Ministry for the Environment
Manatū Mō Te Taiao

Review of the Ambient Air Quality Guidelines

Health Effects of Eleven Hazardous Air Contaminants and Recommended Evaluation Criteria

Final Report

Prepared by Chiodo *et al.*, for the Ministry for the Environment's Review of the *Ambient Air Quality Guidelines*

October 2000

Air Quality Technical Report No. 13

Technical Report Prepared by Chiodo *et al*. for
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This document is available on the Ministry for the Environment's Web site:
http://www.mfe.govt.nz

Foreword by the Ministry for the Environment

This technical report has been prepared by Chiodo *et al*. for the Ministry for the Environment's review of the *Ambient Air Quality Guidelines 1994* (the 1994 Guidelines). It examines the health effects of 10 contaminants that were not covered in the 1994 Guidelines plus lead, and recommends monitoring methods and evaluation criteria for each contaminant. Generically these contaminants are commonly referred to as hazardous air contaminants (HACS) or hazardous air pollutants (HAPS).

In the 1994 Guidelines, the Ministry[1] recommended a precautionary approach to managing HACs under the Resource Management Act 1991 that was to minimise their discharge into the environment as far as practicable. As a consequence, different approaches and guideline values have been used to: assess discharges of hazardous contaminants, apply the principle of minimisation and to analyse the significance of ambient monitoring results. These different approaches have caused confusion and concern for industries, councils and communities and there have been calls for improved guidance and better national consistency. The Ministry aims to address these concerns by preparing national guidance on how to assess, manage and monitor the impacts of priority HACs.

To develop guidance, this report was commissioned by the Ministry. The report describes how 11 priority hazardous air contaminants for New Zealand were chosen, critiques international research on the effects of these contaminants on humans, discusses overseas standards and guideline values, recommends monitoring methods and recommends two sets of criteria to protect human health and well-being. One set is for analysing the results of ambient monitoring (essentially ambient guideline values) and the other is for assessing the results of atmospheric dispersion modelling of industrial discharges (typically referred to as modelling design concentration values or design ground level concentrations).

The draft version of this report was reviewed and discussed by around 50 practitioners from industry, councils, non-government organisations, universities and air quality management at workshops in March 2000. In response to discussions and written comments from the reviewers the Ministry has included information on lead, and the risk estimates for each of the annual criteria values are now included in tables in Annexes D and E.

The information and evaluation criteria in this report have been used to develop the Ministry's *Proposals for Revised and New Ambient Air Quality Guidelines for New Zealand – Discussion Document*. Any comments on this technical report should be included with your submission on the Ministry's Discussion Document. The other technical reports prepared for the review of the Guidelines are available from the Ministry's website on:

http://www.mfe.govt.nz/monitoring/epi/airqualtech.htm

This report does not discuss dioxins and polychlorinated biphenols as these have already been determined as priority contaminants and are addressed through the Ministry's Organochlorines Programme. Also, the focus of this review is on "waste" air pollutants rather than substances that are typically used in processes or applications (such as agrichemicals), although there is some overlap – such as toluene and xylene. Where these substances are used in processes they are controlled under the Hazardous Substances and New Organisms Act (HSNO) regulations

[1] List of 189 hazardous air contaminants regulated by the US Clean Air Act 1990 contained within the Ambient Air Quality Guidelines, Ministry for the Environment, 1994

and their off-site effects, such as spray drift, by the Resource Management Act 1991. Other Ministry work is investigating these issues.

As well as being part of the review of the 1994 Guidelines, this review of HACs contributes to the Ministry's Hazardous Waste Management Programme. Together with the Organochlorines Programme, and the HSNO Act, these programmes and laws aim to set national approaches for managing and controlling hazardous contaminants in the environment.

Acknowledgements

The Ministry thanks the following experts for writing and peer reviewing this report.

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The Ministry also thanks reviewers involved in the meetings to discuss the draft version of this report, and who provided written comments.

Contents

Tables and Figures

1 Introduction

Hazardous air contaminants are a variety of potentially airborne chemicals with toxic or carcinogenic properties. Although their levels in ambient air are generally very low, they can, under some circumstances, be released into the air environment in sufficient quantities to be potentially hazardous to humans and other species. The Organisation for Economic Co-operation and Development (OECD) defines these substances as "gaseous, aerosol or particulate pollutants which are present in air in trace amounts with characteristics such as toxicity or persistence so as to be a hazard to human, plant or animal life".

