR additive. This is because of the commercial-in-confidence nature of the information provided by companies on the market share for the individual AVSR additives. Moreover, the risk assessments were conducted under two separate scenarios based on generic AVSR additive use patterns. This is because the use of AVSR additives is governed by a declining population of older vehicles requiring these fuel additives.

The first scenario ('present use') assumes a continuation of the current LRP market of 2500 ML per year with 90% of the AVSR additive delivered as bulk LRP by oil companies and 10% as aftermarket products for consumer applications. The second scenario ('2004') assumes a decline of the LRP market to 1000 ML with the AVSR additive delivered completely as an aftermarket product for consumer applications. These two use scenarios are based on motor vehicle statistics and forecasts from the Australia Bureau of Statistics and the Australian Institute of Petroleum. The occupational health and safety, public health and environmental consequences of these volumes and modes of delivery of AVSR additives are considered in this assessment report accordingly.

Bulk alkyl phosphate additive (Valvemaster™ Concentrate) which contains 5.1% phosphorus is manufactured in Australia for the domestic and export market while the alkyl phosphate 10 mL single-use (containing 5.1% phosphorus) and 250 mL multi-treat (containing 2.5% phosphorus) Valvemaster™ aftermarket products are imported. The 'present use' and '2004-use' scenarios equate to approximately 1150 and 460 tonnes of Valvemaster™ Concentrate per annum, respectively. These estimates are based on the typical treatment rate as recommended by the manufacturers for the alkyl phosphate additive in LRP of 600 mg/kg or 30 mg phosphorus per kg fuel.

16.1 Health hazards

Health hazards addressed in this report relate not only to those associated with the alkyl phosphate products but also with phosphoric acid, the postulated combustion by-product.

16.1.1 Alkyl phosphate additive

The scope of the alkyl phosphate additive assessment of toxicity in this report is limited to that of the kerosene-based product DMA-4 (which is synonymous with Valvemaster™ Concentrate).

Animal (mammalian and avian) toxicity studies of DMA-4 indicate the alkyl phosphate additive is not acutely toxic by oral or dermal exposure, although it has some possible irritant effects. The inclusion of kerosene in DMA-4 can not be discounted as a contributing factor to the irritancy effects observed in the animal studies. No repeated dose DMA-4 toxicity data are available. While there are no toxicokinetic data available for the alkyl phosphate additive, exposure has the potential to add to the total body phosphate/phosphorus load.

There are no published case reports, epidemiology or other studies addressing the human health effects of the alkyl phosphate additive or its proprietary products, e.g. DMA-4. There is insufficient toxicology data for classification of the alkyl phosphate additive products in accordance with the NOHSC *Approved Criteria for Classifying Hazardous Substances* (NOHSC, 1999a).

16.1.2 Phosphoric acid

The critical effects from exposure to phosphoric acid relate to its acidic nature and hence corrosive and irritancy effects. The severity of the symptoms for eye, skin or pulmonary irritation depends, however, on the concentration and length of exposure to the acid.

Based on limited animal (mammalian) data, phosphoric acid has low acute oral, dermal and inhalation toxicity. Phosphoric acid does not appear to have been well studied with respect to repeated dose toxicity in animals or humans. There are no adequate reproductive toxicity investigations of phosphoric acid in humans or animals. No information is available on the teratogenicity, neurotoxicity, sensitisation or immunotoxicity of phosphoric acid, nor is there any reliable information on the carcinogenicity potential of phosphoric acid in animals or humans.

The phosphate anion, PO_4^{3-}, which is an ionic form of phosphoric acid, is an essential component of the normal body and skeleton of practically all life forms and is a natural component of diet. At low doses, inorganic phosphate is incorporated into the body's homeostatic mechanisms which maintain the phosphate balance in the body. Assuming an adequate calcium intake, 4.2 g of phosphorus or 12.9 g of phosphate (PO_4^{3-}) equates to the World Health Organization (WHO) calculation of a maximum tolerable daily intake of phosphorus of 70 mg/kg bw for a person weighing 60 kg.

The most relevant route of phosphoric acid exposure due to the use of the alkyl phosphate additive in LRP is inhalation. There is limited human inhalation exposure data for phosphoric acid. No reports have been found in the literature of systemic poisoning following repeated inhalation exposure to phosphoric acid. Weakness, dry cough, chest pain and shortness of breath or dyspnea is reported following an accidental

occupational inhalation exposure to high (unspecified) concentrations of phosphoric acid vapour. Reports of occupational exposure to phosphoric acid are typically complicated by exposure to other chemicals within the workplace.

