NICNAS

National Industrial Chemicals Notification and Assessment Scheme

Sodium Alkylbenzene Sulfonate Anti-Valve Seat Recession Additive

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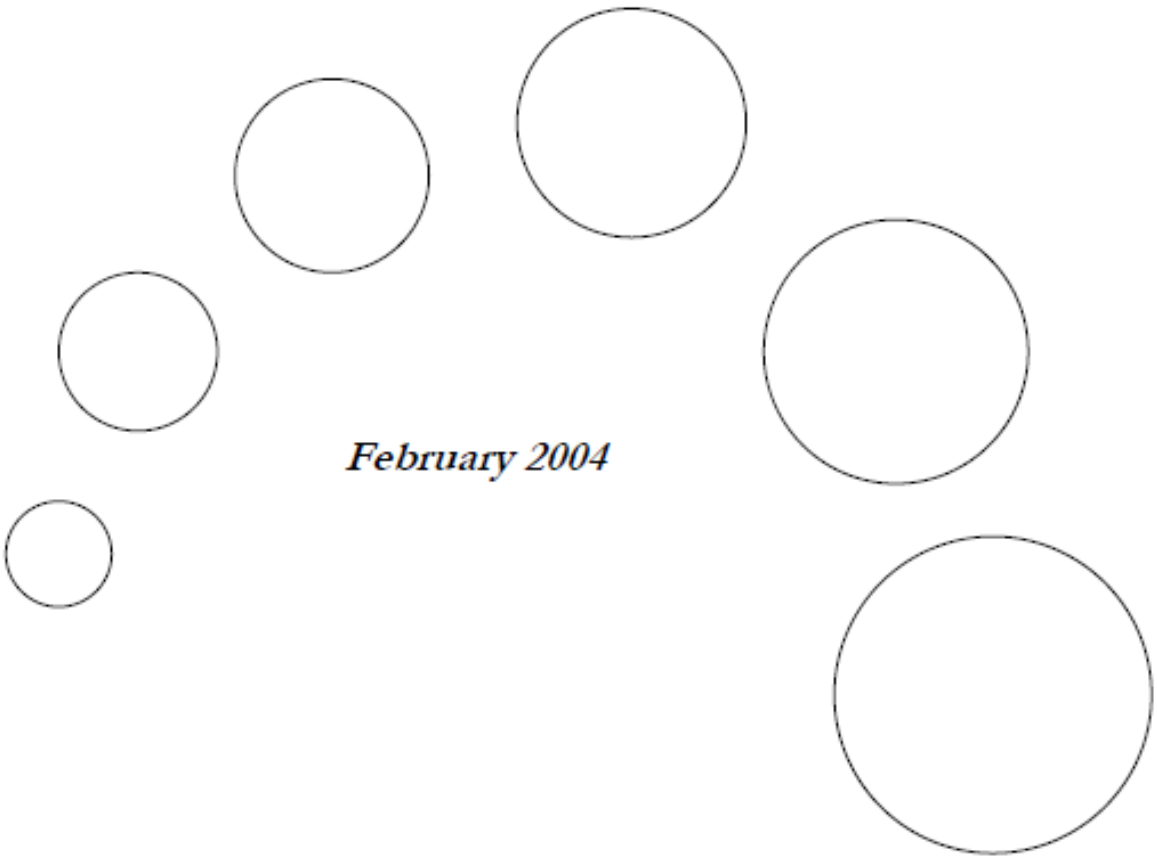
Priority Existing Chemical Assessment Report No. 26



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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 the Department of the Environment and Heritage, which carry out environmental risk assessments.

NICNAS has two major assessment 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 and/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 of NICNAS, 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 web site. Hardcopies are available from NICNAS either by using the order form at the back of this report, or directly from the following address:

GPO Box 58

Sydney

NSW 2001

AUSTRALIA

Tel: 1800 638 528

Fax: +61 (02) 8577 8888

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 by the public because of health and environmental concerns due to their increasing widespread use in automotive lead replacement petrol (LRP). This report concerns the sodium-based AVSR additive which is one of the four AVSR additives notified for assessment: methylcyclopentadienyl manganese tricarbonyl-based, phosphorus-based, sodium-based and potassium-based additives.

AVSR fuel additives are available for both industrial and consumer use and are delivered either by pre-blending to unleaded petrol at the oil refinery (LRP) or purchased and added to unleaded petrol by the vehicle owner (known as aftermarket addition). Sodium alkylbenzene sulfonate (SAS) is a sodium (Na)-based AVSR imported solely for use as an aftermarket fuel additive.

The natural attrition of older cars requiring AVSR additives means a decreasing AVSR market and consequently the general use of AVSR additives is likely to decline with time. The production and infrastructure support of LRP will eventually become economically unviable and aftermarket addition of AVSR additives will be the sole method of providing valve seat protection through fuel. Therefore, the market for aftermarket AVSR fuel additives may increase. This report considered the occupational health and safety, public health and environmental consequences of two separate scenarios for the use of SAS – a Present Use scenario assuming 100% market share and levels of demand and a 2004/2005 scenario assuming attrition of the AVSR vehicle fleet and delivery of SAS via aftermarket addition only.

Literature sources indicate that linear alkylbenzene sulfonates (LAS) have highly variable toxicity depending on carbon chain length, species and test methods employed, and indicate that toxicity increases with increasing chain length. SAS is a LAS with a relatively long carbon chain length; however, toxicity test reports provided by the notifier indicate SAS is not toxic to aquatic organisms up to the limit of water solubility. This suggests that exceeding a certain carbon chain length, LAS bioavailability decreases.

Spill incidents and leaks of SAS to water bodies and land may potentially occur during shipment into Australia, bulk handling and storage. These should be managed through existing Federal, State and Territory legislative frameworks and protocols to mitigate adverse effects to the aquatic environment. Use of SAS as a fuel additive, subsequent degradation through combustion, its diffuse use throughout Australia and generally short persistence in the environment indicate that for existing use patterns, aquatic and terrestrial organisms are unlikely to be exposed to SAS at levels of concern. A low environmental risk is predicted.

The use of SAS as a fuel additive is associated with the generation of combustion products, predominantly sodium sulphate in particulate form. Sodium sulphate is naturally occurring and ubiquitous in the environment, as are the constituent ions. It is an essential nutrient of plants and animals. Environmental exposure to sodium or sulphate compounds arising from combustion of SAS will mostly arise through the gaseous phase. Eventually, these will deposit to land and waters through wet or dry deposition. The emission of sodium sulphate

into the environment from use of LRP containing SAS is unlikely to develop to levels of concern for terrestrial or aquatic environments. The emission of sulphate from SAS combustion is likely to be only a small fraction of that emitted from other sources in Australia. As such, the findings of this assessment have not identified any significant risk to the environment given the considered current use pattern of fuels containing SAS as an AVSR additive.

SAS is currently not listed in the NOHSC *List of Designated Hazardous Substances* (NOHSC 1999b). Animal toxicity studies of SAS and LAS analogues indicate that SAS is not acutely toxic by oral or dermal exposure. Acute irritation studies reveal irritant properties, however data are insufficient for classification of SAS as a hazardous substance (NOHSC 1999a). Repeat dose and reproductive toxicity data are generally lacking. For many toxicological endpoints, extrapolation from analogue data on sodium alkylbenzene sulfonates of shorter alkyl chain length and differing alkali metals were required. There are no published case reports, epidemiology or other studies addressing the human health effects of SAS. Although not meeting the NOHSC *Approved Criteria for Classifying Hazardous Substances* (NOHSC 1999a), a safety phrase Avoid Contact with Skin, or equivalent, is recommended on the basis of possible skin irritation from prolonged or repeated exposure.

The current usage of SAS as a fuel additive marketed to consumers renders the likelihood of occupational use low. Exposure of automotive workers such as automechanics to SAS is possible via dermal and ocular routes through the manual handling of AVSR additive products or fuels supplemented with SAS. However, the low concentration of SAS in additive products and fuels and the limited toxicological profile of SAS indicate that any dermal or ocular irritation is likely to be limited and more likely associated with the irritant properties of petroleum diluents in the products or fuels rather than SAS itself. Therefore, the risk from occupational exposure to SAS is low.

Through its use in AVSR additives marketed to consumers, the public may be similarly exposed to SAS mainly via the dermal route from splashes and spills. However, SAS itself is not expected to be a skin irritant at concentrations present in LRP or in AVSR products. Acute ingestion exposure is possible when siphoning petrol containing SAS or from accidental ingestion by young children if aftermarket products are stored in or around the home. However, under a worst case scenario of ingestion by a child, the oral dose a child would expect to receive is many orders of magnitude lower than the oral LD50 for SAS. Therefore, it can be concluded that there is a low risk of acute health effects for the general public as a result of oral or dermal exposure to SAS in LRP or in aftermarket products.

Sodium sulphate, the main byproduct of SAS combustion, is not a hazardous substance (NOHSC, 1999b) and does not have an associated atmospheric exposure standard (NOHSC, 1995). Modelling reveals expected chronic exposures of workers and the public to only very low amounts of sodium sulphate from the combustion of SAS, many orders of magnitude lower than amounts of sodium sulphate as dissociated Na^+ and SO_4^{2-} ions expected from normal ingestion of water and foodstuffs. A low occupational and public health risk from SAS combustion is expected.

Contents

Abbreviations

ABS	Australian Bureau of Statistics
ACGIH	American Conference of Governmental Industrial Hygienists
ADG Code	Australian Code for the Transport of Dangerous Goods by Road and Rail
AICS	Australian Inventory of Chemical Substances
AMSA	Australian Maritime Safety Authority
ANZECC	Australian and New Zealand Environment and Conservation Council
API	Australian Petroleum Industry
aq	aqueous
ARMCANZ	Agriculture and Resource Management Council of Australia and New Zealand
ATSDR	Agency for Toxic Substances and Disease Registry
AVSR	anti-valve seat recession
bw	body weight
CAA	Clean Air Act
CAS	Chemical Abstracts Service
DNA	deoxyribonucleic acid
DEH	Department of the Environment and Heritage
EC50	median effective concentration
EINECS	European Inventory of Existing Commercial Chemical Substances
FORS	Federal Office of Road Safety
g	gram
h	hour
HAPS	hazardous air pollutants
HQ	hazard quotient
IC25	25th percentile inhibitory concentration
IC50	median inhibitory concentration
IIWL	interim indicative working level
IMO	International Maritime Organisation
ip	intraperitoneal

IPCS	International Programme on Chemical Safety
iv	intravenous
kg	kilogram
K_m	Michaelis constant
L	litre
LAS	linear alkylbenzene sulfonates
LC50	median lethal concentration
LD50	median lethal dose
LL0	no-observed-effect concentration
LL50	median lethal level
LOAEL	lowest-observed-adverse-effect level
LRP	lead replacement petrol
LT50	median lethal time
µg	microgram
µm	micrometre
MATC	maximum acceptable threshold concentration
ML	megalitre
mg	milligram
mL	millilitre
MOE	margin of exposure
MSDS	material Safety Data Sheet
m^3	cubic metre
NAPS	National Air Pollution Surveillance
NDPSC	National Drugs and Poisons Schedule Committee
NEPC	National Environment Protection Council
NEPM	National Environment Protection Measure
ng	nanogram
NHMRC	National Health and Medical Research Council
NICNAS	National Industrial Chemicals Notification and Assessment Scheme
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 Pollution Inventory
OECD	Organisation for Economic Cooperation and Development
PEC	predicted environmental concentration
PNEC	predicted no-effect concentration
PPE	personal protective equipment
ppm	parts per million
RfC	reference concentration
ROS	reactive oxygen species
SAS	sodium alkylbenzene sulfonate
SEM	scanning electron microscopy
SIDS	screening information data set
SOx	oxides of sulphur
SUSDP	Standard for the Uniform Scheduling of Drugs and Poisons
$T_{1/2}$	half-life
TGA	Therapeutic Goods Administration
ThOD	threshold oxygen demand
TLm	median threshold limit
T_{max}	maximum time
TWA	time-weighted average
UKDETR	United Kingdom Department of Environment, Transport and Regions
USEPA	United States Environmental Protection Agency
V_{max}	maximum enzymatic velocity
VSR	valve seat recession
WHO	World Health Organisation

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 increasing widespread use in lead replacement petrol (LRP) and potential adverse effects on the environment and human health.

Applications for the following AVSR additives in use in Australia were received:

- Sodium-based;
- Phosphorus-based;
- Potassium-based; and
- Methylcyclopentadienyl Manganese Tricarbonyl (MMT)-based.

Each AVSR fuel additive has been assessed individually and separate reports are prepared for each. This present report addresses the use of sodium alkylbenzene sulfonate (SAS) (CAS # 78330-12-8) as an AVSR additive.

1.2 Objectives

The objectives of this assessment are to:

- Characterise the chemical and physical properties of sodium alkylbenzene sulfonate;
- Determine the current and potential occupational, public and environmental exposure to sodium alkylbenzene sulfonate as an AVSR additive;
- Characterise the intrinsic capacity of sodium alkylbenzene sulfonate to cause adverse effects on persons or the environment;
- Characterise the risk to humans and the environment resulting from exposure to sodium alkylbenzene sulfonate 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, the report presents a summary and critical evaluation of relevant information relating to the potential health and environmental hazards from exposure to sodium alkylbenzene sulfonate. Relevant scientific data were submitted by the applicant listed in Section 3, obtained from published papers identified in a comprehensive literature search of several online databases up to April 2003, or retrieved from other sources such as the reports and resource documents prepared by overseas regulatory bodies. Due to the availability

of overseas regulatory reviews e.g. International Programme on Chemical Safety (IPCS) Environmental Health Criteria 169: *Linear Alkylbenzene Sulfonates and Related Compounds* (WHO, 1996), not all primary source data were evaluated. However, relevant studies published since the cited reviews were assessed on an individual basis.

The characterisation of health and environmental risks in Australia was based upon information on use patterns, product specifications, occupational exposure and emissions to the environment made available by the importer.

1.4 Peer review

During all stages of preparation, the report has been subject to peer review by NICNAS and Department of the Environment and Heritage (DEH). Expert advice on high temperature combustion products of SAS was obtained from Associate Professor John Mackie of the School of Chemistry, University of Sydney.

2. Background

Sodium alkylbenzene sulfonate (SAS) is an anionic surfactant whose structure is typically characterised by hydrophobic and hydrophilic (polar) functional groups. The chemical is not a single species but a mixture of phenyl positional isomers with different alkyl chain lengths. Recently, SAS has been marketed as an anti-valve seat recession (AVSR) additive for formulation of lead replacement petrol (LRP) by consumers.

2.1 What is an anti-valve seat recession additive?

Anti-valve seat recession 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).

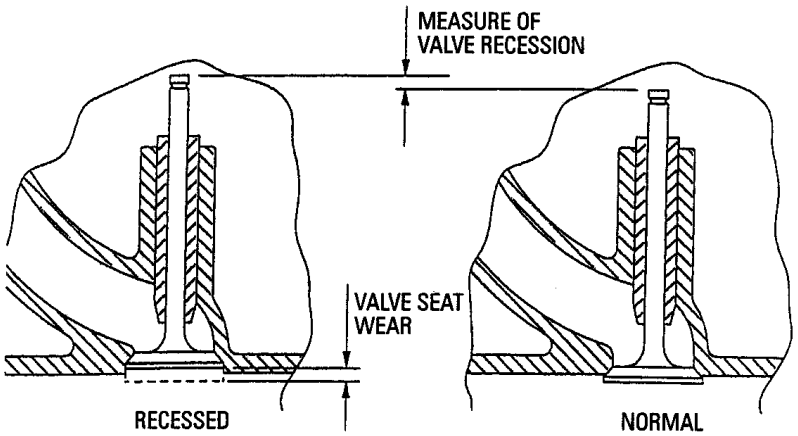


Figure 1. Exhaust valve recession into the cylinder head. From: Barlow (1999)

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

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 result 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 an oxide 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 as lead destroys the capacity of catalytic converters to reduce other pollutants (Lovei, 1998).

A consequence of the reduction of lead in 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 unleaded petrol with no AVSR fuel additive (Lovei, 1998).

The use of AVSR additives has risen with the demand for LRP, 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, environmentally cleaner engines, and improved petrol standards. Consequently, the population of VSR sensitive cars and thus demand for AVSR additives in lead replacement petrol vary from country to country.

2.2 International perspective

In May 1994, the fuel or fuel additive rule (USEPA, 1994b), as mandated in the Clean Air Act (US Congress, 1977), was issued by the USEPA requiring all fuel or fuel additive manufacturers to provide specific mammalian toxicity studies. The marketing of products not registered by the USEPA was prohibited until specific toxicity studies were provided.

Sodium alkylbenzene sulfonate is listed on the USEPA 1990 High Production Volume (HPV) Challenge Programme Chemical List. The US HPV chemicals are those manufactured in or imported into the United States in amounts equal to or greater than one million pounds (453 tonnes) per year. HPV chemicals were identified through information collected under the Toxic Substances Control Act (TSCA) Inventory Update Rule (IUR).

Under the USEPA Chemical Right-To-Know Initiative, basic toxicity information will be provided by Industry on HPV chemicals by 2005. The Industry-sponsored US HPV Chemical Tracking System indicates that SAS and analogues are being sponsored by the American Chemistry Council Health, Environmental and Regulatory Task Group (HERTG) in the HPV Challenge Program with work beginning in 2000. The US HPV Chemical Tracking System also lists a number of analogues of SAS (but not SAS itself) being sponsored by a Soap and Detergent Association LAS/ABS Consortium and the Linear Alkylbenzene (LAB) Sulfonic Acids Coalition.

Under this initiative, Industry has submitted Test Plans with further studies being proposed. To date, incomplete data on SAS or analogues have been provided to the USEPA. As part of the information submitted to the USEPA, HERTG (2001b) indicate that mammalian repeat dose, reproductive/developmental toxicity and acute fish toxicity testing on a SAS analogue (CAS 115733-09-0) as well as water

solubility and biodegradability testing on SAS are planned. However, at the time of writing, these studies were not available.

2.3 Australian perspective

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

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 Assessments by other national or international bodies

Apart from submissions by the American Chemistry Council Health, Environmental and Regulatory Task Group and the LAS/ABS Consortium to the USEPA Chemical Right-To-Know Initiative, no other national or international assessments of SAS are evident.

3. Applicant

Following the declaration of AVSR additives as priority existing chemicals, one company applied for assessment of SAS. The applicant supplied information on the uses of SAS, import quantities, MSDS and labels. In accordance with the *Industrial Chemicals (Notification and Assessment) Act 1989*, NICNAS provided the applicant with a draft copy of the report for comments during the corrections and variation phases of the assessment.

The applicant was as follows:

Morey Oil South Pacific (Australia) Pty Ltd

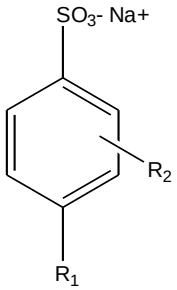
PO Box 1033

Archerfield, Queensland 4108

4. Chemical Identity and Composition

4.1 Chemical identity

Chemical Name (group):	Benzenesulfonic acid, mixed mono- and di-C15-30-alkyl derivatives, sodium salts
CAS No.:	78330-12-8
Chemical Name (generic):	Sodium alkylbenzene sulfonate
Synonyms:	Benzenesulfonic acid, mixed mono- and di-C15-30-alkyl derivatives, sodium salts, benzenesulfonic acid, C15-30-alkyl derivatives, sodium salts
Trade Names:	OS45720G, OS45720S, OS45720L
Molecular Weight:	390-1022
Molecular Formula:	$C_{(21-66)}H_{(35-129)}O_3SNa$
Structural Formula:	



R1 = C15 - C30 alkyl

R2 = H, or C15 - C30 alkyl

4.2 Composition of commercial products

Currently, SAS is being imported to Australia in a single AVSR product marketed as Morey's Upper Cylinder Lubricant and Combustion Chamber Cleaner containing SAS at up to 0.471% v/v.

4.3 Analogues

Based on structural similarity, there are a number of related compounds (analogues) for this chemical.

Sodium alkylbenzene sulfonate is part of the wider alkylbenzene sulfonic acids group of chemicals consisting of a sodium salt sulfonated aromatic ring with attached alkyl chain(s) of various lengths and degrees of branching.

The WHO (1996) document *Linear Alkylbenzene Sulfonates and Related Compounds* reviews the current literature on linear alkylbenzene sulfonates and their salts. In this document, linear alkylbenzene sulfonates (LAS) are defined as mixtures of homologues and phenyl positional isomers each containing a sulfonated aromatic ring with linear alkyl chains attached at any other ring position. These chemicals are anionic surfactants and alkyl carbon chain lengths in applications such as laundry detergents vary typically from C10 to C14 (WHO, 1996).

Because of the lack of a complete data set for SAS, it is necessary to consider analogue data for this assessment, for which the WHO (1996) document is one source. Other sources are test plans and data submitted to the USEPA HPV Challenge Programme (see Section 2.2 International Perspective). Given variations in structure such as in the number and length of alkyl chains and degree of chain branching within the broad chemical groups considered by these sources, not all alkylbenzene sulfonates are considered close analogues to SAS. In addition, few of the analogues are sodium salts. For this assessment, alkylbenzene sulfonates with unbranched alkyl chain lengths of C15 and above are considered the most appropriate analogues.

Table 1 lists typical key data for several close analogues of SAS considered in this assessment. Physicochemical data are from HERTG (2001b).

Table 1: Analogues for Sodium Alkylbenzene Sulfonate (SAS)

Chemical Name	CAS Number	Chain Length	Equivalent Weight[1]	Melting Point (modelled)	Boiling Point (modelled)	Differences to SAS
Benzenesulfonic acid, mixed mono- and di-C15-30-alkyl derivatives, sodium salts (SAS)	78330-12-8	C15-30	390-600	347.25	788.26	none
C15-21 alkaryl sodium salt derivative [2] (Analogue of 78330-12-8)	Unknown	C15-21	390-474	347.25 [2]	788.26 [2]	Shorter chain length
Benzenesulfonic acid, C14-C24 branched and linear alkyl derivatives, calcium salt	115733-09-0	C14-24	393-533	349.84	935.88	Shorter chain length; mix of linear and branched chain; calcium ion
C20-24 alkaryl calcium salt derivative [2] (Analogue of 70024-69-0)	Unknown	C20-24	477-533	349.84 [2]	935.88 [2]	Shorter chain length; calcium ion
Benzenesulfonic acid, mono- and dialkyl derivatives, magnesium salts	71786-47-5	C20-24	461-517	349.84	935.88	Shorter chain length; magnesium ion

[1] Equivalent weight is the weight of one alkylbenzene sulfonate plus the alkali metal. [2] Modelled melting and boiling points are for the parent chemical.

5. Physical and Chemical Properties

5.1 Physical state

Sodium alkylbenzene sulfonate is a viscous liquid composed of 60% active ingredient in 40% mineral oil.

5.2 Physical properties

Table 2. Physical properties of sodium alkylbenzene sulfonate

Property	Value	Reference
Melting point (°C)	347.25 (modelled)	HERTG (2001a)
Boiling point (°C)	788.26 (modelled)	HERTG (2001a)
Density at 15.6 °C	0.935 – 0.965	Confidential
Water solubility	Not determined. The chemical is a surfactant	-
Hydrolysis	Low potential	HERTG (2001a)
Vapour pressure	Not determined	HERTG (2001a)
Henry's law constant	Not determined	-
Partition coefficient (log P_{ow})	Not determined	-
Autoignition temperature	Not determined	-
Flammability Limits	Not determined	-
Flash point (closed cup, °C)	181.7	Confidential

5.3 Chemical properties

The chemical is an aryl sodium sulfonate salt. It does not have oxidising properties and is not expected to have any explosive properties. It is a surfactant and as such will dissociate in water.

6. Methods of Detection and Analysis

6.1 Identification

Infrared spectroscopy was used to characterise SAS. No further identification methodology was provided.

6.2 Atmospheric monitoring methods

None identified.

6.3 Biological monitoring methods

None identified.

6.4 Water monitoring methods

Analytical methods available for determining alkylbenzene sulfonates in water include non-specific methods involving colorimetric, fluorometric, atomic desorption techniques and specific methods involving high-performance liquid chromatography (HPLC), gas chromatography (GC) and GC-mass spectrometry (MS) (WHO, 1996).

The primary method of isolation and analysis for LAS is as methylene blue-active substances.

Colorimetric methods have a common analytical basis i.e. formation of solvent extractable compounds from the anionic surfactant and a colorant cationic dye (commonly methylene blue). Numerous colorimetric methods for anionic surfactants have been established with attempts to improve specificity using separation steps prior to colorimetric extraction. Isolation of LAS from a sample is essential because positive interferences can occur with a number of substances including proteins and amines (WHO, 1996).

6.5 Petrol monitoring methods

None identified.

6.6 Soil monitoring methods

High-resolution GC and GC-MS techniques allow determination of homologues of positional isomers of LAS in both liquid and solid environmental samples (WHO, 1996). HPLC techniques with or without first step trace enrichment of the surfactant have also been described (WHO, 1996).

7. Importation and Use of Sodium Alkylbenzene Sulfonate

7.1 Importation

Sodium alkylbenzene sulfonate is imported only. Neither the manufacture of SAS nor the formulation of products containing SAS occur in Australia.

SAS is imported to Australia in a single product - Morey's Upper Cylinder Lubricant and Combustion Chamber Cleaner. The product is formulated in New Zealand and imported into Australia pre-packaged in 250ml, 1 L and 5 L plastic bottles and 20 L and 50 L steel drums.

7.2 Uses

The product containing SAS is available only as an aftermarket fuel additive for use by consumers. Unlike other AVSR additives, it is not presently available pre-blended in fuel or available at petrol stations as a "forecourt" additive.

The product is added to fuel by consumers via the fuel filler or via a "Power Booster" device which allows automatic metering of the product into the inlet manifold from a reservoir fixed within the automotive engine bay.

The Upper Cylinder Lubricant product contains SAS at up to 0.471% v/v. At a recommended treat rate of fuel of 1 L Upper Cylinder Lubricant for 650 L fuel, the final concentration of SAS in fuel is < 0.001% v/v.

7.2.1 Demand for anti-valve seat recession additives

Anti-valve seat recession fuel additives are available for both oil refinery/terminal and consumer use. AVSR fuel additives may be delivered either by pre-blending to unleaded petrol at the oil refinery or terminal (LRP) or purchased separately and added to unleaded petrol 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/manufacturing quantities and uses of their chemicals for 2000 and 2001. This information was used to estimate a total LRP market for 2001 of approximately 2500 ML, 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 Research, 2001).

The market share of individual AVSR fuel additives in Australia has not been disclosed in this assessment 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 percent of the total LRP market in 2001.

In Australia, vehicles requiring leaded petrol are the major consumers of LRP. These vehicles requiring leaded petrol 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. However, these vehicles and engines are not expected to represent a significant component of the AVSR product market.

There is a declining Australian market for LRP sales (Australian Petroleum Gazette, 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).

By 2004, bulk sales of LRP are expected to decline to less than 5 percent 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. Phase-out by the oil refineries and terminals of the provision of bulk LRP is yet to be announced by the Australian petroleum industry.

Aftermarket addition of AVSR fuel additives rather than bulk treatment by the oil refineries and terminals is likely to eventually become, therefore, 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 technical and practical needs of the program and understanding and acceptance by the public.

7.2.2 Use scenarios

Two use (exposure and emission) scenarios have been assessed in this report – the present state of the market, based on industry information available for 2001, and that likely to occur at 2004/2005. 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 and the consequent increasing use of aftermarket AVSR products and also the attrition from the Australia motor vehicle population of VSR sensitive vehicles. Details of the AVSR additive use-scenarios are presented in Table 3.

Table 3. Summary of the AVSR additive use scenarios

Present Use Scenario
Present AVSR additive LRP market: 2500 ML for 2 500 000 vehicles.
10 % aftermarket: 90 % bulk AVSR additive market.
2004/2005 Scenario
AVSR additive LRP market in 2004/2005: 1000 ML for 1 000 000 vehicles.
100 % aftermarket AVSR additive market.

The Present Use scenario was based upon import and manufacturing data provided by industry for the calendar year 2001. The calculation of 2 500 000 vehicles is based upon 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).