Sources of hazardous air contaminants are many and varied. They include industrial processes such as chemical and paint manufacturing; oil refining and power generation; domestic activities such as lawn-mowing, house painting and building maintenance; motor vehicle usage; other forms of transport such as buses, trucks and aircraft; and natural sources such as volcanic and geothermal activity and vegetation. Most hazardous air contaminants are usually specific to a few sources or source categories and have only local impacts. Others, including those emitted by motor vehicles, are more widespread and have correspondingly wide potential impacts.

Potential adverse effects of hazardous air contaminants are also many and varied. They range from minor effects such as skin and eye irritation, to more serious effects of severe respiratory impairment, nerve and organ damage, increased risk of leukaemia and cancer, and premature death. The effects can be acute (occur at or shortly after exposure) or chronic (develop over a long period). Adverse effects are usually proportional to the dosage received and/or the concentration of the pollutant.

Some pollutants have a threshold, that is, an ambient concentration below which no adverse effects are known to occur, while others have no identifiable threshold. Some very stable chemicals can bioaccumulate and exert their impact via the food chain. Some, such as solvents and thinners, are produced for direct use in a variety of applications, or as building blocks for other products such as plastics. Many hazardous air contaminants are unwanted by-products, or are inadvertently produced or released in various processes.

Public concerns about hazardous air contaminants have increased as awareness about the large variety of chemicals has increased and better information about their potential hazards has become available. This has led to increasing demands for better controls on their production, use and release to the environment. Approaches for reducing the hazards from these pollutants have been developed, and these are outlined in detail in Annex C.

2 Establishing priority hazardous air contaminants

Because of the very large number of substances that can be referred to as hazardous air contaminants, it is impracticable to develop guidelines and approaches to reduce potential hazards from all them. The New Zealand Department of Labour lists occupational health and safety standards for more than 500 substances. The National Institute for Water and Atmospheric Research Ltd (NIWA) has compiled a draft database of hazardous air contaminants (Hazardous Ambient Air Contaminants Database, or HAAC) considered relevant for New Zealand[2]. The HAAC database (NIWA, 1997c) provides a convenient starting list for prioritisation. It contains over 200 contaminants, which are listed in Annex B. The priorities developed by different groups and organisations are also indicated in Annex B and discussed below.

In the United States, the 1990 amendments to the Clean Air Act list 189 pollutants or chemical groups (later amended to 188) that are considered hazardous. The amendments required the US Environmental Protection Agency (EPA) to develop a list of at least 30 substances from area-based sources judged to be of the greatest potential threat to public health in the largest number of urban areas. An initial list of 40 was reduced to a draft list of 33 high-priority hazardous air contaminants, and published by the US EPA in the Federal Register on 4 September 1998 as part of its draft national strategy for air toxics (that is, hazardous air contaminants).

During 1995–96 the Victorian Environment Protection Authority conducted trials of a National Pollutant Inventory (NPI) in four locations in Australia representing different population densities and industry mixes. The trials included emission estimates for industrial, commercial and domestic sources, and motor vehicles. A basic list of 26 pollutants was selected, based on their importance. This selection was made on the professional judgement of a steering committee with expertise in air quality. Minor adjustments were made in each area following local input. The pollutants in the NPI trials are indicated, as is the emission estimate for the location emitting the greatest quantity of each pollutant.

As part of the process for implementing an NPI in Australia, a technical advisory panel developed a comprehensive system for ranking pollutants for inclusion in the NPI. Approximately 400 substances were ranked.[3] The approach involves generating a hazard score by considering human health effects (acute, chronic, reproductive, and carcinogenic) and ecological effects (acute and chronic), and a potential exposure score by considering emissions (point and diffuse sources), quantities, and ultimate fate in the environment. Expert judgement is necessary to generate exposure scores due to the lack of relevant data for many substances. A risk score (range 0–18) for each substance is then obtained by multiplying a normalised hazard score by a normalised exposure score. The risk score for each substance and the NPI rank are shown in Annex B.

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[2] The Ministry will consider further development of this database following the completion of the Guidelines review.

[3] Details of the approach are contained in the panel's report to the National Environment Protection Council (1997).

A similar system for ranking pollutants for ambient air monitoring was developed by the Victorian Environment Protection Authority as part of a review of hazardous air contaminants for the Australia and New Zealand Environment and Conservation Council (ANZECC). The potential exposure estimate was based on ambient measurements. Because of the paucity of local data, the use of data from various sources was necessary. Both the basis and the normalisation process for effects also differed from those of the NPI Technical Advisory Panel. A panel ranking by Victorian Environment Protection Authority air-quality staff was also used. Both scores (range 0–20) are shown in Annex B.