All available animal (mammalian) inhalation studies relating to phosphoric acid are based on studies which generate smokes based on phosphorus pentoxide. The smokes typically generated in these animal inhalation studies are complex, with the use of phosphorus pentoxide possibly overestimating the inhalation hazard of phosphoric acid due to its strong dehydrating potential in the lungs.

A concentration-dependent bronchiolar fibrosis is reported from two parallel 13-week inhalation studies in rats using an aerosol containing up to approximately 80% phosphoric acids generated from burning 95% red phosphorus and 5% butyl rubber report. The study reports a NOAEL of 50mg/m^3 and LOAEL of 180mg/m^3 for the smoke.

16.2 Environmental hazards and risks

Use of the alkyl phosphate in internal combustion engines and subsequent degradation through combustion indicate that aquatic and terrestrial organisms are unlikely to be exposed to the active component (the alkyl phosphate additive) or proposed exhaust emission, phosphoric acid, at or above levels of concern. A low environmental risk is, therefore, predicted.

The alkyl phosphate formulation DMA-4 (synonymous with Valvemaster™ Concentrate) has a median lethal toxicity (LC50) to fish of 230 mg/L. Based on this limited data, the alkyl phosphate formulation DMA-4 (synonymous with Valvemaster™ Concentrate) has low acute aquatic toxicity. Spill incidents and leaks to water bodies and land should be managed through existing federal, state and territory legislative frameworks and protocols to mitigate adverse effects to the environment. Such accidental incidents may potentially occur during shipment into Australia, bulk transport, handling and storage.

No toxicity data were available on the effects of DMA-4 nor the alkyl phosphate additive chemical on plants. Whilst there are no biodegradation studies of the alkyl phosphate additive, it is not expected to be highly mobile in landfills and is expected to degrade to inorganic phosphate and aliphatic carbon chains which are further degraded by bacteria.

Phosphorus, which is found in the alkyl phosphate additive products, is naturally occurring and ubiquitous in the environment. It is an essential nutrient for plants and animals. Phosphorus pollution, leading to eutrophication and nuisance algal blooms in aquatic ecosystems, is a major environmental issue in Australia and in general, phosphorus reduction in waterways is the current Australian policy. Cumulative effects from an additional anthropogenic source of phosphorus together with existing natural and anthropogenic catchment sources in the Australian environment increases the risk to the aquatic environment of eutrophication and nuisance algal blooms. Nevertheless, the predicted incremental increase in phosphorus in the environment from use of fuels containing the phosphorus-based AVSR additive is unlikely to develop to levels of concern to aquatic environments given the current use and declining future use pattern.

There is the potential for leakage from USTs to occur. Such leakages represent localised, point source discharge. Although a large number of USTs in Australia have been replaced or have had leak detection systems or other measures installed in recent

years due to increased awareness of environmental pollution from these sources, many USTs do not have leak detection systems. Although there is potential for risk to the environment from leakage of fuel (which may or may not contain the alkyl phosphate additive) from USTs, the risk would be site specific.

The findings of this assessment have not identified any significant risk to the environment given the current use pattern of fuels containing the alkyl phosphate AVSR additive.

16.3 Occupational health and safety risks

Manufacture of the alkyl phosphate additive products and blending of bulk LRP are typically enclosed processess and, therefore, exposure is expected to be low. Exposure to the the alkyl phosphate additive is possible during handling of LRP, alkyl phosphate additive aftermarket products and automotive fuel system components. This is expected to be infrequent, minor and of short duration. Mild irritation is possible on contact with fuels or fuel additives containing the alkyl phosphate additive but given the significant dilution of the AVSR additive with petroleum distillates, irritation is likely due to the irritant properties of the petroleum distillates more than the the alkyl phosphate additive itself. Overall, the risks to workers posed by the alkyl phosphate additive during manufacture and during handling of LRP, aftermarket alkyl phosphate additive products and automotive fuel system components contaminated with the alkyl phosphate additive is low. The current identified controls for occupational health and safety are considered to be adequate for the hazard and risk profile for the alkyl phosphate additive.