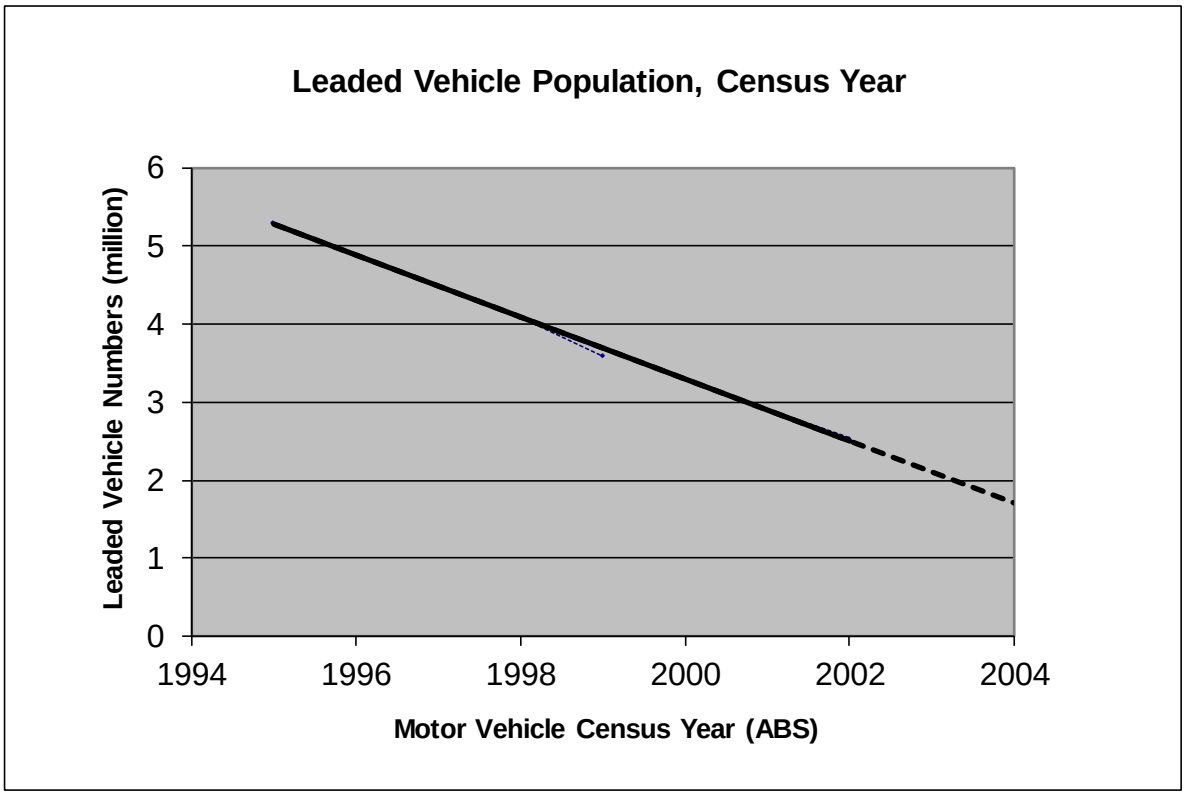
The calculated figure of 2 500 000 vehicles for the Present Use scenario (Table 2) is slightly lower than the ABS Motor Vehicle Census 31 March 2001 of 2 904 342 vehicles. This is attributed to the inclusion in the ABS data of all leaded vehicles irrespective of the requirement for or use of an AVSR 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 calculated figure of 2 500 000 vehicles is, however, similar to the most recent ABS Motor Vehicle Census 31 March 2002 total of 2 543 029 vehicles that require leaded fuel.

The forecast of 1 000 000 vehicles for the 2004/2005 Scenario was extrapolated from Australian Bureau of Statistics motor vehicle census data (Australian Bureau of Statistics, 1998, 2001, 2002). One million VSR susceptible vehicles equates to a demand for LRP of approximately 1000 ML 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 remaining niche market of VSR-sensitive older vehicles and engines requiring leaded petrol is expected (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.

Figure 2. The Number of Vehicles Using Leaded Petrol



(—) 1995-2002

(–·–·) 2002-2004 (Forecast)

(Australian Bureau of Statistics, 1998, 2001, 2002)

7.2.3 Import volumes for SAS

SAS is only available as an aftermarket fuel additive and not preblended in LRP. Therefore, for the Present Scenario, assuming present treat rates, 100% market share and use as an aftermarket additive in 10% of a total LRP market of 2500 ML, a total of up to 400 000 L of product containing approximately 2000 L of SAS is required. However, for the 2004/2005 Scenario, assuming 100% market share in a total LRP market of 1000 ML where aftermarket addition is the only method of providing AVSR protection in fuel, the required import volume increases to approximately 1.54 ML of product containing up to 8000 L SAS.

8. Exposure

The use of SAS as an AVSR agent additive for use in LRP involves relatively small and minor releases of the compound to the environment. In general, these releases are also generally of a very diffuse nature since the motor fuel is used throughout Australia. Further, the compound is susceptible to biodegradation over time and consequently SAS, if released to the environment, is not expected to be persistent.

The product is commercially available only as an aftermarket fuel additive, and is not available pre-blended in bulk fuel at petrol stations. As such, potential issues associated with leaking of underground fuel storage tanks (UST) do not apply and have not been discussed.

Most of the used SAS is destroyed in the cylinders and exhaust trains of motors with production of a variety of typical combustion products (i.e. CO_2, CO, steam H_20, traces of light hydrocarbons, hydrogen and soot) as well as inorganic salts (i.e. Na_2SO_4) and oxides of sulphur (Mackie, 2003; see Appendix 2). It is likely that only a small proportion of this inorganic material is released to the atmosphere from the exhaust systems in association with small particles in the respirable size range (i.e. < 2% of total $PM_{2.5}$; Mackie, 2003; see Appendix 2).

The emissions of exhaust gases such as unburnt hydrocarbons, oxides of sulphur and particulate material are also pertinent to the overall environmental effect of fuel combustion, and available data on the influence of SAS on these exhaust emissions is briefly reviewed.

8.1 Environmental exposure

8.1.1 Release of SAS to the environment

Environmental releases of SAS may potentially arise due to spillage during application by people using the aftermarket product and from disposed containers of the product.

Pre-packaged after-market products are imported in various sized containers including 250 mL, 1 L and 5 L plastic bottles, and 20 L and 60 L steel drums. These are sold through consumer outlets to be added to the fuel in the vehicle tanks by the owners. Releases are expected to occur from spillage and from disposal of remnants of SAS left in emptied containers. No information on this issue was provided, but it is not unreasonable to assume that up to 5% of the formulation could be spilled or be left in the containers after the majority of the contents has been added to the fuel, equating to an annual release of approximately 100-400 L. The emptied bottles are placed into landfill and due to the Australia-wide use of these additives, the associated release of SAS from disposal of the emptied bottles overall will be diffuse and at low levels.

Steel drums containing SAS that are not disposed of to landfill are likely to be cleaned at drum recycling facilities and any residual SAS become incorporated into waste sludge. This is either placed into landfill or incinerated.

The transfer of lubricant in aftermarket products to fuel tanks of consumer vehicles is expected to release a negligible amount of SAS, with most of that released becoming associated with stormwater or soil surrounding the area where the lubricant is applied. This might occur typically at service stations. SAS has a much lower vapour pressure of $< 10^{-7}$ kPa (at 25°C) (HERTG, 2001b) compared with approximately 70 kPa for the hydrocarbon constituents of petrol (Environment Australia, 2000). Consequently, spilled SAS is likely to be left on the concrete aprons of service stations following evaporation of the more volatile fuel components, and it is possible that this residual SAS could be washed from the concrete aprons into stormwater drains or onto surrounding soil. However, most service stations have stormwater collection facilities to capture and manage stormwater pollutants. In the environment, processes of volatilisation, hydrolysis and photolysis are unlikely to have a marked effect on the chemical, and it is likely to be highly mobile in soil and sediment. Biodegradation is the most likely environmental fate process affecting SAS over time. However, in reality, very little is expected to enter the water or soil compartments from these use areas due to the nature of the aftermarket pattern of use and the nationwide and diffuse releases at low concentrations.

Most of the SAS present in the fuel is likely to be destroyed during combustion within the engine (Mackie, 2003).

8.1.2 Exhaust release of compounds from combustion of SAS

According to the consultancy report commissioned by NICNAS (Mackie, 2003; see Appendix 2), the likely reactions and products following combustion of SAS include:

- cleavage of the alkyl side chain(s) from benzene rings; and
- desulfonation of the sulfonate group.

Reactions of radicals present in the engine (e.g. H, OH, and O) with the alkyl side chain(s) will lead to the usual combustion products including CO_2, steam, CO, traces of light hydrocarbons and hydrogen together with some soot.

Combustion of benzene is likely. However, it is less readily oxidisable and some benzene is expected in exhaust gases from LRP. However, the maximum level of benzene that could arise from combustion of SAS in the unlikely event that each aromatic ring formed benzene would be negligible (e.g. < 2 mg/kg fuel) compared to the 20 000 mg/kg of benzene in LRP (Mackie, 2003).

Sulfonate groups ($-SO_3Na$) and free sulfonic acid groups ($-SO_3H$) are expected to be desulfonated from benzene rings during combustion, leading to the formation of molten and solid sodium sulphate (Na_2SO_4) forms. Experimental studies (Halle and Stern, 1980) indicate that molten sodium sulphate is thermally stable to 1100 °C above which temperature it dissociates by the reaction:

$$Na_2SO_{4(l)} \rightarrow 2\ Na_{(s)} + SO_{2(g)} + O_{2(g)}$$

However, the short residence time in the engine means that much of the sulphate should not react. Some will be deposited in the vicinity of the exhaust valve as expansion of the combustion products leads to rapid cooling and the remaining sulphate will be emitted as solid sodium sulphate (Mackie, 2003).

Sodium is the limiting element in determining this level of emission, and about 0.3 to 0.4 ppm sulphur (S) is potentially available for emission, probably as suphur dioxide (SO_2) (Mackie, 2003). However, this level of sulphur is negligible compared to that in LRP.

8.1.3 Emission rate

If all of the sodium were emitted from the tailpipe as $Na_2SO_{4(s)}$, an emission rate of 1.5 mg/kg of LRP used may be expected (Mackie, 2003). With a density of LRP of 0.74 kg/L and an anticipated aftermarket use pattern involving an increase from the current 250 ML of LRP to 1000 ML in 2004/2005, extrapolation of this emission rate indicates an average Australia-wide emission of $Na_2SO_{4(s)}$ of 277.5 kg per annum (current) and 1.11 tonnes per annum in 2004/2005.

For an average fuel consumption of 8 L/100 km, it is estimated there will be approximately 10 m^3 of exhaust gases per L of LRP consumed. As such, the emission of $Na_2SO_{4(s)}$ is, at maximum, 0.1 mg/m^3 at end of tailpipe. Atmospheric dilution will reduce this level.

It should be noted that all these data indicate that sodium sulphate emissions from vehicle exhausts with concomitant exposure potential through particulate inhalation are expected to be significantly higher in areas of high traffic density where the vehicles are undergoing alternate periods of acceleration and braking i.e. typically under conditions of urban driving during business hours.

8.1.4 Effect of SAS on exhaust gases (NOx, CO, CO_2, hydrocarbons, particulates) and onboard pollution control equipment

There have been few studies conducted to evaluate the impact of SAS in fuel on exhaust gases and fuel efficiency. Information provided for assessment includes test data for two studies covering efficacy as an AVSR additive, with some information on fuel economy and emission rates (refer below).

An evaluation of port fuel injector plugging on vehicle emissions and fuel economy indicated that the effect is a function of average injector flow reduction and the maximum difference in flow rates among injectors. Exhaust emissions tended to increase as average flow reduction and the maximum difference between injectors increased. The inclusion of SAS as well as a polybutene amine dispersant chemical in fuel was found to keep all injectors up to 99.5% of original flow, compared to an average of 92% (or 8% flow reduction and 11% maximum difference) for normal gasoline. The stated benefits of the product containing SAS @ 600 ppm as a percentage benefit compared to base fuel include:

- hydrocarbons (HC) 21%
- carbon monoxide (CO) 8.1%
- oxides of nitrogen (NOx) 4.7%
- fuel economy 1.6%

The effect of a fuel additive product containing SAS on the changes in exhaust emissions (HC, CO, NOx) and fuel economy was assessed for four vehicle types, all cars made 1983-85, run for 10 000 km using the fuel additive. No comparison was made to base conditions in cars not using the additive. The report indicates that

results for HC were variable, with HC, CO and NOx emissions better or worse in specific vehicles. Fuel economy was apparently improved after 10 000 km compared to the start of the tests. No raw data were provided and the results above should be qualified in that no indication of the variability and thus significance of the results can be made.

Dynamometer and exhaust analyser emission tests by CNG Installations (1985) using a Mitsubishi L300 van running with a power booster resulted in cleaner burning of fuel, as indicated by a reduction in CO emissions.

Testing of exhaust emissions before and after use of Morey's Oil Stabiliser and Upper Cylinder Lubricant has been undertaken by the Motor Vehicle Museum of NSW (1993). Vehicles tested contained 1% of product in fuel, and the product was used over a two week period. Exhaust gases CO, CO_2, O_2 and fuel consumption from nine motor vehicles were monitored using a Flux 2000 4 Gas Monitor prior to and then after use of the product for two weeks. The results indicate reductions in exhaust emissions of CO (-7.9% to -100%) and O_2 (-2.4% to -100%) with use of Morey's product. CO_2 emissions increased 0.1% to 73.5% in all but one vehicle. Fuel efficiency increased by 8% to 21.1%.

Overall, insufficient information is available to rigorously evaluate the effect of SAS on exhaust gases (NOx, CO, CO_2, hydrocarbons, particulates) and onboard pollution control equipment. Nevertheless, the information provided suggests some benefits or at least no significant adverse effects over base fuel resulting from use of SAS.

8.2 Occurrence in the environment and fate

8.2.1 Geochemical

Unlike the exhaust emission products, SAS probably does not occur naturally in the environment. Limited test data were available to estimate the actual fate of SAS in the environment. Although it is expected that little will be released into the environment from its use as a fuel additive, there are data in the literature addressing the environmental fate of chemical analogues, and these as well as the fate of combustion products are briefly summarised in the following subsections.

Sodium sulphate occurs naturally as the mineral thenardite, a non-marine evaporite sulphate class mineral. Sodium sulphate is readily soluble in water, and consequently the mineral mainly forms in arid regions, dry caves, mines, as a salty precipitate around fumaroles and in playa (salt/clay) lakes. Many other minerals contain sodium and/or sulphate e.g. salt NaCl and the key elements of Na, S and O occur in the environment associated with biogeochemical cycles involving the atmosphere, rocks, soils, sediments, surface and ground waters (USEPA, 1990). Natural sodium deposits are formed by a long geologic process of the erosion of igneous rocks, the transportation of sodium from these rocks and chemical reactions when in contact with sulphur e.g. iron sulphide, volcanic sources, gypsum.

8.2.2 Atmosphere

With a vapour pressure of $< 10^{-7}$ kPa at 25°C (HERTG, 2001b), volatilisation of SAS to the atmosphere is unlikely to be a major migration pathway for the

chemical from spills to soil or waters. This low volatility is typical of most LAS compounds (HERTG, 2001b; HSBD, 2003).

No information was available on the half-life of SAS in the atmosphere. Information from structurally similar chemicals suggests that the chemical may exist in a particulate phase in the atmosphere, and may not photolyse rapidly, but may be removed from the atmosphere to land through wet deposition (HSBD, 2003). The structurally similar compound sodium dodecylbenzene sulfonate is most susceptible to light irradiation of 215 nm wavelength and has an atmospheric half-life of 23.7 hours at an atmospheric concentration of $5x10^5$ hydroxyl radicals/cm^3 (Meylan and Howard, 1993).

8.2.3 Waters and sediments

In general, sulfonic acids and their salts should dissociate almost completely up to the critical micelle concentration resulting in a negatively charged solvated ion (USEPA, 2002b). Like other LAS compounds, SAS in water is unlikely to be volatile or bioaccumulative (HSDB, 2003) and photodegradation is not expected to be a major degradation process (HERTG, 2001b). In general, alkaryl sulfonates are likely to resist hydrolysis because they lack hydrolysable moieties (HERTG, 2001b).

Kaplin et al. (1965) indicate that alkylarene sulfonates degrade extremely slowly in natural waters (30 to 90 days); however, CLER (1996a) highlight a relatively shorter half-life of LAS compounds in freshwater of 0.15 to 0.5 days. As such, if SAS were present in aquatic ecosystems it would be expected to biodegrade over time.

LAS compounds have been detected in sediments in the environment (CLER, 1999; Holysh et al., 1986; Takada and Ishiwatari, 1987, 1991; Prats, et al., 1993; Tabor et al., 1993), suggesting partitioning to sediments and reduced biodegradation in anoxic conditions.

8.2.4 Sewage sludge

Concentrations of LAS in sewage sludge range from 1 to 10 mg/kg (dry weight) (Holmstrup et al., 2001). Under sewage treatment plant conditions, CLER (1996a), Hennes-Morgan and de Oude (1994; cited in ANZECC/ARMCANZ, 2000c) and HSDB (2003) indicate that anionic surfactants are rapidly biodegraded usually with >90% loss, with a large proportion compartmentalised with sludges. However, USEPA (2002b) indicates that, in general, alkaryl sulfonates are poorly biodegraded in activated sewage sludges under laboratory test conditions i.e. <9.1% degradation after 28 days.

The ready biodegradability of benzenesulphonic acid, polypropene derivatives, sodium salt and sodium borate has been investigated by Ricerca (1998). This compound is considered structurally similar to SAS. The test method followed the Organisation for Economic Cooperation and Development's (OECD) test method 301F (Manometer Respiratory Test) (OECD, 1992). Activated sludge was obtained from Mentor wastewater treatment plant. The inocula were adapted to the test substance for 15 days prior to the test. A measured volume of inoculated mineral medium was prepared containing 100 mg/L of the test substance as the nominal sole source of organic carbon. The mixture was continuously stirred in a closed flask at a constant temperature for up to 28 days. The consumption of oxygen was

monitored using an electrolytic respirometer system. The degradability was then expressed as the ratio of measured biological oxygen demand (BOD) and the theoretical oxygen demand (ThOD), which was calculated from elemental analysis of the substance. The test substance was not readily biodegradable with only 18.5% degradation after 28 days duration. The data indicate the potential for biodegradation; however, the rate is slow.

The Danish Environment Protection Agency (Danish EPA, 2001) indicates that the initial step in the biodegradation of LAS under aerobic conditions is ω-oxidation of the terminal methyl group of the alkyl chain to form a carboxylic acid. Further degradation proceeds by a stepwise shortening of the alkyl chain by β-oxidation leaving a short-chain sulfophenyl carboxylic acid. In the presence of molecular oxygen, the aromatic ring structure hydrolyses to form a dihydroxy-benzene structure, which is opened before desulphonation of the formed sulfonated dicarboxylic acid. The final degradation steps have not been investigated in detail but are likely to occur by general bacterial metabolic routes involving a total mineralisation and assimilation into biomass (Steber and Berger, 1995). Both the initial ω-oxidation and the hydroxylation of the ring structure of LAS require molecular oxygen, and they are not expected to take place under anoxic conditions (Steber and Berger, 1995).

8.3 Fate

Limited test data were available to estimate the actual fate of SAS in the environment. Although it is expected that little will be released into the environment from its use as a fuel additive (Section 8.1.1) there are data in the literature addressing the environmental fate of chemical analogues, and these are briefly summarised in the following subsections.

Sodium alkylbenzene sulfonate is a detergent (surfactant) that is structurally similar to compounds within the anionic linear alkylbenzene sulfonate (LAS) group. Sewage treatment plants are major sources of LAS compounds (ANZECC/ARMCANZ, 2000c), which originate from household and industrial use of detergents containing these compounds. Negligible quantities of SAS are expected to be discharged to sewer or enter this waste system due to its AVSR additive use pattern.

8.3.1 Vehicle exhaust system residues

The sodium sulphate residues remaining in the engines and exhaust systems of vehicles would ultimately be placed into landfill with discarded cars and exhaust systems, or if these are recycled for metal recovery, the residues would become associated with slag and other products from the blast furnaces.

8.3.2 Atmosphere

Most of the SAS used as an AVSR additive in fuel within Australia will be combusted and as described above will be converted to sodium sulphate. Most of this is expected to emit with exhaust gases in a solid form, but is unlikely to be predominant in association with fine particles (< 2.5 μm; Mackie, 2003).

Small particles have very low quiescent air sedimentation velocities of around 1-2 cm/hour and less, and are consequently not expected to settle under gravity prior to being precipitated.

For example, the settling velocity of the particles in non-turbulent air can be estimated using Stokes law (CRC, 1976), which gives the settling velocity for a particle of radius r (cm) as:

$$Vset = 2gr2d/9\eta$$

where g is the acceleration of gravity, d is the density of the particle (gm/cm^3) and η is the viscosity of air, which is around 180×10^{-6} gm/cm-sec at 25°C. Taking r as 1.5 μm (= 1.5×10^{-4} cm), and assuming d is 2 g/cm^3, Vset is calculated as 3.75×10^{-4} cm/sec (1.35 cm/h). Consequently, the small particles emitted from the exhaust pipes are expected to remain suspended in the air for prolonged periods.

Atmospheric sodium sulphate is expected to react and dissolve in water to form sodium and sulphate ions, which are likely to react with water to form sulphuric acid. In air, sulphuric acid will react with other chemicals present such as ammonia, magnesium and calcium to form salts, which neutralise the acid. The acid particles dissolve in clouds, fog, rain, or snow, resulting in very dilute acid solutions. This may impact the environment as wet acid deposition ("acid rain"). It should be noted that acid rain is not a significant environmental issue in Australia. In clouds and moist air it will travel along the air currents until it is deposited. In waterways it readily mixes with the water (Environment Australia, 2002; Humphries, 2003).

Rainwater contains variable concentrations of salts depending on the distance from oceans. Rainwater may contain from 6 to 50 mg/L of salts, and sodium and sulphate concentrations may approximate 2.0 and 0.6 mg/L, respectively. Seawater contains a relatively constant concentration of salts, with sodium and sulphate concentrations of approximately 10 800 and 2700 mg/L, respectively (Munns, 2002).

Sulphates are also produced in ambient air through oxidation and dissolution of sulphur dioxide (SO_2) and sulphur trioxide (SO_3) (Humphries, 2003). There are many natural and anthropogenic sources of SOx (SO_2, SO_3, SO_4) including volcanoes and hot springs, sea spray from the ocean, natural decay of vegetation, fuel combustion, coal combustion (eg power stations), and metallurgical roasting facilities (Bast, 1991). Sulphate is found in diesel exhaust emissions (ToxProbe Inc, 2002) and from the decay of ocean biota i.e. dimethylsulphide gas forming sulphate and methanesulphonic acid.

Sulphur dioxide emissions from motor vehicles are directly related to the fuel sulphur level and are released from the exhaust system. Essentially all sulphur present is oxidised to form sulphur dioxide. Although sulphuric acid is not normally found in motor vehicle exhaust emissions, it is present in diesel fuel combustion emissions (ToxProbe Inc., 2002) and it can also arise through reaction of sulphur trioxide with water.

In a study by the United States Environmental Protection Agency, taken from 1980-86 involving 54 000 samples from 381 sites, ambient sulphate levels ranged from 0.2 to ~200 $\mu g/m^3$ (USEPA, 1990). Average ambient air concentrations from these sites ranged from 1.4 to 20 $\mu g/m^3$.

Average wet deposition of sulphate in Australia ranges from ~5 to 29 meq/m^2/annum, which is lower than that measured in several other countries (Table 4).

Table 4. Annual average wet deposition of sulphate

Location	Wet Deposition of Sulphate (milli-equivalents/m^2/year)	Reference
Katherine (NT)	5.4	Ayers and Yeung (1993)
Coffs Harbour (NSW)	14	Ayers and Manton (1991)
Jabiru (NT)	7.4	Gillet et al. (1990)
Wagga Wagga (NSW)	7	NSW EPA (1994)
Latrobe Valley (VIC; 4 sites)	9-14	Ayers (1992)
Hunter Valley (NSW; 8 sites)	15-29	
Northeast USA*	66	NAPAP (1990)
South Sweden	70	Rodhe (1989)
China-Beijing	100	
China-Guijang	400	
Hong Kong	122	Ayers and Yeung (1993)

* 5% of the area of the eastern US had wet deposition greater than this value in 1985-87.

Approximately 20 000 tonnes of SOx were emitted to the Sydney airshed in 1992, 2780 tonnes from motor vehicles (NSW EPA, 1995; refer Table 5). About 86% was derived from sources other than motor vehicles. Motor vehicles, while an important source, are not the main SOx contributor to the Australian environment (Environment Australia, 2003a).

Table 5. Estimated annual emissions of SOx in 1992 (tonnes).

Source	Greater Metropolitan Area*	Sydney
Mobile sources (transport)	5730	2780
Domestic/Commercial Activity	5180	4310
Industrial/Commercial Activity	234 910	12 700
Annual Total (tonnes)	245 820	19 790

* Annual industrial emissions data includes emissions from the Hunter Valley and Western Power stations and a cement manufacturing facility in northwest NSW.

The National Pollutant Inventory for the 2001-2002 period (Environment Australia, 2003a) indicates emissions of SO_4 and SO_2 at a rate of 26 000 and 1.4 Mtpa, respectively, in Australia, with practically all SO_2 emitted to air, of which motor vehicles contributed 9900 tonnes (<1%). Approximately 6500 tonnes (25%) of SO_4 was emitted to air during 2001-2002, with the remaining 75% to waters or land.

Sulphur dioxide emissions in Australia are quite small relative to other western countries, with the main source being industrial activity with materials that contain sulphur. These include power generation from coal, oil and gas. Sulphur dioxide is considered one of six common air pollutants in Australia and measures to reduce emissions have been instigated (Environment Australia, 2003b).

In perspective, SOx generation from use of SAS in LRP represents a small fraction of the SOx already generated from LRP fuel combustion (~0.14%; Mackie, 2003) and other natural and anthropogenic sources (~0.00001%; Environment Australia, 2003a).

8.3.3 Soils

Sulphate in soil is subjected to many transformation processes including adsorption/desorption, immobilisation/mineralisation, oxidation/reduction, and biological assimilation e.g. plant uptake. Loss of sulphate from a catchment may occur through leaching from soils and subsequent export by run-off leaving the catchment (Humphries, 2003).

Sulphate in soils, sediments and waters may be formed following oxidation of hydrogen sulfide by sulphate reducing bacteria (e.g. *Thiobacillus* spp). Biological oxidation occurs in the pH range 8.5 to 1.9, and a succession of species with optimal pH ranges may be involved, which may also reduce the pH of the soil. Elemental sulphur is converted to sulphate by *Thiobacillus* spp, but the rate is temperature dependent, being slower in cooler temperatures.

Weathering of sulfide minerals to sulphates is generally relatively slow in the environment, except where bedrock contains large quantities of sulphides (Mitchell et al., 1998). In a forested watershed, Humphries (2003) estimated that the major contribution of sulphate originated from atmospheric deposition, either wet as precipitation or dry as gases and particulates.

Oxidation of pyrite (FeS) in soils when exposed to air leads to the production of sulphuric acid (H_2SO_4). Such soils are referred to as acid sulphate soils (ASS). Natural buffering capacity of the soil may neutralise some of the sulphuric acid, but that not neutralised may migrate through soil, acidifying soil interstitial (pore) water, groundwater, and eventually surface waters. This phenomenon has received considerable attention in recent years due to its effects on the environment, including fish kills in estuarine areas of Australia, and its exacerbation due to widespread land disturbance (Tulau, 1999; NSW EPA, 1995).

8.3.4 Waters and sediments

Sulphate can occur naturally at concentrations up to thousands of milligrams per litre, particularly in groundwaters. Mine waste waters, tannery wastes and other industrial discharges often contain high concentrations of sulphate, while the use of alum as a flocculent may increase the levels of sulphate in stock drinking water (ANZECC/ARMCANZ, 2000a).