Steveson and Mills (1999) evaluated likely exposure levels to a range of hazardous air contaminants for the general population in ambient, indoor, in-vehicle and environmental tobacco smoke situations. They used New Zealand information on exposure levels where available and international information where there was no data. Chronic reference values from several international agencies including the WHO, Californian EPA and the USEPA reference concentrations (RfCs) where used in the analysis. Formaldehyde, acrolein, diesel exhaust, tobacco smoke and 1,4– dichlorobenzene (mothballs) were found to pose the greatest risk to human health, and there were considered to have a lower risk than the exiting guidelines pollutants such as particles and nitrogen dioxide.

Two high-priority lists of hazardous air contaminants developed on the basis of expert judgement have been published for New Zealand: one by NIWA (1997a), the other by Bingham (1998). Both are indicated in Annex B. A summary of both lists, and the pollutants common to both lists (approximately one third of those on the combined list) are shown in Table 2.1.

Table 2.1: Hazardous air contaminants considered important in New Zealand

Compound	NIWA (N) Bingham (B)
Acetaldehyde	B N
Acrolein	B
Acrylonitrile	N
Arsenic – elemental and soluble compounds	B N
Benzene	B N
Beryllium – elemental and compounds	B
1,3-Butadiene	B N
Cadmium – elemental and compounds	B N
Carbon tetrachloride	B
Chloroform	B
Chromium – (VI) insoluble compounds	B N
Chromium – (VI) soluble compounds	B N
Chromium – elemental and (II), (III) compounds	B N
1,1-Dichloroethane	N
Dimethylnitrosoamine (N-nitrosodimethylamine)	B
Ethyl acrylate	B
Ethylene oxide	B
Ethylene thiourea	B
Formaldehyde	B N
Hexachlorobenzene	B
Lead – elemental and compounds	B
Mercury – alkyl compounds	B N

Compound	NIWA (N) Bingham (B)
Mercury – aryl compounds	B N
Mercury – elemental and inorganic compounds	B N
Methyl bromide	N
Methylene chloride	N
Nickel – elemental and compounds	N
PAHs – as benzo(a)pyrene	B N
Perchloroethylene	N
Polychlorinated biphenyls (PCBs, aroclors)	B
Styrene	N
2,3,7,8-Tetrachlorodibenzo-p-dioxin	B N
Toluene	N
2,4-Toluene diamine	B
Trichloroethylene	N
Vinyl chloride	B N
m-Xylene	N
o-Xylene	N
p-Xylene	N
Xylenes – mixed isomers	N
Hydrogen fluoride	B
Hydrogen sulphide	N
2-Methoxy ethanol (methyl cellosolve)	B

A review of toxic contaminants (Ministry for the Environment, 1998c) identified an initial priority list of toxic substances for inclusion in the Ministry's Environmental Performance Indicators Programme. The "first tier" list, based on human health considerations, includes the following generic classes of substances, and typical examples:

- volatile hydrocarbons (mainly benzene, toluene, 1,3-butadiene and other compounds found in fuels)
- heavy metals (mainly mercury, arsenic, and other metals found in transport fuels)
- polycyclic aromatic hydrocarbons (PAHs) (mainly from transport emissions)
- organochlorines (mainly dioxins and compounds used in timber treatment)
- pesticides (mainly the components in spray drift).

Carbonyls were an apparent omission from this list. Therefore, the substances considered for this present review are as follows.

- Volatile hydrocarbons – **benzene, toluene, xylene** and **1,3-butadiene**.[4]

[4] A number of other volatile hydrocarbons are important for their potential to react with oxides of nitrogen and generate photochemical smog products, usually measured as ozone. They have not been included here since their importance as hazardous air contaminants is considered lower than those selected. Chlorinated organics, such as dioxins, are not included in this review, because they are being addressed under the Ministry for the Environment's Organochlorines Programme.

- Carbonyls – **formaldehyde** and **acetaldehyde.** These can also be classified as volatile hydrocarbons and organic compounds, collectively referred to as volatile organic compounds (VOCs).
- Polycyclic Aromatic Hydrocarbons (PAHs) – **benzo(a)pyrene.** PAHs are defined either as specific compounds, or in relation to the method used for their measurement. There are 44 different PAHs. Benzo(a)pyrene is considered the most relevant and hazardous, and is commonly used as an indicator species for PAHs.
- Metals – **arsenic, mercury** and **chromium**. The selection of these compounds is to a large extent a matter of judgement. It is not expected that significant quantities would be emitted in an urban environment. The elimination of lead from petrol lowers the priority on lead as a widely occurring hazardous air pollutant. Lead is already covered in the existing Guidelines.