Exposure to phosphoric acid via inhalation may occur in occupations associated with vehicles using LRP, e.g. service stations, car parks and workshops, and particularly in work spaces which are enclosed or poorly ventilated. A worst-case scenario was considered for phosphoric acid exposure of such workers. Using an estimated tailpipe phosphoric acid emission concentration of 8.3 mg/m^3 for a worker within 2 m of the vehicle tailpipe and an exhaust gas dilution rate of 100, a Margin of Exposure of 135 was derived. This is considered a sufficient Margin of Exposure particulary given that the conservative nature of the estimate assumes the alkyl phosphate additive has 100% of the AVSR additive market. Therefore, the occupational health risks associated with phosphoric acid exposure from the alkyl phosphate additive's combustion are assessed as low.

The inclusion of kerosene and solvent naptha in the alkyl phosphate additive formulations presents health hazards such as irritancy and lung damage (if swallowed).

Two MSDSs and three labels for the alkyl phosphate additive products were assessed qualitatively against the NOHSC MSDS and Labelling Codes. In general, the MSDSs and labels adequately reflected the required information, and relevant hazard warnings, risks and safety phrases. The Valvemaster™ Concentrate MSDS did not identify the recommended use.

16.4 Public health risks

Direct public exposure to the alkyl phosphate additive is likely to occur primarily via the dermal route as a result of spills and splashes of LRP and aftermarket products.

In LRP, the alkyl phosphate additive is not expected to be a skin irritant at present concentrations. The risk of acute health effects for the general public as a result of dermal exposure to the alkyl phosphate additive in LRP is considered low given the likely small quantities of such spills and the low acute dermal toxicity for DMA-4.

While dermal exposure to the aftermarket product may potentially cause irritation, such an effect is confounded by the presence of kerosene which is a known irritant. Overall, the risk of acute dermal effects is considered low given the small amounts of additive to which people are likely to be exposed, low dermal toxicity and the fact that any spill on the skin is unlikely to reside untreated for long periods.

Acute health effects due to the alkyl phosphate additive could occur as a result of accidental ingestion of LRP by adults when siphoning fuel or by a child. The health risk to adults from accidental ingestion of LRP containing the alkyl phosphate additive during siphoning or to children following ingestion of LRP stored inappropriately around the home is considered low, given the low level of the alkyl phosphate additive (600 mg/kg petrol) in LRP.

Ingestion of half the contents of the the 10 mL single use aftermarket product by a 10 kg child equates to approximately half of the recommended daily allowance of phosphorus for a child aged 3 to 4 years. This ingested dose corresponds to 368 mg/kg bw for a 10 kg child compared with the oral ALD (rats) of 11 000-17 000 mg/kg bw for the Valvemaster™ Concentrate. Therefore, the alkyl phosphate aftermaket products are not considered a significant acute health risk for children by virtue of the alkyl phosphate additive. The inclusion of kerosene, particularly in the concentrations present in the multi-treat aftermarket product, represents specific health effects relevant to the kerosene content, i.e. irritancy and lung damage (if swallowed).

Phosphate is an ubiquitous component of the human body and is contained in the normal diet. Thus chronic phosphoric acid exposures (from all sources combined) are unlikely to be significantly changed by the use of the alkyl phosphate additive in LRP. Exposure via food represents by far the greatest proportion of the total human phosphate dose.

A chronic inhalation scenario was considered for phosphoric acid exposure by the public as a result of the use of the alkyl phosphate additive in LRP. The use of the alkyl phosphate additive will increase the phosphoric acid dose received by inhalation above assumed negligible ambient air concentrations of phosphoric acid. By comparing a NOAEL with estimated continuous exposures, a Margin of Exposure of 26 786 and 66 964 is calcualted for the ‘present use’ and ‘2004’ scenarios, respectively. These margins of exposure are considered satisfactory and the public risk to inhaled phosphoric acid as result of the use of the alkyl phosphate additive in LRP is considered low.

16.5 Data gaps

The data gaps identified in this assessment are not regarded as significant impediments to an adequate assessement of the risks associated with the current use of the alkyl phosphate additive in LRP.

There are no data pertaining to the alkyl phosphate additive chemical because it is manufactured in situ and never isolated. While limited, all toxicity data relates to DMA-4 which contains the alkyl phosphate additive in a range of 70-90% (Section 4)

and indicate low dermal and oral mammalian toxicty of DMA-4 and possible irritancy effects.