Sources of sulphate to waters include surface flows (streams, rivers, etc), groundwater and atmospheric deposition (dry and wet). The sulphate ion (SO_4^{2-}) is one of the major anions occurring in natural waters (Daniels, 1988, cited in Bast 1991). Most sulphate compounds are soluble with the exception of lead, barium, and strontium sulphates, and dissolved sulphate is considered to be the permanent solute of water. In water, sulphate may be reduced to sulphide, volatilised from water to air as hydrogen sulphide (H_2S), precipitated as an insoluble salt, or incorporated into living organisms (WHO, 1984, cited in Bast, 1991).

Under anoxic conditions bacteria in water can reduce sulphate to sulfide, which results in the release of hydrogen sulfide, causing an unpleasant taste and odour

and increasing the potential for corrosion of pipes and fittings (ANZECC/ARMCANZ, 2000a).

8.4 Environmental concentrations of SAS and sodium sulphate

8.4.1 Sodium alkylbenzene sulfonate

The atmospheric concentration of SAS is expected to be very low due to its low potential to volatilise and the diffuse nature of the releases of the compound. The chemical is unlikely to be persistent in soils due to biodegradation.

Except in the cases of gross spillage of product or petrol containing the chemical e.g. leakage from motor vehicles or pre-mixed storage tanks, very little release to land is likely. Apart from areas in the vicinity of such spills and leaks, no accumulation of SAS is likely in soils and groundwater.

8.4.2 Sodium sulphate in the atmosphere in Australia

No data were obtained on ambient concentrations of sodium sulphate in the Australian atmosphere. The NPI indicates approximately 6500 tonnes of SO_4 were emitted to the Australian atmosphere in the 2001-2002 period (Environment Australia, 2003a). The level of atmospheric Na_2SO_4 resulting from emissions from the combustion of SAS-treated fuel obviously depends on the extent of fuel usage as well as meteorological conditions in the areas where the fuel is used. There are uncertainties associated with both these factors and in order to make some estimates of the likely level of atmospheric Na_2SO_4 resulting from future use of SAS in Australian fuel, it is necessary to make some assumptions based on the following considerations.

All estimates are made for Sydney with a population of 3 800 000, which comprises 20% of the total Australian population (19 million), and covers an area of approximately 1550 square kilometres. Two scenarios (Section 7.2.2) are examined corresponding to:

Present use:

Where the total Australian import volume of SAS is constant at approximately 2000 litres per annum and this is added to petrol for use as an AVSR additive in lead replacement petrol, and

2004/2005:

Where the total Australian import volume is increased to approximately 8000 litres per annum to reflect the expected increased demand for LRP in aftermarket products (and hence for SAS as an AVSR additive).

Since it is reasonable to assume that fuel use reflects population density, it will be assumed that 20% of all petrol in Australia would be used in Sydney.

An atmospheric box model approach has been used to estimate Na_2SO_4 air concentrations in SAS use areas. The same model has been used for other chemicals assessed by NICNAS. 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 Na_2SO_4 accumulation and dispersion of Na_2SO_4 from the atmospheric box.

Two predicted environmental exposure concentrations for Na_2SO_4 in the air have been estimated resulting from the future use of SAS in Australian fuel. These include an average (AVE) estimate and a reasonable maximum exposure (RME) estimate.

The calculation of the AVE air Na_2SO_4 concentration, which represents a long-term average exposure concentration, takes into account the average wind speed across Australian major cities. In order to determine an AVE concentration, daily clearance of accumulated air Na_2SO_4 within the atmospheric box is assumed.

The RME calculation represents the Na_2SO_4 concentration that may potentially accumulate in the air during an extreme weather period of consecutive windless days. The RME estimate represents the highest concentration of Na_2SO_4 reasonably likely to occur in Australian cities; however, 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.

Present use scenario

RME Concentration of Na_2SO_4

Calculation of the RME Concentration of Na_2SO_4 has been summarised below:

Assumed Na_2SO_4 exhaust emission rate	1.5 mg/kg LRP (Mackie, 2003)
Estimated aftermarket share of LRP (10%)	250 ML LRP
Percentage used in Sydney region (20%)	50 ML LRP
Density of LRP	0.74 kg/L
Mass of LRP used in Sydney region	37 000 000 kg LRP
Emission of Na_2SO_4 (annual; Sydney)	5.55×10^7 mg (or 5.55×10^{13} ng)
Emission of Na_2SO_4 (daily; Sydney)	1.52×10^5 mg (or 1.52×10^{11} ng)
Emission of Na_2SO_4 (3 day; Sydney)	4.56×10^5 mg (or 4.65×10^{11} ng)

Combustion of SAS will lead to formation of Na_2SO_4 and assuming that all of the Na_2SO_4 is emitted and none is entrained in vehicle exhaust systems, corresponds to an annual release of approximately 5.55×10^7 mg of Na_2SO_4 into the Sydney atmosphere.

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 5.55×10^7 mg of Na_2SO_4 would be released into an atmospheric volume of 1550 km^2 x 6.15 cubic kilometres, or approximately 10^{13} m^3. However, the assumption that the Na_2SO_4 particles 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).

It is necessary to make some simplifying assumptions, and while these are not entirely realistic they nevertheless allow for a first approximation to the atmospheric Na_2SO_4 level. If it is assumed that the air column is perfectly static, that the particulate matter is homogeneously distributed through the air column volume and that none is precipitated with rain or through other mechanisms, then after one year the atmospheric Na_2SO_4 level is estimated as 5.55 x 10^{13} ng/10^{12} m^3 = 55.5 ng/m^3.

The assumptions made above are considered unrealistic in that no dispersion through wind or by rain is considered. If it is assumed the particles remained suspended for an average of 3 days without removal, as may potentially occur, albeit rarely, following 3 consecutive windless days, then the atmospheric RME concentration could be as high as ~0.5 ng/m^3 (Table 6).

AVE Concentration of Na_2SO_4

An AVE Na_2SO_4 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 5.55 x 10^{13} ng Na_2SO_4/year (1.52 x 10^{11} ng/day) has been estimated above. Emitted into an air volume of 10^{12} m^3 each day, an average daily air concentration of 0.15 ng/m^3 has been estimated using this model.

2004/2005 Scenario

This scenario assumes bulk sales of LRP have declined from 2500 ML to 1000 ML as outlined in the Use Section; however, this is contributed by aftermarket products. The calculations and assumptions for this scenario are identical to the above; however, there is a four-fold increase in use and emissions. Therefore, total annual, RME and AVE concentrations of Na_2SO_4 of ~222 ng/m^3, ~1.8 ng/m^3 and ~0.6 ng/m^3, respectively, have been estimated.

The results of these estimations for AVE and RME concentrations are summarised in Table 6.

Table 6. Estimated average and reasonable maximum atmospheric Na_2SO_4 levels in Sydney – various SAS use scenarios and conditions

	Atmospheric Dispersion [a] (ng/m^3)		
	Nil [b]	AVE [c]	RME [d]
Present Use			
400 L of SAS used as AVSR agent in Sydney fuel.	55.5	0.15	0.46
2004/2005			
1600 L of SAS used as AVSR agent in Sydney fuel	222	0.6	1.8

a. Air column volume of 10^{12} m^3 (i.e. 615 m high x 1550 x 10^6 m^2).
b. No dispersion assumed 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 as to the use rate of SAS and the prevailing atmospheric conditions in particular areas, these estimates of the atmospheric Na_2SO_4 associated with particulate matter originating from exhaust emissions should be treated as indicative only. The instantaneous level of particulate matter in the atmosphere would be very dependent on factors such as rain and wind, and it is likely that ambient and prior weather conditions would impact on any particular daily or hourly measurement.

8.4.3 Release of Na_2SO_4 to the water compartment

If, as in the Present Use scenario above, the use of SAS were restricted to its addition to LRP, then annually approximately 5.55 x 10^{13} ng of Na_2SO_4 would be released into the Sydney atmosphere. Eventually, the material will precipitate to the land or surface. If it is assumed that Sydney with a land area of approximately 1550 km^2 receives an average annual rain fall of 1 metre, then it is possible to estimate the worst case concentration of Na_2SO_4 in storm water, assuming static atmospheric conditions, as 5.55 x 10^7 mg ÷ 1,550 x 10^6 x 1 (cubic metres) = 0.036 mg Na_2SO_4/L (36 μg/L), which, based on molecular weight, would dissolve into SO_4 and Na in a ratio of approximately 2:1 (i.e. 0.024 mg/L of SO_4^{2-} and 0.012 mg/L of Na^+). These are small concentrations when compared to the typical concentrations of sodium and sulphate in seawater of ~11 200 mg/L and ~2650 mg/L, respectively, and the salinity of freshwater of ~680 mg/L (ANZECC/ARMCANZ, 2000a). This estimate does not take into account wind dispersion of Na_2SO_4 from the atmosphere above the urban area, which would reduce the estimated concentration.

8.5 Occupational exposure to SAS

Occupational exposure to SAS is possible during import, transport and handling of petrol additives. Sodium alkylbenzene sulfonate is not formulated in Australia but imported in ready to use fuel additives at a final concentration of up to 0.471% v/v and transported by road and rail to petrol stations and retail outlets.

8.5.1 Importation, storage and retail

Import, transport and retail personnel may be potentially exposed to SAS during handling of imported plastic bottles or steel drums. Containers need not be opened prior to sale and exposure, mainly dermal, is envisaged only in the event of accidental puncture of containers and cleanup.

8.5.2 Petrol stations and maintenance workshops

Mechanics at petrol stations and maintenance workshops may be potentially exposed to diluted SAS in LRP and in fuel additives during maintenance of automotive fuel systems. The extent of potential dermal or ocular exposure during maintenance activities is likely to be highly variable but limited by the low concentration of SAS in the fuel additive products (up to 0.471% v/v). The low expected vapour pressure of SAS precludes significant inhalation exposure.

8.6 Occupational exposure to SAS combustion products

Several classes of workers share occupational environments with operating automobiles and therefore are at potential risk of exposure to products of SAS combustion. These include petrol station attendants, garage mechanics, road and toll booth workers, professional drivers and car park attendants and security personnel.

The main combustion products of SAS are expected to be solid Na_2SO_4 with traces of sulphur dioxide (Mackie,2003). No information is available regarding the size profiles of sodium sulphate particulates that may arise and the proportion in the respirable size range. Overall, given that at expected additive treat rates the sulphur content of SAS is several orders of magnitude lower than the maximum sulphur content of the fuel (Mackie, 2003), the contribution of SAS to sulphurous particulates and gases during combustion of fuel is low.

Assuming that all sodium in SAS is emitted as Na_2SO_4 and with an average fuel consumption (8 L/100 km) producing around 10 m^3 of exhaust gases per litre of fuel, Na_2SO_4 emission from the tailpipe would total around 0.1 mg/m^3 (Mackie, 2003). Atmospheric dilution will significantly reduce this level and the ultimate atmospheric concentrations of particulates in the occupational environment will be highly variable based on automotive use, SAS use in fuel, proximity to operating vehicles and the extent of local ventilation in the work area.

8.6.1 Exposure data and estimates

No exposure data are available for SAS or combustion products.

Accidental dermal and possibly ocular exposure to SAS in petrol is possible for mechanics when servicing fuel systems. The concentration of SAS in fuel is less than 0.001% v/v. Assuming as a worst case, a 70 kg worker spills 200 mL of LRP onto the skin, exposure to a dermal dose of approximately 2 µL (28 nL/kg bw or 27 ng/kg bw) of SAS would occur. Assuming that 100 mL of a fuel additive product containing, for example, 0.471% v/v SAS was spilt onto the skin, then a worker would be exposed to a dermal dose of 471 µL SAS (6.7 µL/kg bw or 6.46 µg/kg bw). Assuming a density of 0.96 kg/L, these equate to 27 ng/kg bw and 6.46 µg/kg bw of SAS for LRP and aftermarket additive spillage respectively. The amounts to

which people will be dermally exposed will be highly variable and normally lower than the above worst case estimates.

Of occupations with potential for exposure to Na_2SO_4 particulates from SAS combustion, auto mechanics may be regarded as a critical occupation when servicing operating automotive engines in poorly ventilated workshops. Assuming as a worst case a fully closed workshop, then the resultant occupational atmospheric level of Na_2SO_4 would reflect the maximum tailpipe emission of 0.1 mg/m^3. Assuming as a worst case, 100% pulmonary deposition and absorption with a typical respiration rate of 20 m^3/day for a 70 kg adult, an 8 hour working day in this environment represents a sodium sulphate dose from SAS combustion of 9.5 μg/kg bw/day.

8.7 Public exposure

8.7.1 Direct consumer exposure

Exposure to SAS is likely to occur as a result of contact during the addition of aftermarket product to fuel tank or contact during the use of LRP as a solvent or cleaner or as a result of substance abuse (petrol sniffing). In the case of deliberate exposure to LRP petrol, the low concentrations of SAS in petrol and expected low vapour pressure limits the extent of exposure to SAS. Since they contain higher concentrations of SAS, exposure to aftermarket fuel additives is likely to be of greater potential concern.

Accidental dermal and possibly ocular exposure to SAS is possible when adding aftermarket products to fuel tanks or when handling fuel containing SAS. The concentration of SAS in fuel is less than 0.001% v/v. Assuming a similar spillage scenario for consumers as for automechanics (200 mL LRP or 100 mL aftermarket additive), a 70 kg consumer would be exposed to a dermal dose of approximately 2 μl SAS (28 nL/kg bw or 27 ng/kg bw) or 471 μL SAS (6.7 μL/kg bw or 6.46 μg/kg bw) respectively.

Accidental ocular exposure as a result of splashes of LRP and/or aftermarket products is also likely to occur only infrequently and involve very small amounts of SAS.

Ingestion exposure is generally unlikely, but, if aftermarket products or fuels are stored inappropriately in or around the home, accidental ingestion might occur in young children. 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). A child (10 kg) ingesting one mL of a product containing 0.471% v/v SAS would receive an oral dose of 4.71 μL (0.47 μL/kg bw or 0.45 μg/kg bw). The aftermarket products are likely to be stored in garages and are not being imported in containers with child resistant closures.

Accidental ingestion of SAS in LRP could occur when syphoning petrol. Australian National Hospital Morbidity Data show approximately 133 hospital discharges/year 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 that 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 siphoned petrol from a car or lawn mower and two had drunk petrol from drink bottles (Ashby and Routely, 1996).

Although no data were available on the amounts of petrol ingested, it is likely that only small amounts of LRP would be accidentally ingested. Data collected by Watson et al. (1983) show 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 SAS in LRP, ingestion would involve potentially only very small amounts of SAS and with the solvent nature of petroleum products, repeated ingestion or ingestion of larger amounts e.g. 100 mL or more is unlikely.

8.7.2 Indirect exposure via environment

As outlined in Section 8.2.2, the atmospheric concentration of SAS is likely to be very low due to low volatility, the diffuse nature of releases and eventual wet deposition of the compound. There are no Australian data on atmospheric concentrations of SAS, and so no estimates of inhalation exposure can be made.

Section 8.2 states that very little release of SAS is expected in the soil compartment of the environment. Therefore, despite a potential for accumulation of SAS in sediments, it is likely that public exposure to SAS as a result of soil contamination is likely to be very low.

Similarly, public exposure to SAS as a result of water contamination is also likely to be very low, since, as outlined in Section 8.2.3, any SAS that does enter the water compartment of the environment would biodegrade over time.

No information is available on the possible contamination of food with SAS. However, public exposure via SAS contaminated food is likely to be very low, since the environmental concentrations of SAS are expected to be very low.

Exposure to Na_2SO_4 via air

Most SAS will be destroyed during combustion in the engine but a proportion of exhaust emissions will contain SAS combustion products in the form of solid sodium sulphate. Using atmospheric Na_2SO_4 concentrations from the most realistic atmospheric dispersion model (Section 8.3.2), an estimate can be made for the potential public inhalation exposure to Na_2SO_4 according to the two use scenarios, firstly where market share is maintained at present levels and use patterns (Present Use scenario) and then when its use is increased in the absence of LRP available at the bowser (2004/2005 scenario).

The estimated atmospheric Na_2SO_4 levels given under the Present Use scenario and 2004/2005 scenario (Section 8.3.2) represent the estimated increase in air Na_2SO_4 concentrations and exposures attributable to SAS combustion when SAS is used as an AVSR agent. As a worst-case, it could be assumed that indoor and outdoor air concentrations of respirable Na_2SO_4 are the same and therefore people will be exposed to ambient air Na_2SO_4 for 24 hours/day. The exposure estimates in Table 7 also assume an average respiration rate of 20 m^3/day for a 70 kg adult and 100% pulmonary deposition. The subsequent calculations of dose assume 100% absorption.

Table 7. Lifetime average estimated human exposure to Na_2SO_4 in ambient air

Scenario	Average Ambient Air Concentration (Na_2SO_4 ng/m^3)	Human Exposure (ng/day)	Human Dose (ng/kg bw/day)
Baseline ($PM_{2.5}$ Sydney)	No data available	No data available	No data available
Increase due to SAS – Present Use: Maintained LRP Market Share	0.15	3.0	0.04
Increase due to SAS – 2004/2005: Increased Market Share	0.6	12	0.17

It should also be noted that ambient air concentrations might not always reflect the actual exposure of individuals living in a given area, because typical human activity patterns result in time spent in microenvironments with higher or lower concentrations of a pollutant and for which there is generally no monitoring data. Hence, a measure of personal exposure to a compound is preferable to ambient air data, and that estimate should be representative of the population of interest throughout the time period of interest.

Assuming complete ionic dissociation in body fluids and based on molecular weight, Na_2SO_4 will dissociate into Na^+ and SO_4^{2-} in a ratio of approximately 1:2. Therefore, a long term human dose of 0.17 ng/kg bw/day Na_2SO_4 from ambient air represents a very low daily dose of Na^+ and SO_4^{2-} of 57 pg/kg bw/day and 113 pg/kg bw/day respectively.

Exposure to Na_2SO_4 via food

It is conceivable that Na_2SO_4 levels in foodstuff may be increased as a result of environmental contamination with the combustion products of SAS. There are no Australian studies on the possible contribution of SAS combustion product to food Na_2SO_4. Given the expected low soil, water and atmospheric levels of SAS combustion products (especially in rural areas), it is considered that the contribution of SAS combustion products to Na_2SO_4 intake from foodstuffs is likely to be very low.

Exposure to Na_2SO_4, Na^+ and SO_4^{2-} via water

The concentration of Na_2SO_4 in stormwater as a result of SAS combustion under quiescent conditions is estimated at 36 μg/L (Section 8.4.3). Based on molecular weight, Na_2SO_4 in solution will dissociate into Na^+ and SO_4^{2-} in a ratio of approximately 1:2 i.e. 12 μg/L of Na^+ and 24 μg/L of SO_4^{2-}.

Assuming that a person drinks up to 2 L/day, intake from drinking water can produce daily intakes of Na_2SO_4 of 72 μg/L (24 μg Na^+ and 48 μg of SO_4^{2-}). Assuming that all is absorbed from the gastrointestinal tract, the systemic doses can be estimated at 1.03 μg/kg/bw day for Na_2SO_4, 343 ng/kg bw/day for Na^+ and 686 ng/kg bw/day for SO_4^{2-} for a 70 kg adult.

For Australian reticulated water supplies, guidance values for Na^+ and SO_4^{2-} are 180 mg/L and 250 mg/L (NHMRC, 1996). These values are based on aesthetic considerations – taste. In Australian water, concentrations vary up to 300 mg/L and 240 mg/L respectively (NHMRC, 1996). The NHMRC recommended daily intakes of Na^+ for adults is 920-2300 mg (13-33 mg/kg bw/day). Currently, there is no NHMRC recommended daily intake for SO_4^{2-} (NHMRC, 1991). The US National Academies, Institute of Medicine is presently conducting a project to establish dietary reference intakes for electrolytes including SO_4^{2-}. This has yet to be completed (National Academies, 2003).

Given the expected low water concentrations of SAS combustion products (especially in water catchment areas), the potential contribution of SAS combustion products to Na_2SO_4, Na^+ or SO_4^{2-} intake from water is very low.

9. Kinetics and Metabolism

No kinetics and metabolism data are available for SAS. Also, no data are available for long chain linear alkylbenzene sulfonates (LAS). Information on short chain LAS are considered here (WHO, 1996). These data are relevant as long chain LAS are likely to be oxidised to shorter chain length species in vivo (Michael, 1968).

It is likely that SAS, being a long chain LAS, will be absorbed generally to a lesser extent than shorter chain LAS.

9.1 Absorption, distribution and excretion

WHO (1996) reports that in experimental animals, LAS are readily absorbed via the gastrointestinal tract. In Wistar rats following oral administration, radiolabelled calcium and sodium salts of LAS with chain length of C12 were detected in plasma after 0.25 hours, with levels reaching a maximum at 2 hours. Biological half-lives were calculated at 10.9 and 10.8 hours respectively. Excretion occurred equally via urine and faeces (Sunakawa et al, 1979, as cited in WHO, 1996).

A rat study of LAS isomers of similar chain length (C12) given orally or intravenously showed differential excretion in urine and faeces depending on the position of sulfonate moieties on the benzene ring (position 2 or 6). For either route of administration, after 48 hours around 75% of position 2 isomer was found in the urine, whereas 78% of the position 6 isomer was found in the faeces. In bile duct cannulated rats following intravenous administration, 89% of the 2 isomer was recovered in the urine whilst 83% of the 6 isomer was found in the bile (Rennison et al, 1987, as cited in WHO, 1996).

In an earlier series of rat studies of radiolabelled LAS of alkyl chain lengths C10-14, 40-58% was excreted in urine and 39-56% in faeces within 72 hours of oral (gavage) administration. Orally administered radiolabelled LAS in bile duct cannulated rats showed 46% recovered in urine, 29% in faeces and 25% in bile after 90 hours. Lymph levels (< 2%) in thoracic duct cannulated rats indicated little absorption of LAS via the lymphatic system (Michael, 1968).

In contrast, dermally applied LAS of chain length C10-14 are not well absorbed. Of radiolabelled LAS applied to dorsal skin of rats for 15 minutes, none was detected in urine or faeces 24 hours after application. An accompanying in vitro study showed no measurable penetration of LAS through isolated human epidermis or rat skin 24 or 48 hours after application (Howes, 1975, as cited in WHO (1996). Similarly, of radiolabelled LAS applied in white petrolatum to the dorsal skin of guinea-pigs, only 0.1% was found in urine and 0.01% in blood and main organs after 24 hours (Hasegawa and Sato, 1978, as cited in WHO, 1996).

Three metabolism studies in rhesus monkeys by Cresswell et al. (1978) were reported in WHO (1996). After a single oral administration of radiolabelled LAS with a mean molecular weight of 349 (150 mg/kg bw), plasma concentrations peaked at 41.2 μg/mL at 4 hours. Concentrations declined during the period 6-24 hours with a biological half-life reported to be approximately 6.5 hours. Concentrations were below the limit of detection at 48 hours.

Similar results for peak plasma levels and half-life were found following 7 consecutive daily oral doses of LAS at 30 mg/kg bw. In the monkeys, the highest concentration of radiolabelled LAS 2 hours after the last dose was found in the stomach (239 μg/g). At this time, high concentrations were found also in the intestinal tract, kidney and liver. Moderate levels were seen in the lungs, pancreas, adrenal and pituitary glands. At 24 hours, high levels seen in the intestinal tract (256 μg/g) and liver were the only levels that exceeded that of plasma. Levels in other tissues lower than plasma indicated no specific tissue-specific accumulation or localisation of LAS and/or metabolites (Cresswell et al, 1978).

After 7 subcutaneous doses of radiolabelled LAS (1 mg/kg bw/day) highest levels were seen at injection sites (114 μg/g) after 2 hours. At this time, levels between 1 and 2.45 μg/g were seen in the lungs, intestinal tract, spleen, kidney, thyroid and pituitary gland. At 4 hours, tissue levels were generally lower than at 2 hours except for the intestinal tract, kidney and liver, these latter two organs associated with biotransformation and excretion. Levels in the intestinal tract were attributable to biliary excretion. At 24 hours, skin injection sites were the only tissue sites for which levels exceeded that of plasma (Cresswell et al, 1978).

A study of the excretion of single oral or subcutaneous doses of radiolabelled LAS in the rhesus monkey showed almost all but approximately 6% of the oral LAS and around 25% of the subcutaneous LAS excreted via urine or faeces within 120 hours. In both instances, excretion occurred predominantly (up to 74%) via the urine (Cresswell et al, 1978).

9.2 Metabolism

According to Michael (1968), the main metabolites from an orally administered radiolabelled mixture of C10-14 LAS in rats were sulfophenyl butanoic acid and sulfophenyl pentanoic acid, formed via ω-oxidation and ß-oxidation of the parent LAS molecules. From thin layer chromatography, these two molecules were also claimed to be the two metabolites found in urine and two of the four metabolites found in faeces in a subsequent test of oral administration of radiolabelled calcium or sodium salts of LAS in rats (Sunakawa et al, 1979, as cited in WHO, 1996).

In rhesus monkeys, thin layer chromatography of urine samples following oral (30 mg/kg bw) or subcutaneous (1 mg/kg bw) administration of radiolabelled LAS showed 5 metabolites (unidentified) and only trace quantities of the original parent compound (Cresswell et al, 1978).

10. Toxicity of Sodium Alkylbenzene Sulfonate

This section deals with toxicity of SAS. The use of SAS as a fuel additive is associated with the generation of combustion products, predominantly sodium sulphate in particulate form. Sodium sulphate toxicity is not covered in detail in this present report. Sodium sulphate is not a hazardous substance (NOHSC, 1999b) and does not have an associated atmospheric exposure standard (NOHSC, 1995). Moreover, modelling reveals the expected release of only very low amounts of sodium sulphate from the combustion of SAS, orders of magnitude lower than amounts of sodium sulphate as dissociated Na^+ and SO_4^{2-} ions likely to be encountered from normal ingestion of water and foodstuffs.

10.1 Available data

Limited toxicological data, in the form of an acute oral toxicity study, and dermal and ocular irritation studies, are available for SAS. However, information on SAS analogues in the form of robust summaries meeting the requirements of the OECD Screening Information Data Set (SIDS) programme was available for this assessment. These were submitted by consortia in response to the United States Environmental Protection Agency (USEPA) High Production Volume (HPV) Challenge Program (HERTG, 2001a,b).

Table 8 contains the available toxicological studies for SAS and analogue chemicals of close structural similarity. Analogues in submissions to the USEPA HPV Challenge Programme are generally of shorter alkyl chain lengths compared to SAS. Data for those analogues with alkyl chain lengths most closely resembling SAS are considered here.

As part of the information submitted to the USEPA, HERTG (2001b) indicate that testing for repeat dose and reproductive/developmental toxicity (as well as acute fish toxicity) on a SAS analogue (CAS 115733-09-0) and water solubility and biodegradability testing on SAS are planned (see Section 17 – Secondary Notification). However, at the time of writing, results from these tests were not available.

For the robust summaries, numerical summary data were rarely provided and the sources of study data for unpublished references were not well documented. Also, for many of the summarised studies, information on test material composition, purity and diluent was lacking.

Table 8. Available mammalian toxicological studies for sodium alkylbenzene sulfonate and structurally similar analogues

Chemical Name	CAS Number	Acute Toxicity	Irritation/ Corrosion	Sensitisation	Repeat Dose Toxicity	Reproductive/ Developmental	Genotoxicity	Carcinogenicity	Reference
Benzenesulfonic acid, mixed mono- and di-C15-30-alkyl derivatives, sodium salts (SAS)	78330-12-8	Oral	Skin Eye	No data	No data	No data	No data	No data	Biosearch Incorporated (1982a,b,c)
C15-21 alkaryl sodium salt derivative (Analogue of 78330-12-8)	Unknown[1]	Oral	No data	No data	No data	No data	OECD 471 test guidelines; in vitro	No data	HERTG (2001a)
Benzenesulfonic acid, C14-C24 branched and linear alkyl derivatives, calcium salt	115733-09-0	Oral Dermal	No data	No data	No data	No data	No data	No data	HERTG (2001a)
C20-24 alkaryl calcium salt derivative (Analogue of 70024-69-0)	Unknown[2]	Oral Dermal	No data	No data	Oral	No data	OECD 474 test guidelines; in vivo	No data	HERTG (2001a)
Benzenesulfonic acid, mono- and dialkyl derivatives, magnesium salts	71786-47-5	No data	No data	No data	Dermal	No data	OECD 473 test guidelines; in vitro	No data	HERTG (2001a)

[1] This chemical is identified in the HERTG (2001a) submission as an analogue of SAS - CAS 78330-12-8, Benzenesulfonic acid, mono- and di-C15-30-alkyl derivatives, sodium salts. This chemical is referred to in HERTG (2001a) as a C15-21 alkaryl sodium salt derivative.