All the above selected pollutants, except toluene and xylene, appear on both of the New Zealand experts' lists. They are high priority on the US EPA list of urban air toxics, and were ranked in the top 60 of the approximately 400 hazardous air contaminants considered by the NPI Technical Advisory Panel. The selected substances, therefore, comprise a suitable subset of hazardous air contaminants for priority evaluation.

3 Sources

Information about emissions of hazardous air contaminants from particular sources in New Zealand comes from emissions inventories compiled for urban population centres. Some regional councils, including Auckland Regional Council (Victorian Environment Protection Authority, 1997) and Canterbury Regional Council (NIWA, 1997b), have compiled emissions inventories for their major urban centres. Inventories of national transport emissions (NIWA, 1996a), industry, natural and area sources (NIWA, 1996b) have also been completed.

The main focus of these inventories is on the widely occurring "common" pollutants (carbon monoxide, sulphur dioxide, nitrogen oxides, lead, and particles) and total VOCs, which react with nitrogen oxides under sunlight to produce ozone and other oxidants (photochemical smog).

The reliability and accuracy of emissions inventories depend on the quality of local data, particularly emissions data for major sources such as motor vehicles. Because of the lack of New Zealand data, interpolation from relevant overseas data has been necessary to generate inventories, which has introduced some uncertainty to the estimates.

Many hazardous air contaminants are either VOCs (for example, benzene) and hence are included in VOC inventories, or occur as particles (for example, metals). Emissions of individual hazardous air contaminants can be calculated by applying speciation factors to total VOCs. Speciation factors have been derived for some air toxics and some source categories based on overseas data. These estimates are subject to the same uncertainty as the VOC estimates. There are no reliable speciation factors for estimating metal emissions from particles.

3.1 Benzene

Based on the Auckland emissions inventory, the total 1993 VOC emissions in the Auckland region were estimated to be about 65,000 tonnes. Of these, 63% came from motor vehicles, 13% from domestic solid-fuel combustion, 3.7% from domestic and commercial surface-coating operations, and 5.3% from industrial coating operations.

Emissions of benzene were estimated at about 7% of total VOCs. Approximately 80% of these came from motor vehicles, the other 20% largely from domestic solid-fuel combustion. The ratio of motor vehicle exhaust to evaporative emissions for benzene was approximately 2:1. Motor vehicle exhaust emissions of benzene are thought to derive partly from unburnt benzene in the fuel, and partly from the dealkylation of other aromatic hydrocarbons in the fuel. Reducing benzene emissions therefore requires controlling not only motor vehicle exhaust emissions, but also the aromatic content of motor vehicle fuels.

Domestic solid-fuel combustion is also a major source of benzene emissions during autumn and winter. Stevenson and Narsey (1998) estimate that domestic and commercial heating contribute about 46% of ambient benzene levels in Auckland during this period. Therefore, appropriate controls on solid-fuel heaters may also be necessary for reducing ambient benzene levels.

Other sources of benzene emissions, which may impact locally in some areas, include oil refining, petrochemicals, and synthetic rubber manufacture.

3.2 Toluene and xylene

Emission data for several sources of toluene and xylene in the Auckland region are shown in Table 3.1. The data shows how much of the total VOC emitted from motor vehicles, surface coating operations and domestic solid fuel heating is toluene and xylene.

Table 3.1: Emissions of toluene and xylene in Auckland

Emission source	Toluene (% of total VOC)	Xylene (% of total VOC)
Motor vehicles	10	8.8
Exhaust to evaporative ratio	~4:1	~2.5:1
Surface-coating operations	2.7	1.3
Domestic solid-fuel combustion	0.7	–

Source: Victorian Environmental Protection Agency, 1997.

Motor vehicle emissions account for ~75% and 85% of toluene and xylene emissions in the Auckland region. Toluene and xylene are used as solvents and most of the remainder of these emissions come from surface-coating operations. As with benzene, local impacts can occur around oil refining, petrochemical industries, and adhesive manufacturing and formulation.

3.3 1,3-Butadiene

There is little New Zealand data for quantifying 1,3-butadiene emissions. Sources include motor vehicle exhausts, and synthetic rubber, latex, and resin production. The most recent inventory for Melbourne (Victorian Environment Protection Authority, 1998) indicates that 76% of 1,3-butadiene emissions come from motor vehicles, 15% from industry, and 8% from domestic/commercial sources; the ratio of 1,3-butadiene to benzene is 0.13.