Similarly, while phosphoric acid does not appear to have been well studied with respect to all toxicity or genotoxicity end points, inorganic phosphate is an essential component of practically all life forms and is a natural component of the diet.

Furthermore, while the studies of phosphoric acid inhalation toxicity are not without weaknesses, they most likely overestimate the adverse pulmonary effects of inhaled phosphoric acid. This is because the inhalation studies are based on phosphorus smokes rather than pre-formed phosphoric acid, with the smokes possibly containing entities other than phosphoric acid, which can likewise affect pulmonary function.

17. Recommendations

This section provides recommendations arising from the Priority Existing Chemical assessment of the alkyl phosphate additive to improve occupational and public health as a results of its use as an AVSR additive.

Whilst there is insufficient toxicology data for classification of the alkyl phosphate additive in accordance with the NOHSC *Approved Criteria for Classifying Hazardous Substances* (NOHSC, 1999a) and OECD *Globally Harmonised System of Classification and Labelling of Chemicals* (OECD, 2002), the data gaps are not regarded as significant impediments to an adequate assessement of the risks associated with the current use of the alkyl phosphate additive in LRP.

In general, the current identified controls for occupational health and safety and public health are considered to be adequate for the hazard and risk profile for the alkyl phosphate additive. One action is recommened.

17.1 Recommendations for importers and manufacture of the alkyl phosphate additive

17.1.1 Hazard communication – MSDS

This assessment finds the MSDSs for the two alkyl phosphate additive products conform well with the requirements of the NOHSC *National Code of Practice for the Preparation of Material Safety Data Sheets* (NOHSC 1994e). It is recommended that the use for which the Valvemaster™ Concentrate is intended be included on the MSDS.

18. Secondary Notification

Under Section 64 of the *Industrial Chemicals (Notification and Assessment) Act 1989*, the secondary notification of a chemical that has been assessed under the Act may be required where an introducer (manufacturer or importer) of a chemical becomes aware of any circumstances that may warrant a reassessment of its hazards and risks. In the case of the alkyl phosphate additive, specific circumstances include:

- use of the alkyl phosphate additive in bulk transport fuels other than LRP and
- additional information has become available to the introducers as to adverse health and or environmental effects of of the alkyl phosphate additive.

The Director (Chemicals Notification and Assessment) must be notified within 28 days of the manufacturer or importer becoming aware of any of the above or other circumstances prescribed under Section 64(2) of the Act.

Appendix 1
Calculation of LRP Volumes for 2004

A. The weekly fill-up rate for vehicles using lead replacement petrol (LRP) was calculated from:

- sales volumes of lead and lead replacement petrol (LRP) during the period from July 2000 to June 2001, which totalled 2937.36 ML (Department of Industry, Science and Resources, 2001) and
- the number of vehicles using leaded petrol as at 31 March 2001 which totalled 2 904 342 (Australian Bureau of Statistics Motor Vehicle Census, 2001)

with the result:

2937.36 x 106 litres/year ÷ 2 904 342 vehicles = 1011 litres/year/vehicle

= 19.4 litres/week/vehicle

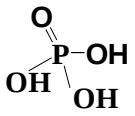
B. LRP volumes in 2004 for 1 000 000 VSR susceptible vehicles were calculated by using a 19.4 litre LRP fill-up rate per week per vehicle, i.e. 1011 litres/year:

1 000 000 vehicles x 1011 litres/year/vehicle = 1 011 000 000 litres/year

~ 1000 ML/year of LRP in 2004

Appendix 2
Physico-chemical Properties of Phosphoric Acid

Table A2.1 - Chemical Identity of phosphoric acid

IUPAC Name	**Phosphoric Acid**
Chemical Name:	**Orthophosphoric acid**
CAS No.:	**7664-38-2**
EINECS No.:	**231-633-2**
Synonyms:	**Phosphoric acid** **Hydrogen phosphate** **o-phosphoric acid** **orthophosphoric acid** **white phosphoric acid**
Molecular Formula:	**H_3PO_4**
Structural Formula:	
Molecular Weight:	**98.00**

Phosphoric acid (orthophosphoric acid) is identified as the major combustion by-product of the alkyl phosphate additive in LRP (Section 8).