[2] This chemical is identified in the HERTG (2001a) submission as an analogue of CAS 70024-69-0, Benzenesulfonic acid, mono- C16-C24 alkyl derivatives, calcium salts. This chemical is referred to in HERTG (2001a) as a C20-24 alkaryl calcium salt derivative .

10.1.1 Acute oral toxicity

In the acute oral (gavage) toxicity study on SAS, ten (5 male and 5 female) albino Sprague-Dawley rats (age unknown) weighing between 200 and 300 g were dosed with 5.0 g/kg bw of SAS. No data on controls were reported.

Observations were made frequently and individual weights were recorded on day 7 and 14 (study termination). Gross autopsies were performed on all animals at death.

Three hours post dosing, animals were reported to be 'ruffled' but appeared normal within 24 hours. Bodyweight increases were noted for both males and females throughout the study. No mortalities occurred. Gross pathological examination results were reported as unremarkable. No clinical signs of toxicity were reported. An LD50 of >5.0 g/kg bw was reported (Biosearch Incorporated, 1982a).

Robust summaries for three additional rat oral (gavage) studies on analogues of SAS apparently conducted according to the test guidelines OECD 401 were available (HERTG, 2001a). The analogues were C15-21 alkaryl sodium salt derivative (claimed in the robust summary as an analogue of SAS, CAS 78330-12-8) (CAS unknown), C20-24 alkaryl calcium salt derivative (claimed in the robust summary as an analogue of benzenesulfonic acid, mono-C20-C24 alkyl derivatives, calcium salts, CAS 70024-69-0) (CAS unknown) and benzenesulfonic acid, C14-C24-branched and linear alkyl derivatives, calcium salts (CAS 115733-09-0).

All three studies examined ten animals (5 male and 5 females) with a single dose of 5.0 g/kg bw of test substance. The studies reported minor clinical changes such as ruffled appearance, slight diarrhoea, slight reduced food intake, anal stains and slight bloody nasal discharge. No deaths were reported. All three studies reported an LD50 > 5.0 g/kg bw.

10.1.2 Inhalation toxicity

No acute inhalation toxicity studies on SAS or structurally similar analogues were available.

10.1.3 Dermal toxicity

Acute dermal toxicity studies were not available for SAS. However, two acute dermal toxicity studies were available on analogues (HERTG, 2001a).

The first study tested rabbits with benzenesulfonic acid, C14-C24 branched and linear alkyl derivatives, calcium salt (CAS 115733-09-0). Robust summary data indicate that this study was conducted prior to development of OECD 402 test guidelines. The reviewer of the study considered deviations from test guidelines with respect to the use of abraded skin not sufficient to change the study outcome.

Ten animals, 5 per sex, were dosed with 5.0 g/kg bw of test chemical held in contact with the skin for 24 hours after which time residual test material was removed. At 24 hours post treatment, skin at the treatment site was red, swollen and stained. Irritation subsided by day 9. However, the skin remained dry, flaky and stained throughout the observation period up to the conclusion of the study at day 14. No systemic signs of toxicity were recorded. During necropsy, 9 rabbits exhibited alopecia, matted fur and flaky skin at or around the test site. One animal

had a friable, white, mottled left front liver lobe and one animal had a small right testis. The LD50 for this study was established > 5.0 g/kg bw.

The second dermal study tested a C20-24 alkaryl calcium salt derivative (CAS number unknown) which is claimed in the robust summary to be an analogue of benzenesulfonic acid, mono-C20-C24 alkyl derivatives, calcium salts (CAS 70024-69-0). The test was apparently conducted according to OECD 402 test guidelines. Ten rats, 5 per sex were given a topical application of 2 g/kg test material that was kept in contact with the skin for 24 hours, then removed. Animals were observed for abnormal clinical signs on the day of dosing and daily thereafter. Individual body weights were recorded regularly throughout the study. Gross necropsies were performed on all animals on day 14.

No evidence of systemic toxicity was observed. All treated animals exhibited skin irritation. Significant differences in mean body weights were observed repeatedly in male animals. At necropsy, multiple pinpoint scabs were observed in three treated males and one treated female. The LD50 for this dermal study was established at > 2.0 g/kg bw.

10.2 Irritation and corrosion

Skin and eye irritation studies were conducted for SAS. No additional skin or eye irritation studies for structurally similar analogues were identified.

10.2.1 Skin irritation

Six New Zealand White rabbits were clipped over a wide area of their backs. On one side, the skin was abraded sufficiently deeply to penetrate the stratum corneum but not the derma. A dose of 0.5 mL SAS was applied to abraded and intact sites on each rabbit and then covered with gauze patches held in place with impervious wrapping for 24 hours. This test differs from OECD 404 test guidelines in that skin was exposed for 24 hours instead of the recommended 4 hours.

After removal of wrappings, the treated areas were examined and scored 24 and 72 hours post commencement of treatment.

At 24 hours, all 6 rabbits showed well-defined erythema (Draize score 2) and slight oedema formation (Draize score 2) at both abraded and intact skin sites. Erythema reactions were increased to moderate/severe (Draize score 3) while oedema reactions were decreased to very slight oedema (Draize score 1) in all animals at both sites 72 hours post treatment.

Sodium alkylbenzene sulfonate was irritating to the skin of albino rabbits (Biosearch Incorporated, 1982c).

10.2.2 Eye irritation

Sodium alkylbenzene sulfonate was tested in six adult albino rabbits. In each animal, a dose of 0.1 mL test material was instilled into the right eye while the left remained untreated. Eyes remained unwashed.

Treated eyes were examined at days 1, 2, 3, 4 and 7 post treatment. At day 1, 4 out of the 6 animals exhibited definite conjunctival redness (Draize score 2). However

all blood vessels returned to normal by day 3 (Draize score 0). No reactions were noted in the cornea and iris at any time.

Sodium alkylbenzene sulfonate was mildly irritating to the eyes of rabbits (Biosearch Incorporated, 1982b).

10.3 Sensitisation

No studies on the skin or respiratory sensitising potential of SAS or structurally similar analogues were identified during the assessment.

10.4 Repeated dose toxicity

No repeat dose toxicity studies were available for SAS. However robust summaries for the following oral and dermal repeat dose toxicity studies for SAS analogues were available (HERTG, 2001a):

- One rat oral (gavage) 28 day study on a C20-24 alkaryl calcium salt derivative (analogue of benzenesulfonic acid, mono- C16-C24 alkyl derivatives, calcium salts, CAS 70024-69-0);
- Two 28 day dermal studies (rat and rabbit) on benzenesulfonic acid, mono- and dialkyl derivatives, magnesium salt (CAS 71786-47-5)

10.4.1 Oral studies

The SAS analogue C20-24 alkaryl calcium salt derivative (CAS unknown) was tested in a 28-day repeated dose rat oral (gavage) study according to OECD 407 test guidelines. Sprague-Dawley CD rats were dosed daily with a single 2 mL/kg bw oral administration of test material in peanut oil at 100, 500 and 1000 mg/kg bw/day for 7 days/week. Controls (number unspecified) were dosed with peanut oil alone.

Animals were observed daily during treatment and in a 14-day recovery period for the control and high-dose animals. Body weights and food consumption were recorded during treatment and recovery. Haematology, clinical chemistry and urinalysis parameters were evaluated at termination of treatment and recovery. Macroscopic examinations were performed on all animals and a range of tissues were examined microscopically.

A pilot 2-week repeated dose range finding study was also conducted. According to the robust summary, two deviations from OECD 407 test guidelines were noted for the main study. Firstly a function observational battery for neurotoxicity was not performed. This was not part of OECD 407 at the time the study was performed. Secondly, microscopic pathology was performed as required by the test guideline at the time the study was conducted. This is not part of the current OECD 407 test guideline.

No deaths related to the test substance were observed. Mean serum cholesterol levels were significantly reduced in the 1000 mg/kg bw/day males and females at termination of dosing and in the 1000 mg/kg bw/day females at the end of the 14-day recovery period. No treatment related effects were observed with respect to clinical state, body weight and body weight gain, food consumption, feed

efficiency, haematology, urinalysis, absolute and relative organ weights and macroscopic or microscopic pathology.

Statistically significant differences from control were observed for some haematology and clinical chemistry parameters. However, these values were within normal range limits, were not associated with histopathological changes and were not considered biologically significant.

Based on a reduction in mean cholesterol values in high dose males and females, a NOAEL was established at 500 mg/kg bw/day (HERTG, 2001a).

10.4.2 Dermal studies

Robust summaries were available for two 28-day dermal repeat dose studies on benzenesulfonic acid, mono- and dialkyl derivatives, magnesium salts (CAS 71786-47-5), conducted according to OECD 410 test guidelines.

In the first study, conducted in 1981, 2 mL/kg/day of test substance in Primol 205 vehicle at concentrations of 0, 25 and 100% (w/v) was applied topically to the clipped, unabraded dorsal skin of male and female adult New Zealand white rabbits. Fifteen rabbits/sex/group were treated for 6 hours/day, 5 days/week for 28 days. Elizabethan collars rather than gauze patches under wrappings were used to prevent ingestion. After each 6 hour dosing, test material residue was removed from the skin with a paper towel. Five animals from each group were retained for an additional 4 weeks for recovery observations.

According to the robust summary, the dosing regime differed slightly from OECD 410 test guidelines as only 2 doses were used instead of the required three doses with the lowest dose free from toxic effects. An untreated control group also was not included.

One control and four high dose animals died or were sacrificed moribund during this study. The cause of deaths was not established.

Alopecia was observed in many of the low and high dose males and females during the last two to three weeks of treatment and during the first two to three weeks of recovery. Several high dose animals also exhibited this condition throughout recovery. Erythema, oedema, desquamation, exfoliation and fissuring were observed in all of the low and high dose animals throughout the treatment period. These findings were not strongly dose related. Atonia was also evident. Most of these findings were seen also during recovery with a decreased severity and incidence. Less severe erythema and desquamation compared to that seen in treatment animals were observed also in control males and females during the treatment and recovery periods.

Body weights during the last two weeks of treatment and first week of recovery were slightly lower (approximately 5%) than controls in low dose animals and were lower (5-15%) than controls in high dose animals.

At termination of treatment, male and female animals at both low and high doses showed statistically significant decreases in mean total leukocyte counts. Mean haemoglobin and hematocrit values and mean erythrocyte count of high dose females were significantly reduced also at this time. Albumin was reduced also in high dose females. Total protein and globulin were decreased and albumin/globulin ratios were increased in all treatment animals in a dose dependent fashion.

In all males, treatment related decreases were observed in the absolute and relative testes and epididymis weights at the end of the treatment and recovery periods. Compared to control, absolute testes weights were decreased 21 and 35% in low and high dose males respectively at the end of treatment and were decreased 22 and 58% respectively following recovery. Treatment related increases were observed in the liver weights of low and high dose males (5 and 30% respectively) and low and high dose females (12 and 23% respectively) following treatment and in high dose males (14%) following recovery.

Treatment related microscopic lesions in the skin of high dose animals were seen at termination of the treatment period. High dose recovery and low dose treatment and recovery animals were not examined. Skin changes included slight to moderately severe hyperkeratosis and epithelial hyperplasia. Possible treatment related liver changes were observed in the high dose group only and included multifocal areas of minimal to moderate hepatocellular degeneration usually accompanied by multifocal areas of necrosis and/or multifocal areas of coarse cytoplasmic vacuolation of hepatocytes. Testicular changes were observed only in high dose males following treatment and recovery. These included aspermatogenesis, reduced numbers of spermatids and multifocal to diffuse tubular hypoplasia. Epithelial hypoplasia of the epididymis accompanied the testicular changes in many animals at termination of treatment but not following recovery.

No NOAEL could be established from this study (HERTG, 2001a).

The second 28-day dermal toxicity study on benzenesulfonic acid, mono- and dialkyl derivatives, magnesium salt (CAS 71786-47-5) in rats was conducted in 1995 according to OECD 410 test guidelines. The test material was applied topically to the clipped, unabraded, dorsal surface of fifteen Sprague-Dawley CD rats at doses of 0 (control), 100, 300 and 1000 mg/kg bw/day for 6 h/day, 7 days/week for 28 days. A high dose satellite recovery group was observed for an additional 14-day recovery period.

Test material was held in contact with the skin by a gauze patch immobilised by wrappings. Clinical observations were made daily.

No treatment related mortality was observed. One 300 mg/kg bw/day female died on Day 19, however this was attributed to the wrapping procedure. One 1000 mg/kg bw/day male died following blood collection at study termination. Desquamation was observed in one 300 mg/kg bw/day female on days 4 and 7. Body weights and food consumption were unremarkable during the treatment and recovery periods. No other significant clinical or dermal irritancy observations were recorded.

There were no treatment related differences from controls in haematology data of the treated animals following dosing or during recovery. Following recovery, there were several statistically significant differences from controls noted in haematology parameters in satellite animals. These included decreases in mean white blood cell count, absolute lymphocytes and basophil counts in females, an increase in mean percentage of large unclassified cells in males and females and an increase in mean corpuscular haemoglobin in the females. All of these differences were within normal ranges.

No notable treatment related changes were evident in serum chemistry values in the treated animals at termination of the treatment period. Following recovery there

were a number of small, but statistically significant differences in the serum chemistry parameters of the satellite animals. These included a decrease in mean blood urea nitrogen and sodium in males, chloride in females, increases in phosphorus and bilirubin in males and increases in calcium, total bilirubin and triglycerides in females. All of these findings were within the range of normal values and comparable to controls following the termination of dosing. These findings in recovery animals were also not considered clinically significant.

Slight alterations were noted in several organ weights at termination of dosing or at recovery. These included a statistically significant increase in absolute and relative liver weight in the 100 mg/kg bw/day females and statistically significant decreases in relative brain and ovary weights in the 1000 mg/kg bw/day females after recovery. These findings did not correlate with any histopathological findings and were not attributed to treatment. No similar effects were reported at higher doses in this study and no aberrant microscopic findings related to the test material were noted in any group.

A NOAEL of 1000 mg/kg bw/day was established for this study (HERTG, 2001a).

10.4.3 Inhalation studies

No repeat dose inhalation toxicity studies were identified for SAS or suitable analogues.

10.5 Reproductive toxicity

No reproductive toxicity studies were available for SAS or suitable analogues.

The HERTG Test Plan submission to the USEPA HPV Challenge Program (HERTG (2001b) indicates that a 28-day dose-range finding study and a one-generation reproductive/developmental toxicity study will be conducted on benzenesulfonic acid, C15-C24 branched and linear alkyl derivatives, calcium salt (CAS 115733-09-0). At the time of writing, results for this study were not yet available.

10.6 Genotoxicity

No genotoxicity studies on SAS were available. However, three robust summaries on suitable analogues were submitted in the HERTG US HPV Challenge Program submission (HERTG, 2001a). The first in vitro bacterial mutagenicity study was conducted on a C15-21 alkaryl sodium salt derivative (CAS unknown, an analogue of CAS 78330-12-8 i.e. SAS) to OECD 471 test guidelines. The second study was an in vitro chromosome aberration assay on benzenesulfonic acid, mono- and dialkyl derivatives, magnesium salts (CAS 71786-47-5) conducted to OECD TG 473 test guidelines. The third study was an in vivo mammalian erythrocyte micronucleus study conducted on a C20-24 alkaryl sodium salt derivative (CAS unknown, an analogue of benzenesulfonic acid, mono- C16-C24 alkyl derivatives, calcium salts, CAS 70024-69-0) to OECD 474 test guidelines.

The first study used *Salmonella typhimurium* strains TA98, TA100, TA1535, TA1537 and TA1538 using the plate incorporation method. The analogue was tested at concentrations of 0.1, 0.3, 1.0, 3.0 and 10 µg/plate with and without metabolic activation using 25 µL/plate of the S9 liver fraction from Aroclor 1254

pre-treated Sprague-Dawley rats. A series of positive controls (2-aminoanthracene, 2-nitroflourene, sodium azide and 9-aminoacridine) at varying concentrations were used to confirm results for each strain.

Slight cytotoxicity was observed in an initial dose range finding study (concentrations from 0.005 to 10 mg/plate) with tester strain TA100 without metabolic activation. Positive controls performed accordingly. The test material was not mutagenic in this assay with or without metabolic activation.

This study was conducted prior to revision of OECD 471 test guidelines, which suggested inclusion of either *Escherichia coli* strain WP2 uvrA or *Salmonella typhimurium* strain TA102. The significance of not testing these additional strains is unknown (HERTG, 2001a).

The second genotoxicity study was a chromosome aberration assay conducted on benzenesulfonic acid, mono- and dialkyl derivatives, magnesium salts (CAS 71786-47-5) to OECD 473 test guidelines. Chinese hamster ovary (CHO) cells were incubated with 10, 20, 40, 80, 120 or 160 μg/mL test material with or without metabolic activation using the S9 liver fraction from Aroclor 1254 pre-treated Sprague-Dawley rats. Cultures were incubated with test substance for 16 hours with repeat assays performed for 16 or 40 hours.

Precipitation was observed at test material concentrations greater than 80 μg/mL. There were no statistically significant differences in the number of chromosomal aberrations at 16 hours with metabolic activation and at 40 hours with and without metabolic activation. A statistically significant difference was observed at one dose level in the initial 16-hour harvest without metabolic activation but not in the repeat harvest. Positive controls performed accordingly. The test substance was not mutagenic in this assay with or without metabolic activation (HERTG, 2001a).

The third study was a mammalian erythrocyte micronucleus test conducted with C20-24 alkaryl sodium salt derivative (CAS unknown) on 50-day-old Swiss albino Crl: CD-1 (ICR) male and female mice. A single dose of 0, 100, 200, 400 and 500 mg/kg was administered intraperitoneally at a rate of 5 mL/kg bw in a peanut oil vehicle to 15 or 18 animals/sex. Selected animals were sacrificed for bone marrow sampling 24, 48 and 72 hours post treatment.

A number of deaths were recorded at the top two doses (400 and 500 mg/kg bw) in both males and females. Other clinical signs of toxicity observed included palpebral closure, decreased motor activity and weakness.

No biological or statistically significant increases in the number of micronucleated polychromatic erythrocytes were observed in any treatment group compared to vehicle controls. Ratios of micronucleated versus non-micronucleated polychromatic erythrocytes for individual animals were in the expected ranges for control animals. Positive controls performed accordingly. Overall, the test material did not induce micronuclei in bone marrow erythrocytes of mice.

10.7 Carcinogenicity

No studies are available on SAS or suitable analogues.

10.8 Human exposure

No human exposure studies are available on SAS or suitable analogues.

11. Hazard Classification

This section discusses the classification of the health effects of SAS according to the NOHSC *Approved Criteria for Classifying Hazardous Substances* (the Approved Criteria) (NOHSC, 1999a) or, in the case of physicochemical hazards, the *Australian Code for the Transport of Dangerous Goods by Road and Rail* (ADG Code) (FORS, 1998). The Approved Criteria are cited in the NOHSC *National Model Regulations for the Control of Workplace Hazardous Substances* (NOHSC, 1994d) and provide the mandatory criteria for determining whether a workplace chemical is hazardous or not.

Human data were unavailable for this chemical; therefore the classification for health hazards has been based on experimental studies (animal and in vitro tests) on SAS and structurally similar analogues. In extrapolating results from experimental studies to humans, and from analogue data to SAS, consideration was given to relevant issues such as study quality, analogue similarity to SAS, interspecies variability and weight of evidence.

Classification of SAS in accordance with the OECD *Globally Harmonized System* (GHS) *of Classification and Labelling of Chemicals* (OECD, 2002) can be found in Appendix 3. This is provided for guidance only, is not mandatory, and has no legal status at present.

Sodium alkylbenzene sulfonate is not listed in the NOHSC *List of Designated Hazardous Substances* (NOHSC, 1999b).

11.1 Physicochemical hazards

Sodium alkylbenzene sulfonate is a viscous liquid with a modelled melting point of 347 ^{o}C and boiling point of 788 ^{o}C and a measured flash point (closed cup) of 181.7 ^{o}C.

With respect to the ADG Code (FORS, 1998), SAS does not meet the criteria for classification as a dangerous good on the basis of these physicochemical data.

11.2 Health hazards

11.2.1 Acute toxicity

Results from a 14-day oral (gavage) toxicity study of SAS on rats with doses up to 5.0 g/kg bw showed no mortality, clinical or pathological signs of toxicity. An LD50 of > 5.0 g/kg bw was reported.

The three additional rat oral (gavage) studies on C15-21 alkaryl sodium salt derivative (an analogue of 78330-12-8) (CAS unknown), C20-24 alkaryl sodium salt derivative (analogue of 70024-69-0) (CAS unknown) and benzenesulfonic acid, C15-C24 branched and linear alkyl derivatives, calcium salt (CAS 115733-09-0) all reported an LD50 of greater than 5.0 mg/kg bw.

Two acute dermal toxicity studies on chemicals benzenesulfonic acid, C15-C24 branched and linear alkyl derivatives, calcium salt (CAS 115733-09-0) and C20-24

alkaryl calcium salt derivative (analogue of CAS 70024-69-0) (CAS unknown) resulted in dermal LD50s > 5.0 and > 2.0 g/kg bw respectively.

No inhalation studies were available on sodium alkylbenzene sulfonate or structurally similar analogues.

Classification:

Based on the acute oral (gavage) study on SAS and oral and dermal studies on structurally similar analogues, SAS does not meet the Approved Criteria for *acute lethal effects* via the oral and dermal routes, according to the Approved Criteria (NOHSC, 1999a). No data are available to classify SAS for *acute lethal effects* via the inhalation route.

11.2.2 Irritation effects

One skin and one eye irritation study were conducted on SAS.

For the skin irritation study, rabbits showed well defined erythema (Draize score 2) and slight oedema formation (Draize score 2) at both abraded and intact skin sites at 24 hours post treatment. Erythema reactions were increased to moderate/severe (Draize score 3) while oedema reactions were decreased to very slight oedema (Draize score 1) in all animals at both sites 72 hours post treatment.

The Approved Criteria specifies a dermal exposure of 4 hours, whereas this study on SAS used a 24 hour exposure.

Four out of 6 animals tested for eye irritancy potential of SAS exhibited conjunctival redness (Draize score 2) at day 1. However all vessels returned to normal by day 3. No reactions were noted in the cornea and iris.

No data are available on respiratory irritation for SAS or close structural analogues.

Classification:

Based on the acute skin irritation studies, SAS does not meet the Approved Criteria (NOHSC, 1999a) for *Inflammation of the Skin* as data are insufficient for classification.

Sodium alkylbenzene sulfonate does not meet the Approved Criteria (NOHSC, 1999a) for ocular lesions and no data are available for classification against the Approved Criteria (NOHSC, 1999a) for *respiratory system irritation*.

11.2.3 Sensitising effects

There are no animal studies or human case reports of skin or respiratory sensitisation to SAS or close structural analogues.

Classification:

Data are insufficient for the classification of SAS against the Approved Criteria (NOHSC, 1999a) with respect to *sensitising effects* (R42, R43).

11.2.4 Effects from repeated or prolonged exposure

No human long term or animal repeat dose toxicity studies were available on SAS. However one oral and two dermal repeat dose studies were available on analogues.

Oral study

An OECD 407 28-day repeated dose rat oral (gavage) study on a C20-24 alkaryl calcium salt derivative (CAS unknown) (analogue of CAS 70024-69-0) found a reduction in mean cholesterol values in the males and females treated at the 1000 mg/kg dose level. A NOEL of 500 mg/kg bw/day was assigned.

Dermal studies

Two dermal 28-day repeat dose toxicity studies on rabbits using benzenesulfonic acid, mono- and dialkyl derivatives, magnesium salts (CAS 71786-47-5) were available.

In the first study conducted in 1981, 2 mL/kg/day of test substance at concentrations of 0, 25 and 100% (w/v) was applied topically to 15 New Zealand White rabbits/sex/group for 6 hours/day, 5 days/week for 28 days. On the basis of toxic effects seen in the lowest dose animals, no NOAEL was identified for this study.

The second dermal 28-day study in rabbits conducted in 1995 used standardised doses of 0, 100, 300 and 1000 mg/kg bw/day in 5 rabbits/sex treated for 6 hours/day, 7 day/week for 28 days. An additional (satellite) group dosed at 1000 mg/kg bw/day under observation for a further 14 days.

No treatment related deaths were recorded in this study. No significant treatment related effects were recorded with regards to clinical observations, haematology, serum chemistry, post-mortem observations or organ weights. A NOAEL of 1000 mg/kg bw/day was established for this study.

Classification:

Based on these 28-day repeat dose oral and dermal studies, SAS does not meet the Approved Criteria (NOHSC, 1999a) for *severe effects after repeated or prolonged exposure via the oral or dermal routes*.

No repeat dose inhalation toxicity studies were identified for SAS or suitable analogues and so data are insufficient to classify SAS for *severe effects after repeated or prolonged exposure via the inhalation route*.

11.2.5 Reproductive effects

No reproductive studies were available on SAS. The HERTG (2001b) test plan submission on Petroleum Additive Alkaryl Sulfonate Category to the USEPA HPV Challenge Program indicates the proposal for a 28-day repeated dose oral dose range finding and one generation reproductive/developmental study to be conducted on the SAS analogue benzenesulfonic acid, C15-C24 branched and linear alkyl derivatives, calcium salt (CAS 115733-09-0).

Classification:

Data are insufficient to classify SAS against the Approved Criteria (NOHSC, 1999a) with respect to *fertility/developmental effects* (R60, R61, R62).

11.2.6 Genotoxicity

Robust summaries on 3 SAS analogues were available - a bacterial mutagenicity study conducted on a C15-21 alkaryl sodium salt derivative (CAS unknown, an analogue of CAS 78330-12-8 i.e. SAS), an in vitro chromosome aberration assay on benzenesulfonic acid, mono- and dialkyl derivatives, magnesium salts (CAS 71786-47-5) and an in vivo mammalian erythrocyte micronucleus study conducted on a C20-24 alkaryl sodium salt derivative (CAS unknown, an analogue of benzenesulfonic acid, mono- C16-C24 alkyl derivatives, calcium salts, CAS 70024-69-0).

All three studies showed no mutagenic effects for the test substance.

Classification:

On the basis of analogue data, SAS does not meet the Approved Criteria (NOHSC, 1999a) for *mutagenic effects* (R40, R46).

11.2.7 Carcinogenicity

No studies were available for SAS or suitable analogues.

Classification:

Data are insufficient for the classification of sodium alkylbenzene sulfonate against the NOHSC Approved Criteria (NOHSC, 1999a) with respect to *carcinogenic effects* (R40, R45, R49).

12. Effects on Organisms in the Environment

This section provides information on the effects of SAS and sodium sulphate, the predominant combustion by-product, on animals and plants. Based on SAS use patterns, the review of effects has included the potential effects to organisms that inhabit terrestrial and aquatic environments.

Limited ecotoxicity data were available for SAS and information has been considered from LAS analogues of similar chemical structure.

Ecotoxicity data have been obtained from various sources but principally the Australian and New Zealand Water Quality Guidelines (ANZECC/ARMCANZ, 2000a,b). As this reference source has been peer reviewed previously, the publications obtained from this source have not been peer reviewed for this present report (see citation for specific reference sources).

12.1 Terrestrial animals

12.1.1 SAS

Higher order animals

Kinetics/metabolism and toxicity of SAS to mammals (e.g. rats, mice, monkeys, rabbits) have been presented in Sections 9 and 10, respectively. No information was available on the potential effects of excessive SAS exposure to birds or other terrestrial wildlife species. The use pattern indicates a low likelihood of wildlife exposure to SAS.

Soil-dwelling organisms

SAS may be released to soils through spills and leaks. However, the use pattern would suggest a low, diffuse exposure to the environment.

No data were available on the effects of SAS to soil-dwelling organisms. Following is a summary of soil ecotoxicity data for soil-dwelling invertebrates and microbial communities exposed to analogues of potentially similar ecotoxicity.

In a study of the effects (survival, reproduction, growth) of a C10-13 sodium-based LAS on six species of soil invertebrates inhabiting a sandy agricultural soil test substrate, effects were detected in the soil concentration range of 40-60 mg/kg (Holmstrup and Krogh, 2001). Reproduction was 4 times more sensitive in earthworms and enchytraeids than in springtails and mites. A predicted no effect concentration (PNEC) of 4.6 mg/kg was derived by Jensen et al. (2001) based on these data.

The effects of LAS-spiked soil on newly hatched and adult Collembola *Folsomia fimetaria* were investigated over a 3-week exposure period (Holmstrup and Krogh, 1996). Mean C chain length of this LAS was reported as 11.53. Over the range 0-1000 mg/kg adult survival rate was unaffected. However, juveniles were affected

and EC10 values were 163 mg/kg (growth), 185 mg/kg (moulting frequency), and 147 mg/kg (reproductive output).

Gejlsbjerg et al. (2001) investigated the effects of LAS on two soil invertebrates (*F. candida* and *Enchytraeus albidus*) and five different soil microbial processes (aerobic respiration, nitrification, denitrification, anaerobic CH_4 production and anaerobic CO_2 production). LC50 and EC50 values for the invertebrates were similar, in the range of 1143-1437 mg/kg. EC50 values for nitrification and CH_4 production were 431 mg/kg and 277 mg/kg. Aerobic respiration and denitrification were not inhibited.

Jensen and Sverdrup (2002) investigated the effect of LAS on *F. candida* survival and reproduction in a sandy loam soil of 2.8% humus content (1.6% organic carbon), deriving EC50 and EC10 values of 803 mg/kg dry weight (95% CI 560 to 1000 mg/kg) and 161 mg/kg (95% CI 113 to 209 mg/kg), respectively. The LC50 for LAS was >800 mg/kg.

Functional diversity of an aerobic heterotrophic bacterial community, tested by a community-level physiological profile, was not affected when exposed for 1, 2, or 4 weeks to LAS-spiked sandy soil at two tested concentrations of 22 and 174 mg/kg (Vinther et al., 2003). Most (93-98%) of the LAS was biodegraded during the tests and bacteria growth was promoted at a concentration of 174 mg/kg over 4 weeks. No inhibitory effects were observed.

Microbial effects of LAS have been investigated. LAS inhibited microbial growth in the short term (1-2 weeks) with an EC10 range of 3 to 39 mg/kg. Inclusion of LAS in sewage sludge reduced the inhibitory effects with an EC10 of 8 to 102 mg/kg (Elsgaard et al., 2001a). Using assays of 10 different microbial soil parameters, Elsgaard et al. (2001b) derived EC10 values in the range of 8 to 22 mg/kg for LAS, indicating that the small range probably reflects a similar mode of action of LAS toxicity, ascribed to cell membrane interactions and showed that sensitivity to LAS was similar for various soil micro-organisms.

In summary, it is unlikely that there would be any effects from SAS due to the low and diffuse exposure resulting from the use pattern.

12.1.2 Sodium sulphate

Mammalian toxicity data for sodium sulphate and sulphuric acid (H_2SO_4) resulting from SAS combustion have been presented in Section 11. No information was available on the potential effects of excessive sodium sulphate exposure to birds or other wildlife species.

Sodium is an essential element in more advanced organisms. Animal diets must contain a certain amount of sodium. The sodium cation is the main extracellular cation in animals. The ratio of sodium/potassium concentrations in intercellular and extracellular fluid is responsible for the transport of ions through the cellular membranes, the regulation of the osmotic pressure inside the cell, the transmission of nervous pulses and other electrophysiological functions in animals. The importance of sodium as salt in the diet was recognized well before sodium itself was understood to be an element. Losses derive from the functional supply of cells and tissues, sweating and loss of body fluids. It is most important that the sodium ion is replaced through proper diet, as there is no reserve store of sodium ions in the animal body. Depletion results in serious symptoms and potentially death.

Symptoms of sodium deficiency include thirst, anorexia and nausea, lassitude, muscle cramps and mental disturbances. Sodium compounds are relatively harmless to animals, as long as they are not ingested in excess.

Sulphur is essential to life. It is a minor constituent of fats, body fluids, and skeletal minerals. Sulphur, through reduction of dissolved sulphate, is a key component in most proteins since it is contained in the amino acids methionine and cysteine. Sulphur-sulphur interactions are important in determining protein tertiary structure.

Nutritional polio (polioencephalomalacia, a central nervous system disorder) may develop in cattle exposed to an excess of sulphate in the diet (e.g. excess in food and/or drinking water). Physiological effects include diarrhoea, reduced growth, reproduction and lactation, blind staggers and death. Such conditions may arise in areas through consumption of run-off from alkaline soils, alkaline groundwater, and may be exacerbated during drought due to salt accumulation in waterholes. Water concentrations leading to polioencephalomalacia range from 2800 to 9000 mg SO_4/L (Society for Range Management, 2003).

The Australian water quality guidelines for drinking water for the protection of livestock indicate no adverse effects to stock are expected if the concentration of sulphate in drinking water does not exceed 1000 mg/L (ANZECC/ARMCANZ, 2000a). Adverse effects may occur at sulphate concentrations between 1000 and 2000 mg/L, especially in young or lactating animals or in dry, hot weather when water intake is high. These effects may be temporary and may cease once stock become accustomed to the water. Levels of sulphate greater than 2000 mg/L may cause chronic or acute health problems in stock (ANZECC/ARMCANZ, 2000a). The guideline is consistent with values recommended for sulphate in livestock drinking water in Canada (CCREM, 1987; Environment Canada, 2002) and South Africa (DWAF, 1996).

12.2 Terrestrial plants

12.2.1 SAS

No information was available on the toxicity of SAS to terrestrial plants.

12.2.2 Sodium sulphate

High levels of salts including sodium and sulphate in soil water may inhibit plant growth by two processes. A water-deficient (osmotic) effect may occur whereby soil salinity reduces the ability of plants to take up water, leading to a reduction in growth rate. In addition, if excessive amounts of salts enter a plant in the transpiration stream there may be injury to cells in the transpiring leaves and this may cause further reductions in growth (Greenway and Munns, 1980).

Sodicity is a condition that degrades soil properties by making the soil more dispersible and erodible, restricting water entry and reducing hydraulic conductivity (the ability of the soil to conduct water). These factors limit leaching so that salt accumulates over long periods of time, giving rise to saline subsoils. Furthermore, a soil with increased dispersibility becomes more susceptible to erosion by water and wind (ANZECC/ARMCANZ, 2000a).

A high proportion of sodium in soil can result in dispersion and, once dry, soils may become dense, cloddy and structureless, destroying natural particle

aggregation. Because the relative proportions of exchangeable cations in a given soil are determined by the relative concentration of cations in the soil solution, the composition of irrigation water can influence soil sodicity (Rengasamy and Olsson, 1995). Plants can be affected through sodicity and effects on soil fertility.

Sulphate is a readily available and essential plant nutrient (ANZECC/ARMCANZ, 2000a). Elemental sulphur and organic sulphur are unavailable to plants, requiring microbial transformation. Plants absorb the sulphate ion (SO_4^{2-}) in soils through their roots, and some SO_2 may be absorbed from the atmosphere through stomata in the leaves. Within the plant, sulphate is reduced to and incorporated in various organic molecules, particularly amino acids (cysteine and methionine) as components of proteins.

Sulphate deficiency symptoms include wilting and uniform chlorosis. Sulphate phytotoxicity may occur in soils through over-fertilisation or irrigation resulting in accumulation in soils. When sulphur is supplied to soils, plants have the capacity to take up an excess amount. While this may not affect the plant, it may affect herbivores that consume the plants.

Eaton (1942) investigated the effect of sulphate (sodium salt) and chloride on various plant species, finding that effects occur gradually over a range of concentrations rather than above a threshold limit, and that sulphur accumulates in plants. Effects were greater as temperature increased. Plant leaves may be affected (burnt) by excessive exposure to sulphate.

The information available indicates that sulphate is an essential nutrient for plants and of low toxicity but exposure to high to very high soil sulphate concentrations, combined with low pH soil conditions, or excessive foliar sulphate may lead to adverse effects in plants. However, use of SAS is unlikely to lead to excessive soil SO_4 and toxicity from foliar application.

A range of salt tolerances (includes other salts, e.g. chloride) are expressed by plant species depending on physiological adaptations to environmental conditions (e.g. extremely saline soil waters >8800 mg/L to non-saline <1100 mg/L; WA Agriculture, 2003; ANZECC/ARMCANZ, 2000a; Eaton, 1942).

ANZECC/ARMCANZ (2000b) indicates that the derivation of a single irrigation water quality guideline for salinity and sodicity is not possible, with interactive factors including irrigation water quality, soil properties, plant salt tolerance, climate, landscape (i.e. geological and hydrological features), and water and soil management.

12.3 Aquatic organisms

12.3.1 SAS

Release of SAS to freshwater or marine environments is unlikely under the proposed use pattern. Spill incidents involving containers of SAS may potentially occur, but these would generally be of a small scale and unlikely to adversely affect the wider environment beyond the spill area. The following provides a summary of aquatic toxicity data for SAS and analogues of potentially similar ecotoxicity.

The applicant provided freshwater fish (rainbow trout) and crustacean (*Daphnia magna*) acute aquatic toxicity test reports for SAS. Information was also available for SAS analogues (e.g. HERTG, 2001a,b). Other literature sources were also reviewed.

A Test Plan for the HPV Challenge Program for petroleum additive alkaryl sulfonate category chemicals was submitted to USEPA by HERTG (2001b). The Test Plan and Robust Summaries included outlines of freshwater toxicity tests performed with several SAS analogues on fish sheepshead minnow *Cyprinodon veriegatus* (Springborn Bionomics, 1986a-c), fathead minnow *Pimephales promelas* (Wilbury, 1993a,b), *Daphnia magna* (Wilbury, 1993c-e) and algae *Selenastrum capricornutum* (Wilbury, 1994a-c). The toxicity data are summarised below.

In their review of the Test Plan (HERTG, 2001b), the USEPA (2002a) indicated that the OECD test method for difficult substances (OECD, 2000) is most relevant to testing chemicals analogous to SAS, indicating that water accommodated fraction (WAF) studies are not useful for risk assessment of chemicals such as SAS. Water dispersibility concentrations were not determined in any of the tests included in HERTG (2001a). Furthermore, the USEPA (2002a) indicated that total organic carbon (TOC) content of the test media were greater than the required < 2.0 mg/L level. USEPA indicated that the chemicals tested are likely to have low water solubility, high octanol/water partition coefficients, and low vapour pressures, indicating also that these chemicals may be more water dispersible than soluble. Overall, the USEPA concluded that the submitter's justification for using the studies as supporting information is inadequate. The USEPA has recommended HERTG undertake further aquatic toxicity testing.

Fish toxicity data

Safepharm Laboratories Limited (2001b) reported on the acute toxicity of SAS to juvenile rainbow trout (*Onchorhynchus mykiss*) using a WAF method employing a single phase separation period. Test methods followed OECD 203 test guidelines (OECD, 1984a) and were undertaken in accordance with good laboratory practice under a quality assurance program.

No mortality occurred and no sublethal effects were observed at the highest nominal test concentration (i.e. 96 h LC50 >1000 mg/L WAF and NOEC at least 1000 mg/L WAF). In view of difficulties associated with the evaluation of aquatic toxicity of poorly water-soluble test materials, a modification of the standard method for the preparation of aqueous media was performed (ECETOC, 1996; OECD, 2000). To obtain the WAF, the test media (21.00 g) was mixed with dechlorinated tap water (21 L) at 30 rpm for 24 hours, then stopped and allowed to settle for 2 hours. The aqueous WAF was then extracted by siphon (first 75-100 mL discarded), filtered, and used for testing. Test conditions included 16 hours light: 8 hours dark with 20 minute dawn/dusk transition lighting, 14°C, glass fibre fish tanks, dissolved oxygen >9.4 mg O_2/L (recorded daily) and no feeding during tests. The concentration, homogeneity and stability of the test substance were not measured. While the chemical was probably not toxic up to its limit of water solubility, the significance of these omissions to the test result cannot be evaluated, but it creates a source of uncertainty in the interpretation of the toxicity data as it is not possible to establish what concentration the test organisms were exposed to.

USEPA (2002a) review comments on the inadequacy of WAF studies (abovementioned) apply to this study.

In HERTG (2001a), Wilbury (1993b) reported the effects of an alkaryl sulphonate (benzenesulfonic acid, mono-and dialkyl derivatives, magnesium salts; CAS 71786-47-5) on fathead minnow. Test dilutions involved control, 100, 300 and 1000 mg/L (nominal) WAF loading rates, static renewal, and followed USEPA Toxic Substances Control Act 797.1400 (USEPA, 1985a) and OECD TG 203 guidelines. No mortality was observed and a 96 h NOEC of >1000 mg/L based on WAF loading rates was derived.

As reported by HERTG (2001a), Springborn Bionomics (1986c) also tested the effects of benzenesulfonic acid, mono-and dialkyl derivatives, magnesium salts (CAS 71786-47-5) on sheepshead minnow. Test dilutions involved control and 10 000 mg/L WAF loading rates, static renewal, and followed OECD TG 203 guidelines. The 96 h LL50 (≈ LC50) was > 10 000 mg/L based on WAF loading rates. This is equivalent to a 96 h LL0 (≈ NOEC) of 10 000 mg/L WAF as this substance concentration did not increase mortality or have any toxic effects on the fish. USEPA (2002a) review comments on the inadequacy of WAF studies (abovementioned) apply to this study.

Acute and chronic effects of LAS (mean C11.5) on an estuarine goby (*Pomatoschistus microps*) have been investigated (Christiansen et al., 1998). Dilution series included 0 (control), 2.0, 2.5, 3.0, 3.5, 4.0, 4.5 and 5.0 mg/L for survival tests, 0 (control), 0.05, 0.1, 0.5, 0.75 and 1.0 mg/L for growth tests, and 0 (control), 0.1, 0.5, 0.75 and 1.0 mg/L for respiration tests. Test conditions included 800 mL glass beakers pre-rinsed with LAS, salinity ~15 %, pH 7.0-7.5, 10 light: 14 dark, and temperature ~16°C. A static test 96 h LC50 of 2600 µg/L ±490 µg/L (95% C.I.) was derived. The LAS used consisted of the following carbon chain length proportions: C10 (5-10%), C11 (43-50%), C12 35-40%, C13 (7-12%) and C14 (≤ 1%). The acute responses to LAS were excess mucous secretion and apathy at 1000 µg/L, air gulping and distension of the mouth and opercula and changes in pigmentation and erratic swimming at 2000 µg/L and 3000 µg/L and loss of balance and reactivity at ≥ 4000 µg/L. A concentration of 100 µg/L (LOEC) over 28 days led to a significant decrease in growth and respiration but ingestion was not affected.

Sulphophenyl carboxylates, which are derived from the biodegradation of LAS, are not considered to possess estrogenic activity based on a recombinant yeast screen (Navas, 1999; Routledge and Sumpter, 1995). This would suggest that the reviewed chemical is also non-estrogenic.

The Danish EPA (2001) provides a review summary of aquatic toxicity data for fathead minnows and rainbow trout exposed to LAS compounds of various chain lengths. Acute toxicity data from several tests with fathead minnows demonstrate 96 h LC50 values in the range 500 μg/L (C14) to 57 500 μg/L (C10) and a 7 day LC50 of 710 μg/L (C11.9) has also been derived. For minnows, 48 h LC50 values range from 400 μg/L (C14) to 43 000 μg/L (C10). Thirty day NOEC for minnows of 150 μg/L (C13) and 900 μg/L (C11.8) have also been derived. A 96 h LC50 of 360 μg/L (95% C.I. 0.25-0.51) for rainbow trout has been derived based on exposure to LAS with a carbon chain length of C10-15. These data highlight differential aquatic toxicity depending on analogue carbon chain length.

Fish acute toxicity test results highlight a contrast in effect levels, with 96 h LC50 values ranging from 360 μg/L to > 10 000 mg/L WAF due to different test methods, analogue and species used. Lowest freshwater and marine NOEC values correspond to 80 μg/L and 25 μg/L, respectively.

Aquatic invertebrates

Safepharm Laboratories Limited (2001a) reported on the acute toxicity of SAS to adult *Daphnia magna* using a WAF method described above (Safepharm Laboratories Limited, 2001b). Tests were performed in accordance with OECD 202 test guidelines and were undertaken in accordance with good laboratory practice under a quality assurance program.

No mortality occurred at the highest nominal test concentration (i.e. 24-48 h LC50 > 1000 mg/L WAF and NOEC at least 1000 mg/L WAF), and no adverse effects were observed. Test conditions included a dilution series of control, 10, 100 and 1000 mg/L (nominal) loading rates, 10 animals per 250 mL test container, 21°C, and 16 hours light: 8 hours dark with 20 minute dawn/dusk transition lighting. The concentration, homogeneity and stability of the test substance were not measured. While the chemical was probably not toxic up to its limit of water solubility, the significance of these omissions to the test result cannot be evaluated and it is not possible to establish what concentration the test organisms were exposed to. USEPA (2002a) review comments on the inadequacy of WAF studies (abovementioned) apply to this study.

As reported in HERTG (2001a), Wilbury (1993c) reported the effects of an alkaryl sulphonate (benzenesulfonic acid, C14-C24 branched and linear alkyl derivatives, calcium salts; CAS 115733-09-0) on the cladoceran *Daphnia magna*. Test dilutions involved control, 100, 300 and 1000 mg/L (nominal) WAF loading rates, static, and followed USEPA Toxic Substances Control Act 797.1400 (USEPA, 1985b) and OECD 202 test guidelines (1984b). No mortality was observed and a 48 h NOEC of >1000 mg/L based on WAF loading rates was been derived. USEPA (2002a) review comments on the inadequacy of WAF studies (abovementioned) apply to this study.

As reported in HERTG (2001a), Wilbury (1993e) reported the effects of an alkaryl sulphonate (benzenesulfonic acid, mono- and dialkyl derivatives, magnesium salts; CAS 71786-47-5) on the cladoceran *Daphnia magna*. Test dilutions involved control, 100, 300 and 1000 mg/L (nominal) WAF loading rates, static, and followed USEPA Toxic Substances Control Act 797.1300 (USEPA, 1985b) and OECD 202 test guidelines (1984b). No effects were observed and a 48 h NOEC of > 1000 mg/L based on WAF loading rates was derived. USEPA (2002) review

comments on the inadequacy of WAF studies (abovementioned) apply to this study.

The marine copepod *Acartia tonsa* is sensitive to the effects of LAS (C10-C13; Kusk and Petersen, 1997). With two salinities, 18 % and 28 %, the 48 h LC50 values were 2100 µg/L and 8800 µg/L, respectively (nominal concentrations). Toxicity decreased with increasing salinity. Eight day L(E)C10 (larval survival and development) were in the range of 200-300 µg/L, with an 8 d LC50 of 540 µg/L.

The Danish EPA (2001) provide a review summary of aquatic toxicity data for *Daphnia magna* exposed to LAS compounds of various chain lengths. Acute toxicity data from several tests demonstrate 48 h LC50 values in the range 110 µg/L (C16) to 29 500 µg/L (C10), and 96 h LC50 values from 2190 µg/L (C13) to 3940 µg/L (C13). Twenty-one day L(E)C50 values of 1170 µg/L (C13) and 1110 µg/L (C13; reproduction) have also been derived.

ANZECC/ARMCANZ (2000c) provide a summary of NOEC values for aquatic invertebrates (refer Table 9).

Like fish, aquatic invertebrate toxicity test results highlight a contrast in effect levels due to different test methods used, analogues of different carbon chain length and species. LC50 values for LAS have been found in the range of 1000 to 10 000 µg/L when *Daphnia* were exposed to C10-C13. However, toxicity increases with increasing chain length and LC50 values for ≥ C14 are < 1000 µg/L.

Algae

As reported in HERTG (2001a), Wilbury (1994a) reported the effects of an alkaryl sulphonate (benzenesulfonic acid, C_{14}-C_{24} branched and linear alkyl derivatives, calcium salts; CAS 115733-09-0) on the freshwater alga *Pseudokirchneriella subcapitata*. Test dilutions involved control, 100, 300 and 1000 mg/L (nominal) WAF loading rates, static, and followed USEPA Toxic Substances Control Act 797.1050 (USEPA, 1985c) and OECD 201 test guidelines (1984c). No effects were observed and a 72-96 h NOEC of > 1000 mg/L based on WAF loading rates was derived. USEPA (2002) review comments on the inadequacy of WAF studies (abovementioned) apply to this study.

As reported in HERTG (2001a), Wilbury (1994c) reported the effects of an alkaryl sulphonate (benzenesulfonic acid, mono and dialkyl derivatives, magnesium salts; CAS 71786-47-5) on the *P. subcapitata*. Test dilutions involved control, 100, 300 and 1000 mg/L (nominal) WAF loading rates, static, and followed USEPA Toxic Substances Control Act 797.1050 (USEPA, 1985c) and OECD 201 test guidelines (1984c). No effects were observed and a 72-96 h NOEC of > 1000 mg/L based on WAF loading rates was derived. USEPA (2002a) review comments on the inadequacy of WAF studies (abovementioned) apply to this study.

Utsunomiya et al. (1997) examined the effects of LAS (C12) on the salinity-tolerant algae *Dunaliella* sp. and freshwater alga *Chlorella pyrenoidea*. The 24 h EC50 value was 380 µg/L (*Dunaliella*) and 96 h EC50 (*Chlorella*) was 280 µg/L.

Although toxicity data vary among tests, the Danish EPA (2001) indicates that data are most variable for algae species. Approximately 90% of the toxicity data fall between 100 µg/L and 100 mg/L. Typical EC50 values are 1000 µg/L to 100 000 µg/L for freshwater species and < 1000 µg/L to 10 000 µg/L for marine species. The alga *Gymnodium breve* is particularly sensitive to surfactants, with an EC100

of 25 μg/L). Seventy-two hour EC50 values for *Selenastrum capricornutum* range from 18 000 μg/L (C14) to 270 000 μg/L (C10), with NOECs in the range of 7000 μg/L (C14) to 80 000 μg/L (C14) and 96 h EC50 values in the range 29 000 μg/L (C12) to 116 000 μg/L (C13). Ninety-six hour EC50 values for *Microcystis aeruginosa* range from 900 μg/L (C12) to 5000 μg/L (C13).

ANZECC/ARMCANZ (2000c) provide a summary of NOEC values for aquatic invertebrates (refer Table 9).

Planktonic organisms

Planktonic freshwater organisms (e.g. protozoan ciliates and heterotrophic nanoflagellates) exhibited adverse effects when exposed to LAS over a 5 day period. NOEC values were ≥ 20 μg/L, but varied by several orders of magnitude between test organisms (Jorgensen and Christoffersen, 2000).

Sediment-dwelling organisms

No data were found on the effects of SAS to sediment-dwelling organisms. The Danish EPA (2001) indicates that the toxicity of LAS bound to sediment is relatively low compared to LAS in solution. LAS sorb to sediments with partition co-efficients of 50 to 1000. NOEC and LOEC values of 319 mg/kg and 993 mg/kg have been derived for *Chironomus riparius*. The corresponding NOEC for LAS in solution was 2400 μg/L, indicating that only a small fraction sorbed is bioavailable (Painter, 1992). The effects of LAS dissolved in water have been investigated and acute effects found in the range of 250 μg/L to 200 000 μg/L depending on the sediment-dwelling species used. Copepods and sea urchin embryos were most sensitive. Chronic effects (reduced growth) have been identified in the marine mussel (*Mytilus galloprovincialis*) when exposed to dissolved LAS; however, LAS sorbed to sediment did not produce similar effects. The 96 h LC50 values for sediment LAS were 182.5 mg/kg and 200 mg/kg for bivalves *Unio elongatus* and *Anodonata cygnea*. The effects of branched alkylbenzene sulphonate (ABS) to the amphipod *Hyalella azteca* are mediated by sediment organic carbon (peat moss), indicating toxicity related to interstitial water concentration.

The data available indicate that LAS sorbs strongly to sediment particles and has a relatively low bioavailability and thus toxicity to sediment-dwelling organisms.

Sewage sludge micro-organisms and respiration

The effect of SAS on the respiration of activated sludge (as O_2 consumption) has been investigated under laboratory conditions by Woodward-Clyde (1994). The test followed OECD 209 test guidelines (1984d). Activated sludge was obtained from the Cottonwood Subdivision wastewater treatment plant, Franklin, Tennessee. Test duration was 3 hours followed by 1 hour of O_2 monitoring. Dilution series included 10, 100, 1000, and 10 000 ppm, a duplicate control group, and a reference toxicant control. The tests passed quality control protocols. The respiration rates for test dilutions were 51.0, 44.2, 44.7, 48.0 and 36.0 mgO_2/L/hr, respectively. These data translate into growth inhibitions as percentages with respect to controls of 13.1%, 2.0%, 0.9%, -6.4% and 20.2% (all not significantly different from control). The calculated NOEC based on inhibition compared to the control was 10 000 ppm. The EC50 was calculated to be >10 000 ppm. As such, SAS did not inhibit sewage respiration. Results for the reference toxicant were as expected.

Water quality guidelines for the protection of aquatic life

No published water quality guidelines were available for SAS. CLER (1996a) indicate effects levels from LAS exposure to aquatic organisms in the range >100 µg/L (Kimerle, 1989; Danish EPA, 2001). ANZECC/ARMCANZ (2000c) provide a summary of acute aquatic NOEC data (refer Table 9).

Table 9. Summary of freshwater and marine NOEC values for LAS compounds

Organisms	Number of Data	Concentration (µg/L)
Freshwater Organisms		
Fish	5 spp.	250-3200
Crustaceans	2 spp.	1400-3200
Insects	2 spp.	2800-3400
Mesocosm study	1 study	300
Algae	6 spp.	80-15 000
Marine Organisms		
Fish	1 sp.	50
Crustaceans	1 sp.	120
Mussels	2 spp.	25

Source: ANZECC/ARMCANZ (2000c).

ANZECC/ARMCANZ (2000c) have derived a high-reliability water quality trigger value for linear alkylbenzene sulphonates (LAS) for the protection of freshwater ecosystems of 280 µg/L using a statistical distribution method with 95% protection. A low-reliability marine indicative working level (IWL) of 0.1 µg/L has also been derived using an assessment factor approach with an assessment factor of 200 due to limited data availability.

12.3.2 Sodium sulphate

No aquatic toxicity data were available for sodium. It is a common ion in most surface waters, usually in association with chloride (Munns, 2002).

Sulphate is generally not considered of ecological concern, except perhaps when it is a dominant component of total dissolved salts. Most aquatic toxicity data for sulphate are based on acute studies reporting LC50 values in the range of 450 mg/L. A probable chronic threshold of 400 mg/L was developed by URS (2001) for evaluation of sulphate in surface waters for the protection of freshwater aquatic ecosystems.

As a result of acid generation from acid sulphate soils, where soil acidity (pH) falls to below 4, iron, aluminium and other metals may become soluble in toxic quantities and their precipitates can affect water quality. Massive kills of aquatic life can occur. Chronic effects on aquatic systems are common and widespread and include habitat degradation, altered aquatic plant communities, weed invasion by acid-tolerant plants, secondary water quality changes, the presence of disease, reduced aquatic food resources, reduced migration potential of fish and reduced recruitment, survival and growth rates across a wide range of aquatic species (Sammut and Lines-Kelly, 1996; Sammut et al., 1999).

12.4 Summary of environmental effects

12.4.1 SAS

Due to the paucity of aquatic toxicity data for SAS, data for analogues from reports supplied by the applicant as well as robust summaries as supplied to the USEPA (HERTG, 2001a) were used (Table 10). Numerous studies of aquatic toxicity have been performed using LAS compounds and the results are highly variable depending on the analogue, species and test conditions. LAS of longer alkyl chain length are generally more toxic (Danish EPA, 2001). The relatively long carbon length of SAS suggest a relatively high toxicity to aquatic organisms; however, the toxicity test reports for SAS indicate low toxicity up to the point of water solubility. It is suspected that above a certain carbon chain length, water solubility, and thus bioavailability and toxicity, decline. However, there are insufficient data to determine the chain length above which toxicity decreases. In consideration of this uncertainty, the trigger value for LAS derived by ANZECC/ARMCANZ (2000a) for the protection of freshwater organisms of 280 μg/L has been adopted as a predicted no effect concentration (PNEC) for SAS for this assessment. The marine trigger value for LAS derived by ANZECC/ARMCANZ (2000c) of 0.1 μg/L is also adopted as a PNEC for this assessment. This value is considered protective of marine organisms referred to above not considered by ANZECC/ARMCANZ (2000c).