The physico-chemical properties of phosphoric acid (H_3PO_4) are well described: (Corbridge, 1985; Commission of the European Communities, 1992; and Environment Canada, 1985). Phosphoric acid is a triphasic oxyacid of pentavalent phosphorus that dissociates in water ($pKa_1 = 2.15$, $pKa_2 = 7.1$, $pKa_3 = 12.4$ at 25°C) to hydrogen and phosphate anion (PO_4^{3-}) as represented in the following scheme (Corbridge, 1985):

$$H_3PO_4 \Leftrightarrow H^+ + H_2PO_4^- \Leftrightarrow 2H^+ + HPO_4^{2-} \Leftrightarrow 3H^+ + PO_4^{3-}$$

Phosphoric acid is odourless (Commission of the European Communities, 1992). Phosphoric acid is stronger than acetic, oxalic and silicic acid but weaker than sulphuric, hydrochloric, nitric and chromic acid (Commission of the European Communities, 1992).

Between pH 2 and 7, most of the phosphate ions in an aqueous phosphoric acid solution will exist as $H_2PO_4^-$ and as HPO_4^{2-} between pH 7 and 12. A 0.1N aqueous solution has a pH value 1.5 and a 1% solution has a pH value of 2.0 to 2.2 (Merck, 1983).

The vapour pressure of aqueous phosphoric acid solutions is highly dependent on temperature and concentration (Health Directorate, 2000; Commission of the European Communities, 1992). For example, the vapour pressure of a 10% aqueous phosphoric acid solution decreases from 2.31 K Pa at 20°C to nil for a 100% solution. (Commission of the European Communities, 1992) The boiling point, freezing point and density of an aqueous phosphoric acid solution depend on the concentration of phosphoric acid (Commission of the European Communities, 1992).

At room temperature, phosphoric acid is likely to occur predominantly in aerosol/droplet form, although vapour may be appreciable at high temperatures (Health Directorate, 2000).

Phosphoric acid has essentially no oxidising properties below 350-400°C. At higher temperatures it is fairly reactive to metal, being reduced by it. (Corbridge, 1985) Phosphoric acid is not explosive or flammable, nor is it very corrosive. Phosphoric acid is described as corrosive to ferrous metals and alloys. Any corrosive behaviour due to phosphoric acid is attributed to impurities, e.g. sulphuric acid, chlorine, and is exacerbated by heat (Becker, 1989).

4.08 mg/m^3 H_3PO_4 = 1 ppm (20°C, 101 kPa) (Health Directorate, 2000)

Appendix 3
MSDS Assessment Summary

Selected Core Information as per Code	Number of MSDS containing correct information	General Comments
INTRODUCTORY AND COMPANY DETAILS		
Date of issue (month/year)	2/2	
Statement of hazardous nature	2/2	
Name of Australian company and address	2/2	
Telephone number	2/2	
Emergency telephone number	2/2	
IDENTIFICATION		
Product name	2/2	
Other name	2/2	
UN Number	1/1	MSDS states UN Number 3082 for Valvemaster™ VM11.
Dangerous goods class	1/1	MSDS states Class 9 Packing group 111 for Valvemaster™ VM11.
HAZCHEM code	NA	MSDS states HAZCHEM code 2X for Valvemaster™ VM11.
Poisons Schedule number	1/1	MSDS states Schedule 5 for Valvemaster™VM11.
Use	1/2	No uses identified for Valvemaster™ Concentrate.
Physical Description/Properties		
Appearance	2/2	
Boiling point/Melting point	2/2	
Vapour pressure	2/2	Vapour pressure of Valvemaster™ Concentrate stated as not known

Specific gravity	2/2	
Flashpoint	2/2	
Flammability limits	2/2	
Solubility in water	2/2	
Ingredients	2/2	
Proportion details	2/2	
HEALTH HAZARD INFORMATION		
Eye irritation	2/2	
Skin irritation	2/2	
Damage to lungs if swallowed	2/2	Both MSDS state "risk of aspiration into the lungs".
First Aid for ingestion, inhalation, skin and eye exposure	2/2	
Advice to doctor	2/2	
PRECAUTIONS FOR USE		
Exposure limits	NA	Both MSDS state no exposure standard is established for the products
Engineering controls	2/2	
Personal protective equipment	2/2	
Flammability information	2/2	MSDS describe both products as C1 combustible liquids
SAFE HANDLING INFORMATION		
Storage and transport	2/2	
Spills and disposal	2/2	
Fire/Explosion hazard	2/2	
CONTACT POINT		
Contact details (Australian)	2/2	

NA = not applicable.

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