There is currently no environmental hazard classification system in Australia. In accordance with the OECD Globally Harmonized System of Classification and Labelling of Chemicals (OECD, 2002), LAS compounds in general would be classified as Acute 1 Very Toxic to Aquatic Life based on LC50 values in the range < 1 mg/L. However, limited aquatic toxicity data for SAS would suggest this classification statement over-estimates the aquatic toxicity of SAS.

A PNEC for soil-dwelling organisms of 0.3 mg/kg has been derived for SAS based on a study in which an EC10 of 3 mg/kg was recorded for the effect of LAS on soil microbial growth (Elsgaard et al., 2001a). An uncertainty factor of 0.1 has been multiplied by the EC10 value to derive this PNEC.

The use pattern for SAS would suggest that it is unlikely that SAS would be released to the environment in significant quantities.

12.4.2 Sodium sulphate

Combustion of SAS-containing fuel in motor vehicle engines will result in emission of sodium sulphate, a chemical ubiquitous in the environment with elements essential for life. No effect levels were available for sodium and sulphate, and freshwater PNECs of 2000 μg/L and 600 μg/L, respectively, have been derived based on the estimated amount of these salts present in rainfall (Munns, 2002; refer Section 8.3). Natural salt levels in catchment runoff is likely to contribute to total salts in catchment runoff due to geological weathering of sodium containing minerals. In marine waters, PNEC values for sodium and sulphate of 10 800 mg/L and 2700 mg/L, respectively, have been derived based on concentrations found in seawater (Munns, 2002; refer Section 8.3).

Table 10. Available ecotoxicological studies for sodium alkylbenzene sulfonate and structurally similar analogues

Chemical Name	CAS Number	Terrestrial plants and animals	Fish	Invertebrates	Algae	Biodegradation
Benzenesulfonic acid, mixed mono- and di-C15-30-alkyl derivatives, sodium salts (SAS)	78330-12-8	-	Rainbow trout	Waterflea *Daphnia magna*	-	-
Benzenesulfonic acid, C14-C24 branched and linear alkyl derivatives, calcium salt	115733-09-0	-	-	Waterflea *Daphnia magna*	*Pseudokirchneriella subcapitata*	-
Na-LAS (C10-13)	Unidentified	Earthworm and enchytraeids		-	-	-
LAS (mean C 11.5; other LAS unidentified)	Unidentified	*Collembolla*; 2 soil invertebrates & 5 microbial processes; 2 soil invertebrates; Aerobic heterotrophic bacteria; Microbial effects	Flathead minnow; Estuarine goby- (mean C11.5)	Copepod *Acartia tonsa;* Waterflea *Daphnia magna*	*Chlorella pyrenoidea*	Chemicals released from biodegradation of LAS
Benzene sulfonic acid, mono- and dialkyl derivatives, magnesium salts	71786-47-5	-	Flathead minnow Sheephead minnow	Waterflea *Daphnia magna*	*Pseudokirchneriella subcapitata*	-

13. Risk Characterisation

13.1 Environmental risk

This section provides a characterisation of risks to the environment from use of fuels containing SAS as an AVSR additive.

A hazard quotient (HQ) approach has been used to predict the hazard to terrestrial and aquatic organisms. To predict a low environmental risk, the ratio of PEC to PNEC needs to be 1 or less (i.e. HQ ≤ 1).

Overall, the use pattern of SAS indicates a low risk to terrestrial and aquatic environments due to the expected small and diffuse environmental releases.

13.1.1 Terrestrial risk

Most of the SAS used each year will be destroyed during combustion within internal combustion engine cylinders. SAS is not volatile and is unlikely to occur in the atmosphere in significant quantities. In water bodies, SAS is likely to biodegrade over time.

Following combustion, exhaust emissions include typical combustion products (i.e. CO, CO_2, steam, traces of hydrocarbons, hydrogen and soot), and sodium sulphate (Na_2SO_4). While a proportion may remain in the exhaust train, the majority is expected to be emitted. It is expected that only a small proportion of sodium sulphate discharged in vehicle exhaust emissions will be associated with fine particles (< 2% of total PM_{10}). Over time, much of the exhaust products are likely to disperse or react with moisture to form very dilute H_2SO_4, which is expected to fall as wet deposition (i.e. rainfall). The very minor acidity is likely to be buffered by natural alkalinity in the environment.

Levels of Na and SO_4 in soils (using the worst case example of urban runoff in Sydney) are unlikely to reach levels of concern. With the SAS use scenarios developed in Section 7, the estimated concentration of Na and SO_4 deposition from air to land is unlikely to result in unacceptable soil concentrations for soil-dwelling organisms or plants.

13.1.2 Aquatic risk

A $PNEC_{Freshwater}$ for SAS of 0.28 mg/L for LAS has been adopted from ANZECC/ARMCANZ (2000b), which was derived using a statistical approach. A $PNEC_{marine}$ of lower confidence of 0.1 µg/L has also been adopted from this source.

$PNEC_{freshwater}$ for Na^+ and SO_4^{2-} of 2 mg/L and 0.6 mg/L, respectively, have been derived based on estimated background concentrations in rainwater; refer Section 8.3). $PNEC_{Marine}$ for Na^+ and SO_4^{2-} are considerably greater than freshwater due to the higher natural concentration of these analytes and PNECs of 10 800 mg/L and 2700 mg/L, respectively, have been derived.

The PECs for Na^+ and SO_4^{2-} in stormwater derived from urban runoff occurring as a result of atmospheric washout in rainfall may approximate 24 µg SO_4/L and 12 µg Na/L in high SAS use areas (using the Sydney example from Section 8.4) in a

worst-case scenario. Hazard quotients for estimated Na^+ and SO_4^{2-} discharge to freshwater and marine ecosystems from urban runoff via an atmospheric washout source have been summarised below:

Table 11. Hazard Quotients for freshwater and marine ecosystems

Predicted Environmental Concentration, PEC (µg/L; refer Section 8.4)		Predicted No Effect Concentration, PNEC (µg/L; Refer Section 8.3)		Hazard Quotient, HQ	
Sodium (Na^+)					
Freshwater	24	$PNEC_{Freshwater}$	2000	$HQ_{Freshwater}$	0.012
Marine	< 24 dilution	$PNEC_{Marine}$	10 800 000	HQ_{Marine}	< 0.00001
Sulphate (SO_4^{2-})					
Freshwater	12	$PNEC_{Freshwater}$	600	$HQ_{Freshwater}$	0.02
Marine	< 12 dilution	$PNEC_{Marine}$	2 700 000	HQ_{Marine}	< 0.00001

The above table supports a conclusion of a low expected risk to the aquatic environment from atmospheric washout of Na^+ and SO_4^{2-}, thus supporting use of AVSR products containing SAS for the uses prescribed and the volumetric use rates estimated. The abovementioned HQ values are based on current estimated LRP demand (Present Use scenario), and risks are likely to increase marginally as demand for LRP aftermarket products increases over time. However, it has been assumed that SAS has 100% AVSR additive market share, which is unrealistic given that alternative AVSR products also likely to be available, and the PECs are likely to be much lower than estimated above. Sodium alkylbenzene sulfonate is unlikely to contribute significantly to the existing environmental releases and biogeochemical cycling of Na^+ and SO_4^{2-} in the environment from natural and other anthropogenic sources.

13.2 Occupational risk

A margin of exposure methodology is used frequently in international assessments to characterise risks to human health (European Commission, 1996). Where a NOAEL is available, the risk characterisation is conducted by comparing quantitative information on exposure to the NOAEL and deriving a Margin of Exposure (MOE) as follows:

1. Identification of the critical effect(s);
2. Identification of the most appropriate/reliable NOAEL (if available) for the critical effect(s);
3. Where appropriate, comparison of the estimated or measured human dose or exposure (EHD) to provide a Margin of Exposure:

   MOE = NOAEL/EHD;

4. Characterisation of risk, by evaluating whether the Margin of Exposure indicates a concern for the human population under consideration.

The MOE provides a measure of the likelihood that a particular adverse health 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 intra/inter species variability. Default uncertainty factors for intra- and inter-species variability are usually 10-fold each and so a MOE of less than 100 is usually considered a flag for concern.

13.2.1 Critical health effects

Sodium alkylbenzene sulfonate is of low acute toxicity via the oral and dermal routes. Based on limited analogue information, repeat dose toxicity also appears low. No mutagenic effects were observed. Data are presently unavailable to determine respiratory irritation, sensitisation, reproductive or carcinogenic effects.

The critical health effect from acute exposure is skin irritation. Although data are insufficient for hazard classification (NOHSC, 1999a), the potential for irritation from repeated or prolonged exposure to SAS remains. SAS also shows mild eye irritant properties but ocular effects are insufficient for hazard classification.

13.2.2 Occupational health and safety risks

Importation, storage and retail

Neither SAS nor AVSR products containing SAS are manufactured in Australia. Sodium alkylbenzene sulfonate is imported in finished products in low concentration (up to 0.471% v/v). Containers need not be opened prior to sale. The low probability of exposure renders the risks to importation and distribution personnel low.

Petrol stations and maintenance workshops

Its current usage as a fuel additive marketed to consumers renders the likelihood of occupational use low. If occupational use occurs, the likelihood of exposure to SAS by ingestion would be regarded as very low. Similarly, the expected low vapour pressure of SAS renders inhalation exposure to SAS vapours unlikely. Exposure to SAS is possible via dermal and ocular routes through the manual handling of AVSR product or fuels supplemented with SAS. However, the low concentration of SAS in additive products and fuels and the limited toxicological profile of SAS indicate that any possible dermal or ocular irritation is likely to be due to irritant properties of the petroleum diluents in the products or fuels rather than SAS itself (see Public Health Risk, Section 13.3.1 below).

Petrol station personnel, and in particular maintenance workshop personnel, may be exposed to atmospheric sodium sulphate particulates from combustion of SAS. Assuming as a worst case a fully closed workshop, the resultant occupational atmospheric level of sodium sulphate would reflect the maximum tailpipe emission of 0.1 mg/m^3, and for a typical 70 kg adult a daily dose of 9.5 μg/kg bw/day. This tailpipe emission of particulates is much lower than the NOHSC exposure standard for dust of 10 mg/m^3 (NOHSC, 1995). Also, this daily dose, assuming 100% absorption, is several orders of magnitude below that expected from drinking water intake based on NHMRC guidance values for Na^+ and SO_4^{2-} in drinking water based on aesthetics (taste) of 180 mg/L and 250 mg/L respectively. Therefore, the

risk from occupational exposure to particulate sodium sulphate as a result of combustion of SAS is low.

Car park and tollbooth personnel, professional drivers and road maintenance workers

Workers in the vicinity of operating automobiles are unlikely to be exposed to SAS but may be exposed to combustion products of SAS, mainly sodium sulphate in particulate form. This exposure is likely to be highly variable depending on the level of separation from the exhaust sources and traffic densities. In a study of the use of methylcyclopentadienyl manganese tricarbonyl (MMT) in Canadian fuels, personal manganese exposure data for Montreal taxi drivers show exposures that are lower than garage mechanics in the same study by an order of magnitude (Zayed et al., 1994). Therefore, exposure of Australian professional drivers to SAS and sodium sulphate is likely to be significantly less than automechanics and so the risk to local workers is considered low. Similarly, exposure to and risk associated with SAS and sodium sulphate for car park and road maintenance workers is considered low.

13.2.3 Uncertainties

Uncertainties exist in the assessment of risk to workers from the use of SAS as an AVSR agent. No Australian personal exposure data exist regarding exposures to SAS or SAS combustion products such as sodium sulphate in the workplace. However, this is of minor consequence to the risk analysis due to the low concentration of SAS in products and low expected exposures.

There are limitations in the toxicity data for SAS. For many toxicological endpoints, extrapolation from analogue data on sodium alkylbenzene sulfonates of shorter alkyl chain length and differing alkali metals were required. In particular, information on repeat dose and reproductive/developmental toxicity were poor. Moreover, certain toxicological data for SAS e.g. acute skin irritation, were unsuitable for classification against the NOHSC Approved Criteria. However, despite toxicity data limitations, occupational risk from SAS is limited due to the low risk of occupational exposure.

13.3 Public health risk

13.3.1 Acute effects

Direct public exposure to SAS may potentially occur primarily via the dermal route as a result of spills and splashes of LRP and aftermarket products. SAS itself is not expected to be a skin irritant at concentrations present in LRP. Dermal LD50 values for SAS analogues were > 2.0 g/kg bw (see Section 10.1.3) and an estimated dermal dose received during exposure to fuel under a worst-case scenario was many orders of magnitude lower at approximately 27 ng/kg bw.

SAS is likewise not expected to be a skin irritant following acute exposure in concentrations present in AVSR products. An estimated dermal dose received during exposure under a worst-case scenario was approximately 6.46 µg/kg bw, many orders of magnitude below the lowest animal dermal LD50 for SAS analogues of > 2 g/kg bw.

Ingestion exposure is possible when orally siphoning petrol containing SAS or from accidental ingestion by young children if aftermarket products are stored inappropriately in or around the home. A child (10 kg) ingesting one mL of a product containing 0.471% v/v SAS would receive an oral dose of 4.71 μL (0.45 μg/kg bw). This dose is many orders of magnitude lower than the oral LD50 for SAS of > 5 g/kg bw. Therefore, it can be concluded that there is a low risk of acute health effects in the general public as a result of oral or dermal exposure to SAS in LRP or in the aftermarket product.

Acute inhalation exposure of the public to SAS combustion products is possible. As with the worst case scenario of exposure of automechanics to combustion products in a fully enclosed workshop (Section 13.2.2), consumers may be similarly exposed in enclosed garages, albeit for shorter periods. As with occupational exposure of automechanics, the expected daily dose in this conservative scenario is several orders of magnitude below NHMRC guidance values for Na^+ and SO_4^{2-} in drinking water based on aesthetics (taste). Therefore, the risk to the public from acute exposure to particulate sodium sulphate as a result of combustion of SAS is low.

13.3.2 Chronic effects

Total exposure of the public to sodium sulphate, Na^+ or SO_4^{2-} is unlikely to be changed significantly from the use of SAS. The ingestion of food and water represents by far the greatest proportion of daily intake for these ions and these sources are not expected to change significantly as a result of the use of SAS.

Data in Table 7 (Section 8.7.2) show expected increases in particulate sodium sulphate in ambient air due to the use of SAS. In the absence of data on NOAELs for sodium sulphate, expected chronic human exposures can be compared cursorily to NHMRC guidance values for drinking water (based on aesthetics rather than health effects).

For the highest level (2004/2005) Scenario, where market share for SAS increases:

> Daily dose = 0.17 ng Na_2SO_4/kg bw/day = 57 pg Na^+/kg bw/day and 113 pg SO_4^{2-}/kg bw/day.
>
> NHMRC water quality guidelines = 180 mg Na^+/L = 5.15 mg Na^+/kg bw/day and 250 mg SO_4^{2-}/L = 7.14 mg SO_4^{2-}/kg bw/day (assuming water consumption of 2 L/day).

These expected chronic doses of Na^+ and SO_4^{2-} from SAS combustion are many orders of magnitude lower than doses expected from adherence to NHMRC aesthetic guidelines for drinking water. Therefore, the risk to the public is low.

13.3.3 Uncertainties

Like uncertainties associated with occupation risk assessment, uncertainties involved in the chronic public health risk assessment are derived from database limitations. There are a lack of suitable Australian air data upon which to base a realistic exposure assessment and very limited measured data that could be used to determine the contribution that SAS combustion might make to ambient air levels of sodium sulphate particulates.

14. Risk Management

14.1 Assessment of current control measures

According to the NOHSC *National Model Regulations for the Control of Workplace Hazardous Substances* (NOHSC, 1994d), 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, 1994a) 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).

14.1.1 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 a substance with a less hazardous substance or the same substance in a less hazardous form.

As indicated in Section 7.2.1, 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 refineries and terminals will become uneconomical at some point.

When bulk LRP is phased out, aftermarket addition of AVSR fuel additives rather than bulk treatment by the oil refineries and terminals will become the only option for motorists with vehicles designed to run on leaded petrol. Given the likelihood of a base population of vehicles for which mechanical alteration of engine components to run on unleaded petrol may be prohibitive e.g. vintage vehicles, the total elimination of AVSR agents from the Australian fuel market is unlikely and the use of SAS as an aftermarket additive may continue indefinitely.

Several AVSR additives that are potential substitutes for SAS are available on the Australian market (Section 1.1). However, users need to consider the efficacy, cost, health, safety and environmental effects of each in considering these as alternatives to SAS.

14.1.2 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.

The product containing SAS is not manufactured in Australia but imported in a ready-to-use form.

The controls for public and occupational users of aftermarket products containing SAS consist presently of graduated plastic containers with screw caps and steel drums with screw lids. Additionally, the AVSR product can be contained in a "Power Booster" device consisting of a reservoir and delivery lines within the engine bay by which the product can be automatically metered into the engine inlet manifold.

14.2 Hazard communication

14.2.1 Labels

The NOHSC *National Code of Practice for the Labelling of Workplace Substances* (NOHSC, 1994b) is applicable to labels for workplace substances. Labels of consumer products are required to comply with the *Standard for the Uniform Scheduling of Drugs and Poisons* (SUSDP) (NDPSC, 2003). SAS is currently not listed in the SUSDP and is not classified as a hazardous substance according to the NOHSC Approved Criteria (NOHSC, 1999a). However, general duty of care requirements call for the provision of basic safety information.

A label for the AVSR aftermarket consumer product (1 L) containing SAS was available for assessment. To assess the provision of basic information, the label was assessed for requirements under the NOHSC *National Code of Practice for the Labelling of Workplace Substances* (NOHSC, 1994). 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.

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 and/or dangerous goods class;
- Product name; and
- Details of manufacturer or importer.

The supplied label for the 1 L Morey's Upper Cylinder Lubricant AVSR product contains the product name, recommended treatment rates, an overseas contact address only, the risk phrase Harmful if Swallowed and the safety phrases Keep

Out of Reach of Children, Avoid Contact with Eyes or Prolonged Contact with Skin, Wash Hands after Use, If Swallowed, Call a Doctor Immediately and For Eye Contact, Flush with Water.

As a minimum, as required by the NOHSC Labelling Code (NOHSC, 1994b), the label should also contain:

- Details of local importer including telephone number;
- Reference to the Material Safety Data Sheets (MSDS).

14.2.2 MSDS

Material Safety Data Sheets 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, 1994d) 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 employees with potential for exposure to the chemical.

An MSDS for the AVSR product Morey's Upper Cylinder Lubricant (Morey Oil South Pacific Ltd, 2000) was available for assessment against the NOHSC *National Code of Practice for the Preparation of Material Safety Data Sheets* (NOHSC, 1994c). An MSDS for the SAS polybutene copolymer diluent and a proprietary product containing SAS used for formulating the AVSR product overseas were also available but were not assessed, as they do not relate to the AVSR product and are not in use in Australia.

The MSDS complied well with the NOHSC requirements (NOHSC, 1994c). However, the following data were lacking and should be included on the MSDS:

- Details of local importer including emergency telephone number.

The MSDS noted the presence of other potentially hazardous ingredients in the product. According to the MSDS, the main petroleum diluent in Morey's Upper Cylinder Lubricant is distillates, petroleum, hydrotreated light naphthenic (CAS 64742-53-6), present at < 75%. According to the NOHSC *List of Designated Hazardous Substances* (NOHSC, 1999b), this substance is a category 2 carcinogen requiring the risk phrase R45 – May Cause Cancer at a cut off of ≥ 0.1 %, subject to exemption of this hazard classification on the basis of a maximum level of DMSO extractability.

In the light of a lack of detailed information regarding formulation of this ingredient, it is not known whether the presence of this potentially carcinogenic naphthenic distillate is consistent with the statement in the MSDS under "Toxicological Information" that the material (product) has not been identified as a carcinogen.

14.2.3 National transportation regulations

Sodium alkylbenzene sulfonate is not listed in the Australian Code for the Transport of Dangerous Goods (ADG) Code. Morey's Upper Cylinder Lubricant does not meet the criteria for classification as a dangerous good (FORS, 1998) on the basis of physicochemical properties.

14.3 Environmental regulatory controls

This section provides information with reference to international initiatives on the environmental regulatory controls in Australia applicable to SAS. In summary, the management of environmental pollution and waste in Australia is regulated through individual State and Territory regulatory systems rather than at a national level and each State and Territory has legislative frameworks and strategies for managing emissions and environmental pollution to air, land and waters.

14.3.1 Control of major hazard facilities

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

14.4 Public health regulatory controls

SAS is not currently included and present guidelines do not warrant listing in the SUSDP. However, the MSDS indicates that consumer (aftermarket) products containing SAS also contain hazardous petrochemical ingredients. Since these may represent a potential public health risk, some public health regulatory controls may be warranted. These are outside the scope of this present assessment of SAS.

14.4.1 Air quality management

Australia

Potential air quality issues from combustion of fuels containing SAS include exhaust emissions of Na_2SO_4 (Mackie, 2003; see Appendix 2). Emissions of 'air toxics' (defined below) in Australia are regulated through individual state and territory regulatory systems rather than at a national level, and each state and territory has established legislative frameworks and strategies for monitoring and managing air quality. National-level strategies are or have been developed to allow consistent management of ambient air quality throughout Australia. Air toxics are gaseous, aerosol or particulate pollutants that are present in the air in low concentrations with characteristics such as toxicity or persistence so as to be 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 standards are set at a national level rather than State or Territory level. Recently the Australian Government introduced national fuel quality standards aimed at reducing the level of some air toxics in ambient air. Sulphur emissions are regulated by these standards.

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). Sulphate is not specifically included in either of these NEPMs. However, sulphur

dioxide emissions, which indirectly contribute significantly more SO_4 to the environment than direct SO_4 emission sources, are included in the ambient air NEPM. An inventory of emissions of SO_4 (as well as SO_2) from significant emission facilities is included in the National Pollutant Inventory NEPM (NEPC, 1998a). The NPI database contains air, land and water emission data for SO_4 from NPI reporting facilities and non-reporting facilities (including motor vehicles) for 2001-2002. Motor vehicles using SAS are expected to be minor contributors to the total emissions of SOx to the atmosphere.

International air quality management

Several international organisations have introduced regulations or policies that aim to limit the exposure of the general public to air pollutants. The OECD has implemented the Advanced Air Quality Indicators and Reporting Project in OECD member countries, including Australia (OECD, 1999). The project focuses on six major urban air pollutants, including sulphur dioxide. 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 outlines a strategy for addressing cumulative health risks from identified HAPs in urban areas (USEPA, 1999). Sulphur compounds are not included in this strategy. In Canada, a range of air toxics including SO_2 are measured and analysed within the National Air Pollution Surveillance (NAPS) Network. The NAPS network was established in 1969 to monitor and assess the quality of ambient air in Canadian urban areas. In the United Kingdom, sulphur dioxide emissions are managed by the Department of Environment, Transport and Regions (UKDETR), which established a benchmark standard for SO_2 in air. The New Zealand Air Toxics Program establishes air quality standards for eight common pollutants including SO_2.

14.4.2 Aquatic ecosystem management

The Australian water quality guidelines (ANZECC/ARMCANZ, 2000), established under the National Water Quality Management Strategy, provide water and sediment quality guidelines (trigger levels) for freshwater and marine ecosystems throughout Australian States and territories. The guidelines provide a decision-tree framework for the assessment and management of risks from chemicals to water and sediment quality. No trigger values are available for SAS, however, ANZECC/ARMCANZ (2000) provide ambient trigger levels for LAS compounds for the protection of freshwater and marine ecosystems of 280 μg/L and 0.1 μg/L, which have been adopted for this assessment. Each State and Territory has legislative frameworks and strategies for managing water pollution.

Discharges of SAS or combustion products in runoff are unlikely to pose an unacceptable risk to the aquatic environment.

14.4.3 Disposal and waste treatment

Each Australian State and Territory provides statutory controls on waste generation and management. SAS-containing materials classified as wastes should be sent to licensed waste disposal contractors in accordance with State and Territory requirements. No specific waste disposal guidelines, standards or management issues were identified for SAS wastes. Due to the toxicity of SAS, care should be exercised in disposing of contaminated wastes to avoid pollution of the environment. The MSDS for Morey's Upper Cylinder Lubricant & Combustion

Chamber Cleaner (Morey Oil South Pacific Ltd, 2000) recommends that waste disposal agencies should be consulted for reporting and disposal requirements.

Waste disposal licences are required to be held by waste contractors managing SAS wastes. For example, in NSW, transporters conveying SAS waste in quantities greater than 200 kg per load, or waste facilities treating SAS waste, require a licence under the Protection of the Environment Operations Act (1997) issued by the NSW Environment Protection Authority (EPA).

14.5 Emergency procedures

Fire and spill responses for SAS are included in MSDS for aftermarket product Morey's Upper Cylinder Lubricant & Combustion Chamber Cleaner (Morey Oil South Pacific Ltd, 2000). Recommendations from Morey Oil South Pacific Ltd. for dealing with fires recommend the use of carbon dioxide, dry chemical or foam for extinguishment, self-contained breathing apparatus, and water only for cooling containers. The MSDS warn against exposure to carbon monoxide and asphyxiants.

With regards to accidental spills, the MSDS recommends:

- Containing release;
- Preventing entry into sewers and waterways;
- Picking up free liquid for recycling or disposal; and that
- Residual liquid can be absorbed with inert material.

15. Discussion and Conclusions

Sodium alkylbenzene sulfonate is used in Australia as an AVSR additive in lead replacement petrol (LRP). AVSR fuel additives are added to fuel to prevent excessive valve seat wear and consequent recession into the automotive engine head. Until its reduced use in fuel, tetraethyl lead was the most common AVSR additive.

With the national reduction of lead in petrol, there are now four types of AVSR additives presently marketed in Australia. Methylcyclopentadienyl manganese tricarbonyl (MMT)-based, phosphorus-based and sodium-based AVSR agents are presently being assessed by NICNAS as Priority Existing Chemicals and a potassium-based AVSR additive was assessed by NICNAS as a New Industrial Chemical. These AVSR additives are delivered either pre-blended into LRP or are available as an aftermarket fuel supplement for addition to unleaded fuel by consumers.

Due to commercial sensitivities of information on market share for individual AVSR agents, exposure and risk assessments for each individual AVSR agent assume 100% market share. Additionally, given that the use of AVSR additives is governed by a declining population of older vehicles requiring these fuel additives, risk assessments were conducted under two separate scenarios based on AVSR additive use patterns. The first scenario "Present Use" assumes a continuation of the present LRP market of 2500 ML per year with 90% of AVSR additive delivered in bulk LRP and 10% delivered as aftermarket fuel additives. The second scenario "2004/2005" assumes a decline of the LRP market to 1000 ML with the AVSR additive delivered totally as an aftermarket fuel additive. These scenarios are based on motor vehicle statistics and forecasts from the Australia Bureau of Statistics and 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 accordingly.

Sodium alkylbenzene sulfonate is manufactured overseas and imported only in finished AVSR products for use as aftermarket additives by consumers. The Upper Cylinder Lubricant product contains SAS at up to 0.471% v/v and at a recommended treat rate of 1 L Upper Cylinder Lubricant for 650 L fuel, the final concentration of SAS in fuel is < 0.001% v/v. Assuming SAS has 100% of the AVSR product market, around 400 000 L of product containing 2000 L SAS is currently imported to Australia per year. With a phase-out of bulk LRP, and assuming 100% market share, this is expected to rise to approximately 1.54 ML of product containing 8000 L of SAS.

15.1 Health hazards and risks

Health hazards addressed in this report relate to SAS and particulate sodium sulphate, the predominant SAS combustion product.

Animal toxicity studies of SAS and alkylbenzene sulfonate analogues show that SAS is not acutely toxic by oral or dermal exposure. Acute irritation studies reveal irritant properties, however data are insufficient for classification of SAS as a

hazardous substance (NOHSC, 1999a). Repeat dose and reproductive toxicity data are generally lacking. There are no published case reports, epidemiology or other studies addressing the human health effects of SAS. Although not meeting the NOHSC Approved Criteria for Classifying Hazardous Substances (NOHSC, 1999a), on the basis of possible skin irritation, a safety phrase Avoid Contact with Skin, or equivalent, is appropriate.

The use of SAS as a fuel additive is associated with the generation of combustion products, predominantly sodium sulphate in particulate form. Sodium sulphate is not a hazardous substance (NOHSC, 1999b) and does not have an associated atmospheric exposure standard (NOHSC, 1995). Modelling reveals the expected release of and exposure to only very low amounts of sodium sulphate from the combustion of SAS, orders of magnitude lower than amounts of sodium sulphate as dissociated Na^+ and SO_4^{2-} ions expected from normal ingestion of water and foodstuffs.

15.2 Environmental hazards and risks

Limited ecotoxicity data were available for SAS. Environmental information was therefore also considered for LAS analogues.

Numerous studies of aquatic toxicity have been performed using LAS compounds and the results are highly variable depending on the analogue, species and test conditions. Linear alkylbenzene sulfonates of longer alkyl chain length are generally more toxic (Danish EPA, 2001). The relatively long carbon length of SAS suggest a relatively high toxicity to aquatic organisms; however, the toxicity test reports for SAS indicate low toxicity up to the point of water solubility. It is suspected that above a certain carbon chain length, water solubility, and thus bioavailability and toxicity, decline. However, there are insufficient data to determine the chain length above which toxicity decreases.

In consideration of this uncertainty, the trigger value for LAS derived by ANZECC/ARMCANZ (2000a) for the protection of freshwater organisms of 280 μg/L has been adopted as a predicted no effect concentration (PNEC) for SAS for this assessment. The marine trigger value for LAS derived by ANZECC/ARMCANZ (2000c) of 0.1 μg/L is also adopted as a PNEC for this assessment.

There is currently no environmental hazard classification system in Australia. In accordance with the OECD Globally Harmonized System of Classification and Labelling of Chemicals (OECD, 2002), LAS compounds in general would be classified as Acute 1 Very Toxic to Aquatic Life, based on LC50 values in the range < 1 mg/L. However, limited aquatic toxicity data for SAS would suggest this classification statement over-estimates the aquatic toxicity of SAS.

A PNEC for soil-dwelling organisms of 0.3 mg/kg has been derived for SAS based on a study in which an EC10 of 3 mg/kg was recorded for the effect of LAS on soil microbial growth (Elsgaard et al., 2001a). An uncertainty factor of 0.1 has been multiplied by the EC10 value to derive this PNEC.

Existing diffuse use and exposure patterns for SAS as a fuel additive, its subsequent degradation through combustion and generally short persistence in the environment indicate that for existing use patterns, aquatic and terrestrial

organisms are unlikely to be exposed to SAS at levels of concern. A low environmental risk is predicted.

Sodium sulphate, a by-product from combustion of SAS, is naturally occurring and ubiquitous in the environment, as are the constituent ions. It is an essential nutrient of plants and animals. Environmental exposure to sodium or sulphate compounds arising from combustion of SAS will mostly occur through the gaseous phase. Eventually, these will deposit to land and waters through wet or dry deposition. The emission of sodium sulphate into the environment from use of fuels containing SAS is unlikely to develop to levels of concern for terrestrial or aquatic environments. The emission of sulphate from SAS combustion is likely to be only a small fraction of that emitted from other sources in Australia. As such, the findings of this assessment have not identified any significant risk to the environment given the considered current use pattern of fuels containing SAS as an AVSR agent.

15.3 Occupational health and safety risks

Neither SAS nor AVSR products containing SAS are manufactured in Australia. Sodium alkylbenzene sulfonate is imported in finished products in low concentration. Containers need not be opened prior to sale. The low probability of exposure renders the risks to importation and distribution personnel low.

Its current usage as a fuel additive marketed to consumers renders the likelihood of occupational use low. Exposure to SAS is possible via dermal and ocular routes through the manual handling of AVSR product or fuels supplemented with SAS. However, the low concentration of SAS in additive products and fuels and the limited toxicological profile of SAS indicate that any possible dermal or ocular irritation is likely to be due to irritant properties of the petroleum diluents in the products or fuels rather than SAS itself. Therefore, the risk from occupational exposure to SAS is low.

Petrol station personnel, and in particular maintenance workshop personnel, may be exposed to sodium sulphate particulates from combustion of SAS. Assuming as a worst case a fully closed workshop, modelling of tailpipe emissions shows atmospheric levels of sodium sulphate particulates much lower than the NOHSC exposure standard for dust of 10 mg/m^3 (NOHSC, 1995). Also, the daily dose from occupational exposure assuming 100% absorption is several orders of magnitude below that expected from NHMRC guidance values for Na^+ and SO_4^{2-} in drinking water based on aesthetics (taste). Therefore, the risk from occupational exposure to particulate sodium sulphate as a result of combustion of SAS is low.

Similarly, for workers such as car park and tollbooth attendants, professional drivers and road maintenance workers in the vicinity of operating automobiles, the risk from exposure to SAS and sodium sulphate particulates resulting from SAS combustion are low.

15.4 Public health risks

Direct public exposure to SAS may potentially occur primarily via the dermal route as a result of spills and splashes of LRP and aftermarket consumer products. An estimated dermal dose of SAS likely following exposure to fuel under a worst-case scenario was many orders of magnitude lower than dermal LD50 values for SAS

analogues. Also, SAS itself is not expected to be a skin irritant at concentrations present in LRP or in AVSR products.

Acute exposure via ingestion is possible when orally siphoning petrol containing SAS or from accidental ingestion by young children if aftermarket products are stored inappropriately in or around the home. However, under a worst case scenario of ingestion by a child, the oral dose a child would expect to receive is many orders of magnitude lower than the oral LD50 for SAS. Therefore, it can be concluded that there is a low risk of acute health effects in the general public as a result of oral or dermal exposure to SAS in LRP or in the aftermarket product.

Acute exposure of the public to SAS combustion products i.e. sodium sulphate particulates in enclosed garages, is possible. However, as with occupational exposure of automechanics, the expected daily dose of sodium sulphate particulates from SAS combustion in this scenario is low. Therefore, the risk to the public from acute exposure to particulate sodium sulphate as a result of combustion of SAS is low.

The combustion of SAS will contribute to particulate sodium sulphate in ambient air. However, exposure of the public to sodium sulphate, Na^+ or SO_4^{2-} is unlikely to be changed significantly by the use of SAS. Compared to intake from ambient air, the ingestion of food and water represents by far the greatest proportion of daily intake for these ions and these sources are not expected to change significantly as a result of the use of SAS. Therefore, the risk to the public from increases in particulate sodium sulphate due to the combustion of SAS is low.

15.5 Data gaps

There are limitations in the toxicity data for SAS. For many toxicological endpoints, extrapolation from analogue data on sodium alkylbenzene sulfonates of shorter alkyl chain length and differing alkali metals were required. In particular, information on repeat dose and reproductive/developmental toxicity were lacking. Moreover, certain toxicological data for SAS e.g. acute skin irritation, were unsuitable for classification against the NOHSC Approved Criteria.

Uncertainties exist in the assessment of risk to local workers and to the public from the use of SAS as an AVSR agent. No Australian personal exposure data exist regarding exposures to SAS or SAS combustion products such as sodium sulphate.

Uncertainties involved in the chronic public health risk assessment are derived from database limitations. There is a lack of suitable Australian air monitoring data upon which to base a realistic exposure assessment and there is very limited data that could be used to determine the contribution that SAS combustion might make to ambient air levels of sodium sulphate particulates.

16. Recommendations

This section provides recommendations arising from the Priority Existing Chemical assessment of SAS to improve environmental, occupational and public health as a result of its use as an AVSR additive.

16.1 Classification

Sodium alkylbenzene sulfonate is not considered a hazardous substance according to the NOHSC *Approved Criteria for Classifying Hazardous Substances*. However, animal studies suggest that prolonged or repeated exposure may produce skin irritation. It is therefore recommended to importers of SAS that MSDS and labels for products containing SAS contain the safety phrase Avoid Contact with Skin, or equivalent.

16.2 Hazard communication

16.2.1 MSDS

This assessment found that the MSDS for products containing SAS did not conform completely to the requirements of the NOHSC *National Code of Practice for the Preparation of Material Safety Data Sheets* (NOHSC, 1994b). In order to ensure conformity with this code, it is recommended that importers of SAS products review their MSDS for compliance and ensure the following information is included:

- Details of local importer including emergency telephone number;
- The Safety Phrase Avoid Contact with Skin, or equivalent.

16.2.2 Labels

This assessment found that the sample label for SAS products did not conform completely to the requirements of the *National Code of Practice for the Labelling of Workplace Substances* (NOHSC, 1994b). In order to ensure conformity with this code, it is recommended that importers of SAS products review their labels for compliance and ensure the following information is included:

- Details of local importer including telephone number;
- The Safety Phrase Avoid Contact with Skin, or equivalent.

16.3 Packaging

To prevent backflow and spillage of SAS (and other product ingredients), it is recommended to importers that consumer products designed to be added directly to fuel tanks or to "Powerbooster" reservoirs within engine bays should be packaged in containers with spouts of sufficient length to ensure good insertion of the spout into the reservoir or fuel filler. In the case of multi-use products, these spouts should also be resealable.

17. 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 SAS, specific circumstances include:

- use of SAS preblended in bulk LRP or other bulk transport fuels;
- the manufacture of SAS beginning in Australia;
- additional information becoming available to the introducers as to adverse health and/or environmental effects of SAS or SAS analogues such as those identified in this assessment. Such information includes the results of further mammalian repeat dose and reproductive/developmental toxicity, acute fish toxicity, water solubility and biodegradability testing of alkaryl sulfonates by the American Chemistry Council Health, Environmental and Regulatory Task Group (HERTG) as indicated under the USEPA Chemical Right-To-Know Initiative.

The Director (Chemicals Notification and Assessment) must be notified within 28 days of the introducer 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 2004/2005

The weekly fill-up rate for vehicles using lead replacement petrol (LRP) was calculated from sales volumes of lead and lead replacement petrol (LRP) in July 2000 to June 2001 of 2937.36 ML (Department of Industry, Science and Research, 2001) and from the number of vehicles using leaded petrol at 31 March 2001 of 2 904 342 (Australian Bureau of Statistics Motor Vehicle Census, 2001) as:

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

= 19.4 litres/week/vehicle

LRP volumes in 2004/2005 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 2004/2005.

Appendix 2 – High Temperature Combustion Products of Sodium Alkylbenzene Sulfonate

In the absence of data, NICNAS commissioned expert advice on the expected combustion and final tailpipe emissions by-products of SAS as a consequence of the high temperature combustion environment likely to occur under engine operating conditions.

The advice was sought because of anticipated public and occupational exposure to combustion by-products of SAS. The identification of thermal combustion products was considered a prerequisite for determining the risks associated with the use of the chemical.

Choice of 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 below.

Identity of the AVSR additive

Sodium alkylbenzene sulfonate comprises a mixture of mono- and di- C15-C30 alkyl derivatives (CAS 78330-12-8) (see Section 4). This active ingredient of the AVSR product Morey's Upper Cylinder Lubricant and Combustion Chamber Cleaner is contained in mineral oil identified as 1-propene, 2-methyl-, homopolymer $(C_4H_8)x$, x = 17 (CAS 9003-27-4). The concentration of active ingredient in the Morey's Upper Cylinder Lubricant is stated to be between 0.3147-0.471% v/v and the treat rate is 1 L of Morey's Upper Cylinder Lubricant to 650 L unleaded fuel.

Literature survey

Electronic searching of Chemical Abstracts was conducted using search terms including "Morey's Upper Cylinder Lubricant and Combustion Chamber Cleaner", "Sodium Anti-Valve Seat Recession Additives", "combustion or thermal decomposition of sodium sulfonates". Internet searching was also conducted using the Google search engine.

No relevant references were found using search terms including "Morey". A patent was found for a sodium AVSR additive similar to the present formulation containing sodium monoalkylbenzenesulfonate, polyisobutenyl polyalkylenepolyamine and mineral oil (Johnston and Dorer, 1987). No mention was made in the patent application of tailpipe emissions.

The effectiveness of potassium- and sodium-based alkenylsulfonates and naphthenates in reducing VSR has been studied in Thailand, which has now replaced leaded petrol with unleaded petrol containing these alkali-based AVSR agents. It has been concluded that a treat level of 10 mg of potassium or sodium per kg of fuel is required (Akarapanjavit and Boonchanta, 1996). Degradation of automobile turbochargers using petrols containing sodium- and potassium-based additives in a European country which has banned leaded petrol has been reported to result from the corrosive effects of hot alkali metal sulphates produced in the combustion process (Stott et al., 2000). This reference is most relevant because, although the exact nature of the Na or K based sulphur-containing additives have

not been specified, they are almost certainly sulfonates, universally employed because of their detergent role. This identifies alkali sulphate salts as products of combustion.

Metal sulphates have also been detected in significant amounts in the condensed aqueous phase vapour of vehicle exhausts in Brazil operating with unleaded petrol (containing 22% hydrated ethanol) (Colombara et al., 1999). While it is not argued that the sulphate salts arise solely from a sulfonate additive (as Brazilian petrol can contain by law up to 1000 ppm S as organosulphur (Nogueira, 2002)), this study shows that combustion leads to the emission of significant quantities of metal sulphates. Studies of air in a California tunnel bore of gaseous and particulate emissions from light vehicles operating on gasoline containing an average of 12 ppm S by weight detected a sulphate emission level of 2.12±0.43 mg/kg (as SO_4^{2-}) (Kirchstetter et al., 1999). Sulphates comprised only about 2% of the total PM2.5 particulate emission. The bulk of fine particles was found to be comprised of carbon.

Material safety data sheets for the combustion products of sodium benzene sulfonates typically list carbon dioxide, carbon monoxide and/or oxides of sulphur as potentially hazardous products (Cytec, 1998). However, a study of the thermal decomposition of sodium tetrapropylenebenzenesulfonate found $Na_2S_2O_7$ as a major product (Boros et al., 1963). Also, a quantitative method for microdetermination of alkali metal sulfonates involves the oxidation of the sulfonate to produce the alkali metal sulphate which is then potentiometrically titrated (Campiglio, 1990). In a method for incineration of waste containing sulfinic acid groups, alkali earth acetates are used to convert the waste sulphur into alkali earth sulfonates (Yahata, 1991). These are then incinerated to form the alkali earth sulphate without emission of sulphur oxides. Similar chemistry would be expected for alkali metals.

Finally, thermal decomposition studies on molten sodium sulphate (Halle and Stern, 1980) indicate that it is thermally stable up to about 1100°C above which temperature it dissociates by

$$Na_2SO_4(l) \rightarrow 2Na(g) + SO_2(g) + O_2(g) \qquad (1)$$

Likely combustion products of Morey's upper cylinder lubricant

The mineral oil component (polyisobutylene) which is a hydrocarbon polymeric molecule will undergo combustion under engine conditions to products similar to those produced by the petrol itself, viz., principally carbon dioxide and steam with lesser amounts of carbon monoxide and small amounts of C1, C2 hydrocarbons, hydrogen and some soot. It is the active additive, the SAS, which can potentially introduce new products into the tailpipe emissions.

In the specifications provided by the manufacturers, treat rate was expressed in %v/v. No density information was provided. However, if we assume that the density of LRP is 0.74 kg/L and assume that the density of the lubricant is similar to that of a heavy diesel oil (~0.96 kg/L) then for the stated treat rate the active compound is present at approximately 8 ppm by weight. The maximum possible sodium or sulphur content would arise if SAS were taken as the C15 monoalkyl derivative which would have the molecular weight of 390 g/mol. In this case the maximum Na content is 0.5 ppm (approx.) and the maximum S content 0.7 ppm (approx.) in the fuel at the recommended level of treatment. This maximum sulphur content is very much lower than the stated maximum sulphur content in LRP of 500 ppm supplied in Australia by BP Australia Ltd (BP Oil International Ltd.). Thus even if all the sulphur present in the active ingredient were released from the tailpipe

as sulphur dioxide, it would be negligible compared with that emanating from the sulphur in the fuel itself.

However, the sulfonate group $–SO_3Na$ and the free sulfonic acid group $–SO_3H$, are considered by organic chemists to be good "leaving groups" (Fieser and Fieser, 1953). That is to say, the SO_3Na group in the salt or SO_3H group in the free acid can readily be removed intact from the benzene ring. Thus, sulfonates and sulfonic acids can be readily desulfonated thermally or by steam treatment, favourable conditions for which are present under combustion conditions in the engine. Thus, under combustion conditions, it is very likely that the SAS will be desulfonated and the SO_3Na group oxidized to sodium sulphate. Molten sodium sulphate would be expected to undergo slow decomposition according to reaction (1) but in the short residence time in the engine, much of the sulphate should survive, possibly to be deposited in part in the vicinity of the exhaust valve as expansion of the combustion products leads to rapid cooling. Some of the sulphate will be emitted as solid sodium sulphate. If all of the sodium were emitted from the tailpipe as $Na_2SO_4(s)$ then we would expect this solid to be emitted at the rate of 1.5 mg/kg of LRP fuel. Sodium is the limiting element in determining this level of emission. About 0.3-0.4 ppm S is potentially available for emission, probably as SO_2. However, as stated above, this level of sulphur is negligible compared with the sulphur level in the original fuel. For an average fuel consumption of 8 L/100 km, there will be approximately 10 m^3 of exhaust gases per litre of petrol consumed. Hence the emission from the tailpipe of $Na_2SO_4(s)$ is, at maximum, 0.1 mg/m^3. Atmospheric dilution will reduce this level, so that an appropriate dilution factor should be applied.

The alkyl side chain(s) would also be expected to be cleaved off readily from the benzene ring under combustion conditions. The radicals present in combustion in the engine, viz., H, OH and O, readily react with the side chains leading to the usual combustion products of CO_2, steam, CO, traces of light hydrocarbons and hydrogen together with some soot. The aromatic ring is somewhat less readily oxidized and some benzene can always be detected in the exhaust gases from LRP. However, the maximum level of benzene that could arise from SAS in the unlikely event that each aromatic ring formed benzene would be 1.6 mg/kg of fuel, negligible compared with the approximately 20 g/kg benzene in LRP petrol (BP Oil International Ltd.).

Conclusions

The active ingredient in Morey's Upper Cylinder Lubricant and Combustion Chamber Cleaner, a mixture of sodium mono- and di- alkyl benzenesulfonates, at the stated treatment level for LRP would produce a tailpipe emission of solid sodium sulphate at a maximum level of 1.5 mg/kg of fuel consumed or a maximum emission level of 0.1 mg/m^3 of exhaust gas. The active ingredient is also likely to produce traces of sulphur in the form of sulphur dioxide and benzene but the level of these from the additive would be negligible compared with the level of sulphur and benzene in the original fuel (LRP).

Appendix 3 – Classification under the Globally Harmonized System for Hazard Classification and Communication

In this report, sodium alkylbenzene sulfonate has been assessed against the NOHSC *Approved Criteria for Classifying Hazardous Substances* (Approved Criteria) (NOHSC, 1999a) and, in the case of physicochemical hazards, the *Australian Code for the Transport of Dangerous Goods by Road and Rail* (ADG Code) (FORS, 1998). However, classifications under the Globally Harmonized System (GHS) for Hazard Classification and Communication (OECD, 2002) will come into force when the GHS is adopted by the Australian Government and promulgated into Commonwealth legislation. GHS documentation is available at

http://www.unece.org/trans/danger/publi/ghs/officialtext.html

Based on a literature survey of SAS analogues, in accordance with the OECD Globally Harmonized System of Classification and Labelling of Chemicals (OECD, 2002), LAS compounds in general would be classified as Acute 1 Very Toxic to Aquatic Life based on LC50 values in the range < 1 mg/L. However, only limited aquatic toxicity data are available for SAS and these would suggest this classification statement over-estimates the aquatic toxicity of SAS.

Therefore, presently, data for SAS do not meet or are insufficient to classify against GHS criteria for physicochemical hazards, toxicity or ecotoxicity.

References

Akarapanjavit N & Boonchanta P (1996) Valve seat recession and protection due to lead phase out in Thailand. Society of Automotive Engineers, [Special Publication] SP 1996, SP-1214, 147-154.

AIHW (Australian Institute of Health and Welfare) (2002) National Hospital Morbidity Database. <http://www.aihw.gov.au/hospitaldata/datacubes/index.html#nhmd>. Accessed 2002.

ANZECC/ARMCANZ (Australian and New Zealand Environment and Conservation Council and the Agriculture and Resource Management Council of Australia and New Zealand) (2000b). Irrigation and general water uses, stock drinking water, aquaculture and human consumers of aquatic foods. Australian and New Zealand guidelines for fresh and marine water quality primary industries — rationale and background information. (Chapter 9). volume 3, paper No. 4. Australia and New Zealand Environment and Conservation Council and the Agriculture and Resource Management Council of Australia and New Zealand. National Water Quality Management Strategy. <http://www.ea.gov.au/water/quality/nwqms/volume3.html > Accessed 2003.

ANZECC/ARMCANZ (Australian and New Zealand Environment and Conservation Council and the Agriculture and Resource Management Council of Australia and New Zealand) (2000c). Australian and New Zealand guidelines for fresh and marine water quality, volume 2, aquatic ecosystems - rationale and background information (Chapter 8). Australian and New Zealand Environment and Conservation Council (ANZECC) and Agriculture and Resource Management Council of Australia and New Zealand (ARMCANZ). Paper No. 4. National Water Quality Management Strategy. <http://www.ea.gov.au/water/quality/nwqms/volume2.html > Accessed 2003.

ANZECC/ARMCANZ (Australian and New Zealand Environment and Conservation Council and the Agriculture and Resource Management Council of Australia and New Zealand). (2000a). Australian and New Zealand guidelines for fresh and marine water quality. National Water Quality Management Strategy, Environment Australia, Department of the Environment and Heritage, Canberra. <http://www.ea.gov.au/water/quality/nwqms/volume1.html> Accessed 2003.

Ashby K & Routely V (1996) Childhood domestic chemical and plant poisonings. HAZARD, **28**:1-7. Victorian Injury Surveillance System, Monash University Accident Research Centre.

Australian Bureau of Statistics (1998) 9309.0 Motor vehicle census 31 October 1997. Canberra, Australian Bureau of Statistics.

Australian Bureau of Statistics (2001) 9309.0 Motor vehicle census 31 March 2001. Canberra, Australian Bureau of Statistics.

Australian Bureau of Statistics (2002) 9309.0 Motor vehicle census 31 March 2002. Canberra, Australian Bureau of Statistics.

Australian Petroleum Gazette (1999) Petroleum Gazette **34**: 51.

Ayers G (1992) Latrobe Valley rainwater composition study. Report to the State Electricity Commission of Victoria, Melbourne. (Cited in NSW EPA, 1994).

Ayers G & Manton M (1991) Rainwater composition at two BAPMoN regional stations in SE Australia. Tellus 43B, Copenhagen, pp. 379-389. (Cited in NSW EPA, 1994).

Ayers G & Yeung K (1993) Atmospheric acidity and deposition in Asia, Melbourne (Cited in NSW EPA, 1994).

Barlow PL (1999) The lead ban, lead replacement petrol, and the potential for engine damage. Anti-Corrosion Methods and Materials, **46**

Bast C (1991) Toxicity summary for sulphate. Toxicity profiles. Risk Assessment Information System. Oak Ridge Reservation Environmental Restoration Program, Oak Ridge, TN. June.

Benbarka A (2000) Lead phase-out in sub-saharan countries. The case of three gasoline-importing countries: Benin, Burkina Faso and Senegal. Washington D.C., USA, The World Bank.

Biosearch Incorporated (1982a) Project Number 82-3231A Acute oral toxicity – rats. Pennsylvania, USA.

Biosearch Incorporated (1982b) Project Number 82-3231A Primary eye irritation - rabbits. Pennsylvania, USA.

Biosearch Incorporated (1982c) Project Number 82-3231A Primary skin irritation - rabbits. Pennsylvania, USA.

Boros M & Lorant B (1963) Seifen-Oele-Fette-Wachse. **89;89**(17;18), 531-4, 555-8, Abstract only from Chem. Abstracts CAN 60:6292.

BP Oil International Ltd. Technical Data Sheet. Lead replacement petrol. http://tds-au.bp.com/bpglis/oztds3.nsf/d9626f8424d26a26ca256a790031248a/e04e45278d0e4214ca256b670015f9af?OpenDocument. Accessed 2003.

CNG Installations (1985) Results of dynometer and exhaust analyser tests. Letter to Moreys Oil South Pacific. 23 July 1985.

Campiglio A (1990) The potentiometric microdetermination of sulfur in alkali metal sulfonates and alkyl sulfates after combustion in a modified oxygen flask. Microchem. J. **41**: 132-138.

CCREM (Canadian Council of Resource and Environment Ministers) (1987) Canadian water quality guidelines. Canadian Council of Resource and Environment Ministers, Inland Water Directorate, Environment Canada, Ottawa.

Christiansen PD, Brozek M & Hansen BW (1998) Energetic and behavioural responses by the common goby, Pomatoschistus microps (Kroyer), exposed to linear alkylbenzene sulphonate. Environ. Toxicol. Chem. **17** (10): 2051-2057.

CLER (Council for LAB/LAS Environmental Research) (1996a). LAS Biodegradation and Safety in Rivers and Streams. Fact Sheet. Internet: <http://www.cler.com /facts/rivers.html> Accessed 28 April 2003.

CLER (Council for LAB/LAS Environmental Research) (1996b) LAS biodegradation and safety under anaerobic conditions. Fact sheet. <http://www.cler.com /facts/anaerobic.html> Accessed 28 April 2003.

CLER (Council for LAB/LAS Environmental Research) (1999) LAS biodegradation and safety in sediments. Fact sheet. <http://www.cler.com/facts/sediments.html> Accessed 28 April 2003.

Colombara R, Massaro S & Tavares MFM (1999) Exploring the versatility of capillary electrophoresis for the analysis of ionic species in vehicular emission. Anal. Chim. Acta. **388**: 171-180.

Connell DW & Hawker D (1986) Predicting the distribution of persistent organic chemicals in the environment. Chemistry in Australia, December 1986, pp 428-431.

CRC Press (1976) Handbook of Chemistry and Physics, 57 th edition, p F-121.

Cresswell DG, Baldock GA, Chasseaud LF & Hawkins DR (1978) Toxicology studies of linear alkylbenzene sulphonate (LAS) in rhesis monkeys. II. The disposition of [^{14}C] LAS after oral or subcutaneous administration. Toxicology **11**: 5-17

Cytec (1998) MSDS for AEROSOL DPOS-45 Surfactant http://www.cytec.com/msds/SMS/TPSDWEB/pdf/mU/05000/05234.pdf Accessed 2003.

Daniels JI (1988) Evaluation of military field-water quality. Volume 4. Health criteria and recommendations for standards. Part 1. Chemicals and properties of military concern associated with natural and anthropogenic sources. AD UCRL-21008 4 (1).

Danish EPA (2001) Environmental and health assessment of substances in household detergents and cosmetic detergent products. Danish Environment Protection Agency. CETOX. Environmental Project No. 615.

Department of Industry, Science and Research (2001) Automotive gasoline sales in Australia. Canberra, Australian Government.

DWAF (Department of Water Affairs and Forestry) (1996) South African water quality guidelines, 2nd ed., 5: Agricultural use: Livestock watering. Pretoria, CSIR Environmental Services.

Eaton FM (1942) Toxicity and accumulation of chloride and sulphate salts in plants. Journal of Agricultural Research **64** (7): 357-399. U.S. Government Printing Office.

ECETOC (1996) Aquatic toxicity testing of sparingly soluble, volatile and unstable substances. European Centre for Ecotoxicology and Toxicology of Chemicals. Monograph No. 26 September 1996. Brussels, ECETOC.

Elsgaard L, Petersen SO & Debosz K (2001a) Effects and risk assessment of linear alkylbenzene sulfonates in agricultural soil. 2. Effects on soil microbiology as influenced by sewage sludge and incubation time. Environ. Toxicol. Chem. **20** (8): 1664-1672.

Elsgaard L, Petersen SO & Debosz K (2001b) Effects and risk assessment of linear alkylbenzene sulfonates in agricultural soil. 2. Short-term effect on soil microbiology. Environ. Toxicol. Chem. **20** (8): 1656-1663.

Environment Australia (2000) Setting national fuel quality standards, Paper 2A. Commonwealth Department of the Environment and Heritage.

Environment Australia (2001) Air quality. Sulphur dioxide (SO_2). <http://www.ea.gov.au/atmosphere/airquality/sulphurdioxide.html> Accessed 2003.

Environment Australia (2002) National Pollutant Inventory. Sulphur dioxide and sulphate. <http://www.npi.gov.au> Accessed 2003.

Environment Australia (2003a) National Pollutant Inventory. Sulphur dioxide and sulphate. <http://www.npi.gov.au> Accessed 2003.

Environment Australia (2003b) Atmosphere - air toxics. <http://www.ea.gov.au/atmosphere/airtoxics/introduction.html> Accesssed 2003.

Environment Canada (2002) Canadian environmental quality guidelines. Summary table. <http://www.ec.gc.ca/ceqg-rcqe/English/ceqg/default.cfm.> Accessed 2003.

European Commission (1996) Technical Guidance Document in support of Commission Directive 93/67/EEC on risk assessment for new notified substances and Commission Regulation (EC) No 1488/94 on risk assessment for existing substances. Office for Official Publications of the European Communities, Luxembourg, 1996.

Fieser LF & Fieser M. (1953) Organic chemistry. London, Harrap & Co.,:585-6.

FORS (1998) Australian code for the transport of dangerous goods by road or rail (ADG Code), 6th ed. Canberra, ACT, Federal Office of Road Safety.

Gejlsbjerg B, Klinge C, Samsoe-Petersen L & Madsen T (2001) Toxicity of linear alkylbenzene sulfonates and nonylphenol in sludge-amended soil. Environ. Toxicol. Chem. **20** (12): 2709-2716.

Gillet R, Ayers G & Noller B (1990) Rainwater acidity at Jabiru, Australia, in the wet season of 1983-84. Science of the Total Environment, **92**: 129-144. Amsterdam. (Cited in NSW EPA, 1994).

Gosselin RE, Hodge HC, Smith RP & Gleason MN (1976) Clinical toxicology of commercial products. Acute Poisoning. Baltimore, USA, Williams and Wilkins Co.

Greenway H & Munns R (1980) Mechanisms of salt tolerance in nonhalophytes. Annual Rev. Plant Physiol. **31**: 149-190.

Halle JC & Stern KH (1980) Vaporization and decomposition of sodium sulfate. Thermodynamics and kinetics. J. Phys. Chem. **84**: 1699-1704

Hasegawa H & Sato M (1978) [Permeation of LAS through rat skin] Tokyo, Science and Technology Agency, Research Coordination Bureau, pp172-175 (in Japanese).

Hennes-Morgan EC & de Oude NT (1994) Detergents. In: Handbook of ecotoxicology, vol 2. Calow, P. (ed). London, Blackwell Scientific.

HERTG (Health and Environment Research Task Group) (2001a) High Production Volume (HPV) Challenge Program. Summary Group 3: Alkaryl sulfonates. American Chemistry Council, (ACC) Petroleum Additives Panel, Health, Environmental, and Regulatory Task Group (HERTG). October 2001. <http://www.epa.gov/chemrtk/alklsulf/c13206rs.pdf> (Accessed 17/12/2002).

HERTG (Health and Environment Research Task Group) (2001b) High Production Volume (HPV) Challenge Program. Test plan for petroleum additive alkaryl sulfonate category. American Chemistry Council, (ACC) Petroleum Additives Panel, Health, Environmental, and Regulatory Task Group (HERTG). October 2001. <http://www.epa.gov/chemrtk/alklsulf/c13206tc.htm> (Accessed 17/12/2002).

Heywood R, James R, & Sortwell R (1978) Toxicology studies of linear alkylbenzene sulphonate (LAS) in rhesus monkeys 1- simultaneous oral and subcutaneous administration for 28 days. Toxicology **11**: 245-250.

Holmstrup M & Krogh PH (1996) Effects of an anionic surfactant, linear alkylbenzene sulfonate, on survival, reproduction and growth of the soil-living collembolan *Folsomia fimetaria* – Short communication. Environ. Toxicol. Chem. **15**(10): 1745-1748.

Holmstrup M & Krogh PH (2001) Effects and risk assessment of linear alkylbenzene sulfonates in agricultural soil. 3. Sublethal effects on soil invertebrates. Environ. Toxicol. Chem. **20**(8): 1673-1679.

Holmstrup M, Krogh PH Lokke H, de Wolf W, Marshall S & Fox K (2000) Effects and risk assessment of linear alkylbenzene sulfonates in agricultural soil. 4. The influence of salt speciation, soil type, and sewage sludge on toxicity using the collembolan *Folsomia fimetaria* and the earthworm *Aporrectodea caliginosa* as test organisms. Environ. Toxicol. Chem. **20**(8): 1680-1689.

Holysh M, Paterson S, Mackay D & Bandurraga MM (1986) Assessment of the environmental fate of linear alkylbenzenesulphonates. Chemosphere **15**: 3-20.

Howes D (1975) The percutaneous absorption of some anionic surfactants. J Soc Cosmet Chem, **26**:47-63.

HSBD (Hazardous Substances Data Base) (2003) Sodium dodecylbenzene sulphonate. TOXNET, National Library of Medicine (USA). <http://toxnet.nlm.nih.gov/> Accessed 23 April 2003.

Humphries S (2003) The fate of sulphate in forested catchments: A sulphur isotope approach. Department of Earth Sciences, University of Waterloo. 5 pp.

Jensen J & Sverdrup LE (2002) Joint toxicity of linear alkylbenzene sulfonates and pyrene on *Folsomia fimetaria*. Ecotox. Environ. Safety **52**:75-81.

Jensen J, Lokke H, Holmstrup M, Krogh PH & Elsgaard L (2001) Effects and risk assessment of linear alkylbenzene sulfonates in agricultural soil. 5. Probabilistic risk assessment of linear alkylbenzene sulfonates in sludge-amended soils. Environ. Toxicol. Chem. **20**(8): 1690-1697.

Johnston TE & Dorer CJ (1987) PCT Int. Appl. 1987. Application WO 86-US1592 19860731.

Jorgensen E & Christoffersen K (2000) Short-term effects of linear alkylbenzene sulfonate on freshwater plankton studied under field conditions. Environ. Toxicol. Chem. **19**(4): 904-911.

Kaplin VT, Fesenko NG, Fomina AI & Zhuravleva LS (1965) Rates of decomposition of in natural waters of some synthetic detergents and surface-active substances. Nauchnye-Trudy, Akademiya Kommunal'nogo Khozyaistva im. K. D. Pamfilova, **37**: 97-110. (Russian).

Kimerle RA (1989) Aquatic and terrestrial ecotoxicity of linear alkylbenzene sulphonate. Tenside Surf. Det. **26**: 169-176.

Kirchstetter TW, Harley RA, Kreisberg NM, Stolzenburg MR & Hering SV (1999) On-road measurement of fine particle and nitrogen oxide emissions from light- and heavy-duty motor vehicles. Atmospheric Environment **33**, 2955-2968.

Kusk KO & Petersen S (1997) Acute and chronic toxicity of tributyltin and linear alkylbenzene sulfonate to the marine copepod *Acartia tonsa*. Environ. Toxicol. Chem. **16**(8): 1629-1633.

Lovei M(1998) Phasing out lead from gasoline: worldwide experiences and policy implications. World Band Technical Paper No. 397. Washington D.C., USA, The World Bank.

Mackie JC (2003) Expected high temperature combustion and final tailpipe emissions from the Na-based anti-valve seat recession additive in Morey's Upper Cylinder Lubricant and Combustion Chamber Cleaner. Draft Report to the National Industrial Chemicals Notification and Assessment Scheme (NICNAS) by the School of Chemistry, University of Sydney. 7 pp.

Matthijs E & De Henau H (1987) Determination of LAS. Tenside Surfactants Detergents, **24**: 193-199.

Meylan WM & Howard PH (1993) Computer estimation of the atmospheric gas-phase reaction rate of organic compounds with hydroxyl radicals and ozone. Chemosphere **26**: 2293-2299.

Michael WR (1968) Metabolism of linear alkylate sulfonate and alkyl benzene sulfonate in albino rats. Toxicology and Applied Pharmacology **12**: 473-485

Mitchell MJ, Krouse HR, Mayer B, Stam AC & Zhang YM (1998) Use of stable isotopes in evaluating sulphur biogeochemistry of forest ecosystems. In: McDonnell and Kendall, C (eds): Isotope tracers in catchment hydrology. The Netherlands, Elsevier.

Morey Oil South Pacific Aust Pty Ltd. (2002) Reference: PEC assessment of sodium anti-valve seat recession agent. Letter to NICNAS from A. L. Payne. 10 December 2002.

Morey Oil South Pacific Ltd (2000) Material Safety Data Sheet for Morey's Upper Cylinder Lubricant & Combustion Chamber Cleaner.

Motor Vehicle Museum of NSW (1993) Morey's Oil Test. March 1993.

Munns R (2002) The impact of salinity stress. CSIRO Division of Plant Industry, Canberra. <http://www.plantstress.com/Articles/salinity_i/salinity_i.htm> Accessed 2003

NAPAP (National Acid Precipitation Assessment Program) (1990) Annual Report 1989, Washington DC. (Cited in NSW EPA, 1994).

National Academies (2003) Project: Dietary reference intakes for electrolytes and water. Institute of Medicine Food and Nutrition Board. <http://www.iom.edu/project.asp?id=3969>. Accessed 2003.

National Chemicals Inventories (NCI) (2002) America Chemical Society Reed Technology and Information Service Inc Chemical Abstract Service Opti-Ware Software Copyright. ISSN 1089-6279.

National Heritage Trust (2000) Setting national fuel quality standards. Review of fuel quality requirements for Australian transport, Appendix 6B-10. Canberra, Commonwealth of Australia.

Navas JM, Gonzalez-Mazo E, Wenzel A, Gomez-Parra A, & Segner H (1999) Linear alkylbenzene sulfonates and intermediate products from their degradation are not Estrogenic. Marine Pollution Bulletin **38**(10): 880-884.

NDPSC(National Drugs and Poisons Schedule Committee) (2003) Standard for the Uniform Scheduling of Drugs and poisons. Canberra, Commonwealth Department of Health and Ageing.

NEPC (National Environment Protection Council) (1998b) Ambient air quality. National Environment Protection Measure for Ambient Air Quality. Adelaide, SA.

NEPC (National Environment Protection Council) (2002) Towards a National Environment Protection (Ambient Air Toxics) Measure. Discussion paper. 22 March 2002. Adelaide, SA.

NEPC (National Environment Protection Council) (1998a) National Pollutant Inventory. National Environment Protection Measure for the National Pollutant Inventory. 27 February 1998. Adelaide, SA, NEPC.

NHMRC (National Health and Medical Research Council) (1991) Recommended dietary intakes for use in Australia. Commonwealth of Australia. <http://www.health.gov.au/nhmrc/publications/diet/n6index.htm.> Accessed 2003.

NHMRC (National Health and Medical Research Council) (1996) Australian drinking water guidelines. National Health and Medical Research Council and Agriculture and Resource Management Council of Australia and New Zealand. Commonwealth of Australia.

Nogueira AH (2002) The evolution of fuel quality in Brazil. <http://www.anp.gov.br/palestras/evolution.pdf> Accessed 2003.

NOHSC (National Occupational Health and Safety Commission) (1994a) National code of practice for the control of workplace hazardous substances. Canberra, ACT, Australian Government Publishing Service.

NOHSC (National Occupational Health and Safety Commission) (1994b) National code of practice for the labelling of workplace hazardous substances. Canberra, ACT, Australian Government Publishing Service.

NOHSC (National Occupational Health and Safety Commission) (1994c) National code of practice for the preparation of Material Safety Data Sheets. Canberra, ACT, Australian Government Publishing Service.

NOHSC (National Occupational Health and Safety Commission) (1994d) National model regulations for the control of workplace hazardous substances. Australian Government Publishing Service.

NOHSC (National Occupational Health and Safety Commission) (1994e) Guidance note for the assessment of health risks arising from the use of hazardous substances in the workplace [NOHSC:3017(1994)]. Canberra, ACT, Australian Government Publishing Service.

NOHSC (National Occupational Health and Safety Commission) (1995) Exposure standards for atmospheric contaminants in the occupational environment. Canberra, ACT, Australian Government Publishing Service.

NOHSC (National Occupational Health and Safety Commission) (1996) National standard for the control of major hazard facilities [NOHSC:1014(1996)]. Canberra, ACT, Australian Government Publishing Service.

NOHSC (National Occupational Health and Safety Commission) (1999a) Approved criteria for classifying hazardous substances. Sydney, NSW, National Occupational Health and Safety Commission.

NOHSC (National Occupational Health and Safety Commission) (1999b) List of designated hazardous substances. Sydney, National Occupational Health and Safety Commission.

NSW EPA (1994) Rainfall Quality in the Upper Hunter Valley. NSW Environment Protection Authority, Sydney.

NSW EPA (1995) Assessing and Managing Acid Sulphate Soils - Guidelines for Land Management in NSW Coastal Areas, NSW Environment Protection Authority, Sydney.

OECD (Organisation for Economic Cooperation and Development) (1981) Guidelines for the testing of chemicals, Method 407, Repeated dose oral toxicity – rodent: 28-day or 14-day study.

OECD (Organisation for Economic Cooperation and Development) (1981) Guidelines for the testing of chemicals, Method 410, Repeated dose dermal toxicity: 21/28-day study.

OECD (Organisation for Economic Cooperation and Development) (1984a) OECD Guidelines for testing chemicals. No. 203 Fish acute toxicity test.

OECD (Organisation for Economic Cooperation and Development) (1984b) OECD guidelines for testing chemicals. No. 202 *Daphnia* sp. Acute immobilisation test and reproductive test.

OECD (Organisation for Economic Cooperation and Development) (1984c) OECD Guidelines for testing chemicals. No. 201 Alga growth inhibition test.

OECD (Organisation for Economic Cooperation and Development) (1984d) OECD Guidelines for testing chemicals. No. 209 Activated sludge, respiration inhibition test.

OECD (Organisation for Economic Cooperation and Development) (1987) Guidelines for the testing of chemicals, Method 401, Acute oral toxicity.

OECD (Organisation for Economic Cooperation and Development) (1987) Guidelines for the testing of chemicals, Method 402, Acute dermal toxicity.

OECD (Organisation for Economic Cooperation and Development) (1992) Guidelines for the testing of chemicals, Method 404, Acute dermal irritation/corrosion.

OECD (Organisation for Economic Cooperation and Development) (1992) Guidelines for the testing of chemicals, Method 301F, Manometric respirometry test. July 1992 OECD (1999) Advanced Air Quality Indicators and Reporting. Working Party on Pollution Prevention and Control. Environment Directorate, Environment Policy Committee. ENV/EPOC/PPC(99)9/Final. 27 September 1999.

OECD (Organisation for Economic Cooperation and Development) (1997) Guidelines for the testing of chemicals, Method 471, Bacterial reverse mutation test.

OECD (Organisation for Economic Cooperation and Development) (1997) Guidelines for the testing of chemicals, Method 473, In vitro mammalian chromosome aberration test.

OECD (Organisation for Economic Cooperation and Development) (1997) Guidelines for the testing of chemicals, Method 474, Mammalian erythrocyte micronucleus test.

OECD (Organisation for Economic Cooperation and Development) (2000) Guidance document on aquatic toxicity testing of difficult substances and mixtures. Environment Directorate, Organisation for Economic Co-operation and Development. Environmental

Health and Safety Publications Series on Testing and Assessment No. 23. June 2000. Paris, France.

OECD (Organisation for Economic Cooperation and Development) (2002) Amendment to the GHS. Sub-Committee of Experts on the Globally Harmonized System of Classification and Labelling of Chemicals. Third Session, 10-12 July, 2002). UN/SCEGHS/3/INF.16. 6 pp. Prepared as Annex 3 of OECD Document ST/SG/AC.10/C.4/2001/26.

Painter HA (1992) Anionic surfactants, p. 1-88. In: N.T. de Oude (ed). Detergents - The Handbook of environmental chemistry. Vol. 3. Part F. Anthropogenic compounds. Berlin, Springer-Verlag, (review).

Prats RD, Ruiz BF, Vazquez B, Zarzo MD, Berna JL & Moreno A (1993) LAS homolog distribution shift during wastewater treatment and composting: ecological implications. Environ. Toxicol. Chem. **12**: 1599-1608.

Reinoso H, Sosa L, Colombo C, Ochoa F & Luna V (2000) Morphological and physiological responses of *Prosopis strombulifera* (Lam.) Bent to increasing salt concentrations. Abstract 1033. Plant Biology 2000. American Society of Plant Biologists.

Rengasamy P & Olsson KA (1995) Irrigation and sodicity: An overview. In: Australian sodic soils: distribution, properties and management. Naidu, RT, Sumner, ME and Rengasamy, P. (eds). Melbourne, CSIRO Publications:195–204.

Rennison PM, Powell GM, Olavesen AH, Howes D, & Black JG (1987) Absorption and excretion of linear alkylbenzene sulphonates in rats. Biochem Soc Trans, **15**.

Ricerca Inc (1998) Biodegradation study of test substances OS 121573, OS 41177, OS 84289, OS 67948, OS 24317 and OS 16980 using batch processing respirometry test. Doc. No. 6958-96-0259-AC-001. Unpublished Laboratory Report. 30 October 1998.

Rodhe H (1989) Acidification in global perspective. Ambio **18**(3): 155-160. (Cited in NSW EPA, 1994).

Routledge EJ & Sumpter JP (1995) Estrogenic activity of surfactants and some of their degradation products assessed using a recombinant yeast screen. Environ. Toxicol. Chem. **15**(3): 241-248.

Safepharm Laboratories (2001a) Acute toxicity to *Daphnia Magna* using a WAF preparation method employing a single phase separation period. Safepharm Laboratories Limited SPL Project Number 525/325, Derby, United Kingdom. April 2001

Safepharm Laboratories (2001b) Acute toxicity to Rainbow Trout (*Oncorhynchus mykiss*) using a WAF preparation method employing a single phase separation period. Safepharm Laboratories Limited SPL Project Number 525/324, Derby, United Kingdom. April 2001.

Sammut J & Lines-Kelly R (1996) An introduction to acid sulphate soils. Acid Sulphate Soils Management Advisory Committee (ASSMAC), Dept Environment, Sport and Territories (C'wth), Australian Seafood Industry Council.

Sammut J, Callinan JB & Fraser GC (1999) An overview of the ecological impacts of acid sulphate soils in Australia. 2nd National Conference on Acid Sulphate Soils, Coffs Harbour; and Healthy Rivers Commission Independent Inquiry into the Clarence River System Final Report. November 1999.

Society for Range Management (2003) Preventing sulphur toxicity in livestock. Forage Quality and Livestock Nutrition. Wyoming Section. Wyoming University. http://uwacadweb.uwyo.edu/Wysrm/range_rider/preventing_sulphur_toxicity.htm>

Springborn Bionomics (1986a) Acute toxicity of CMA test material code 504 to Sheepshead Minnow, *Cyprinodon variegates*. Springborn Bionomics Study. No. 10823-0186 100-500-504. Report BW-86-04-1983.

Springborn Bionomics (1986b). Acute toxicity of CMA test material dode 514 to Sheepshead Minnow, *Cyprinodon variegates*. Springborn Bionomics Study. No. 10823-0186-6100-500-514. Report BW-86-04-1993.

Springborn Bionomics (1986c) Acute toxicity of CMA test material code 523 to Sheepshead Minnow, *Cyprinodon variegates*. Springborn Bionomics Study. No. 10823-0186-6100-500-523. Report BW-86-04-1986.

Standards Australia (1996) Australian Standard 4430.1-1996– Evaluation of devices and additives which claim to improve vehicle performance. Part 1: Engines designed for leaded petrol to operate on unleaded petrol. Homebush, N.S.W., Standards Association of Australia.

Steber J & Berger H (1995) Biodegradability of anionic surfactants, p. 134-182. In: Karsa, DR and Porter MR (eds.), Biodegradability of surfactants. Blackie Academic and Professional, Glasgow, United Kingdom (review).

Stott FH, Jordan MP & Barlow PL (2000) Parsons 2000, Advanced materials for 21st century turbines and power plant, Proceedings of the International Charles Parsons Turbine Conference, 5th, Cambridge, U.K., July 3-7, 2000, pp. 671-682

Sunakawa T, Ikida Y, & Odamoto K (1979) [Absorption, distribution, metabolism, and excretion of linear alkylbenzene sulfonate in rats. JJpn Oil Chem Soc [Yuukagaku], **39**(2):59-68

Tabor CF Jr., Barber II LB & Runnells DD (1993). Anionic surfactants in the Mississippi River; A detailed examination of the occurrence and fate of Linear Alkylbenzene sulphonate. Preprint extended abstracts. 205th Annual Meeting, American Chemical Society. **33**: 52-55. Denver, CO, March 28-April 2. Cited in CLER (1999).

Takada H & Ishiwatari R (1987) Linear alkylbenzenes in urban riverine environments in Tokyo: Distribution, Source, and Behaviour. Environ. Sci. Toxicol. **21**: 875-883.

Takada H & Ishiwatari R (1991) Linear alkylbenzenes (LABs) in urban riverine and coastal sediments and their usefulness as a molecular indicator of domestic wastes. Wat. Sci. Technol. **23**: 437-446.

ToxProbe Inc. (2002) Estimated human health risk from exposure to diesel exhaust in Toronto. Prepared for Toronto Public Health by Pavel Muller. <http://www.city.toronto.on.ca/health/de_index.htm.> Accessed 2/5/2003.

Tulau M (1999) Management of acid sulphate soils in New South wales – Policy, organisation, and regulation. The Australasian Journal of Natural Resources Law and Policy , **6**(1).

URS (2001) Aquatic ecological risk screening for Midnite Mine RI/FS. Draft Technical Memorandum. Unpublished consultancy report prepared for USEPA Region 10. August 2001. URS Denver Colorado. Project No. 53F4001800.07.

US Congress. Clean Air Act Amendments of 1977. PL 95-95. Washington: US Government Printing Office, 1977.

USEPA (United States Environmental Protection Agency) (United States Environmental Protection Agency) (1990) National primary and secondary drinking water regulations; synthetic organic chemicals and inorganic chemicals. Federal Register. Vol. **55**. No. 143. 30370. (Cited in Bast, 1991).

USEPA (United States Environmental Protection Agency) (1985a) Toxic Substances Control Act. Fish, acute toxicity test freshwater and marine. Test guideline 797.1400. EPA 712-C-96-118.

USEPA (United States Environmental Protection Agency) (1985b) Toxic Substances Control Act. *Daphnia* sp, acute toxicity test. Test guideline 797.1300.

USEPA (United States Environmental Protection Agency) (1985c) Toxic Substances Control Act. Alga, acute toxicity test. Test guideline 797.1050.

USEPA (United States Environmental Protection Agency) (1993) 40 CFR Part 797. Toxic Substances Control Act Test Guidelines. Final Rules.

USEPA (United States Environmental Protection Agency)(1994b) Fuel and fuel additives registration regulations; final rule. Fed Reg. **59**:33042-33142

USEPA (United States Environmental Protection Agency) (1999) 64 Federal Register (FR). 38705. National Air Toxics Program: The Integrated Urban Strategy (Notice). July 19, 1999.

USEPA (United States Environmental Protection Agency) (2002a) EPA comment on chemical RTK HPV Challenge submission: Petroleum additive alkaryl sulfonates Category. Summary of EPA comments. Letter from Oscar Hernandez, Director, Risk Assessment Division. 30 May, 2002. 6 pp.

USEPA (United States Environmental Protection Agency) (2002b) High Production Volume (HVC) Chemical Challenge Program. Hazard data availability and assessment report for linear and branched alkylbenzene sulfonic acids and derivatives. Prepared on behalf of the LAS/ABS Consortium, December 27, 2002.

Utsunomiya A, Watanuki T, Matsushita K & Tomita I (1997) Toxic effects of linear alkylbenzene sulfonate, quaternary alkylammonium chloride and their complexes on *Dunaliella* sp and *Chlorella pyrenoidosa*. Environ. Toxicol. Chem. **16**(6): 1247-1254.

Vinther FP, Mortensen GK & Elsgaard L (2003) Effects of linear alkylbenzene sulfonates on functional diversity of microbial communities in soil. Environ. Toxicol. Chem. **22**(1): 35-39.

WA Agriculture (2003) Salinity tolerance of plants for agriculture and revegetation. <http://www.agric.wa.gov.au/environment/land/salinity/measurement/Plant_salt_tolerance.htm#nonsaline>. Department of Agriculture, Western Australia.

Wateri N, Torizawa W, Kanai M, & Suzuki Y (1977) Ultrastructural observations of the protective effect of glycyrrhizin for mouse liber injury caused by oral administration of detergent ingredient (LAS). Journal of Clinical Electron Microscopy, **10**: 121-139.

Watson WA, Bradford DC, & Veltri JC (1983) The volume of a swallow: correlation of deglutition with pateint and conatiner parameters. Am. J. Emergency Med., **3**: 278-281.

WHO (World Health Organisation) (1984) Guidelines for drinking water quality. Vol. 2. Health criteria and other supporting information.

WHO (World Health Organisation) (1996) Linear alkylbenzene sulfonates and related compounds. Environmental health criteria 196, Geneva. International Program on Chemical Safety. World Health Organisation.

Wilbury TR (1993a) Acute toxicity of the water accommodated fractions (WAFs) of CMA 60.5 to the Fathead Minnow, Pimephales promelas. T.R Wilbury Study No. 9176-CMA/ESI-605.

Wilbury TR (1993b) Acute toxicity of the water accommodated fractions (WAFs) of CMA 609 to the Fathead Minnow, *Pimephales promelas*. T.R Wilbury Study No. 9176-CMA/ESI-609.

Wilbury TR (1993c) Acute toxicity of the water accommodated fractions (WAFs) of CMA 604 to Daphnia, *Daphnia magna*. T.R Wilbury Study No. 9178-CMA/ESI-604.

Wilbury TR (1993d) Acute toxicity of the water accommodated fractions (WAFs) of CMA 605 to Daphnia, *Daphnia magna*. T.R Wilbury Study No. 9178-CMA/ESI-605.

Wilbury TR (1993e) Acute toxicity of the water accommodated fractions (WAFs) of CMA 609 to Daphnia, *Daphnia magna*. T.R Wilbury Study No. 9178-CMA/ESI-609.

Wilbury TR (1994a) Acute toxicity of the water accommodated fractions (WAFs) of CMA 604 to the Freshwater Alga, *Selenastrum capricornutum*. T.R Wilbury Study No. 73-CM-604.

Wilbury TR (1994b) Acute toxicity of the water accommodated fractions (WAFs) of CMA 605 to the Freshwater Alga, *Selenastrum capricornutum*. T.R Wilbury Study No. 73-CM-605.

Wilbury TR (1994c) Acute toxicity of the water accommodated fractions (WAFs) of CMA 609 to the Freshwater Alga, *Selenastrum capricornutum*. T.R Wilbury Study No. 73-CM-609.

Wilbury TR (1998) Acute toxicity of the water accommodated fraction (WAF) of OS# 60742C to the Freshwater Alga, *Selenastrum capricornutum*. Wilbury Laboratories, Inc, Massachusetts.

Woodward-Clyde (1994) The effect of OS45720L on the respiration of activated sludge (expanded range assessment). Woodward-Clyde Consultants, Franklin, Tennessee. April 1994. Final Report. Unpublished Consultancy Report.

Yahata T (1991) Jpn. Kokai Tokkyo Koho (1991), JP 03195809. Abstract of patent from Chem. Abstr. CAN 115: 262687.

Zayed J, Gérin M, Loranger S, Sierra P, Bégin D & Kennedy G (1994) Occupational and environmental exposure of garage workers and taxi drivers to airborne manganese arising from the use of methylcyclopentadienyl manganese tricarbonyl in unleaded gasoline. Am. Ind. Hyg. Assoc., **55**: 53-58

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