Nelson et al (1998) summarise data from an Australian Federal Office of Road Safety study of emissions from motor vehicles in Melbourne and Sydney. Exhaust emissions of 1,3-butadiene vary from 0.7% of non-methane hydrocarbons (NMHCs)[5] for catalyst cars, to 1.2% for non-catalyst cars. The ratios of 1,3-butadiene to benzene are 0.07 and 0.13 respectively. For evaporative emissions the ratios are 0.22 to 0.08 respectively.

Stevenson and Narsey (1997) found that the 1,3-butadiene to benzene ratio, based on annual average monitoring data in different New Zealand cities, ranged from 0.004 to 0.016. The atmospheric half-life of 1,3-butadiene is quite short (several hours) compared to benzene (several days). More recent data (Stevenson et al, 1999) and review suggests that the results for 1,3-butadiene from the 95/96 sampling (Stevenson and Narsey, 1997) may underestimate 1,3 butadiene levels and proportions to benzene. Stevenson and Mills (1999) suggests using a 15% 1,3 butadiene/benzene ratio for estimation of 1,3-butadiene concentrations from annual average benzene concentrations. The 15% is in general agreement with the 10% suggested below but 10% seems likely to be the bottom of the range rather than the midpoint. Care therefore needs

[5] NMHCs are VOC minus carbonyls; for all practical purposes, NMHCs can be taken as equal to VOC, since carbonyls are only a few percent of VOCs.

to be taken in using ratios for scaling 1,3-butadiene from benzene, using either monitoring data or emissions data from other cities.

In the absence of other data, the Auckland emissions inventory can be combined with the various data on ratios cited above to provide an estimate of the 1,3-butadiene to benzene emissions ratio for urban areas. The estimate of around 0.10 is broadly consistent with the ambient air ratio derived from the Stevenson and Narsey data, allowing for the different atmospheric half-life.

3.4 Formaldehyde

Motor vehicles and domestic solid-fuel combustion are the major sources of formaldehyde in the urban environment. Industrial sources can be locally important, and include the manufacture of particle board, plywood, fabrics and furnishings. Formaldehyde emissions from furnishings and fittings can be important for indoor air quality. There are eight particle board mills in New Zealand, typically discharging 0.5–5 kg/hr of formaldehyde. They are significant locally, but less so on a regional scale.

The atmospheric half-life is quite short (a few hours) so that the main impacts are relatively close to the source. However, formaldehyde is highly reactive and is an important contaminant in causing widespread photochemical smog.

Based on the Auckland inventory, formaldehyde emitted by motor vehicle and domestic solid-fuel combustion are 1% and 0.8% of total VOC emissions in the Auckland region. Monitoring data from a short (17-day) study of transport emissions by NIWA at Khyber Pass Road in Auckland (Kuschel et al, 1998) indicates a ratio of formaldehyde to benzene of 0.15. This is consistent with the Auckland inventory ratio of 0.18 for motor vehicle emissions.

The emissions inventory for Melbourne (Victorian Environment Protection Authority, 1998) indicates that motor vehicles and domestic solid-fuel consumption account for 64% and 24% respectively of total formaldehyde emissions in the Melbourne region. Data from Nelson et al (1998) indicate that formaldehyde emissions from catalyst and non-catalyst cars are 1.2% and 2.9% respectively of vehicle exhaust VOC emissions. These data are in general agreement with the Auckland inventory estimates. In the absence of other data, emissions of formaldehyde in urban areas can be scaled from emission estimates for VOCs.

3.5 Acetaldehyde

Acetaldehyde, like formaldehyde, is very reactive and important in photochemical smog reactions. Major sources are motor vehicle exhaust and domestic solid-fuel combustion. The study by Nelson et al (1998) indicates that acetaldehyde emissions from catalyst and non-catalyst cars are 0.7% and 0.4% respectively of vehicle exhaust VOC emissions. Galbally et al (1998) provide estimates of fleet average emissions of acetaldehyde and formaldehyde for the Melbourne fleet and a summary of overseas data. Emission rates (in milligrams per kilometre) and ratios of acetaldehyde emissions to formaldehyde emissions are given in Table 3.2.

Table 3.2: Emission rates and ratios of acetaldehyde and formaldehyde

Species	Melbourne	Overseas

	Fleet average	Light-duty vehicles	Heavy-duty vehicles
Formaldehyde	20 ± 2.4 mg/km	4.0–16.0 mg/km	32.7–91.5 mg/km
Acetaldehyde	4 ± 1.0 mg/km	1.3–9.5 mg/km	20.0–49.5 mg/km
Ratio of acetaldehyde to formaldehyde	0.25	0.32–0.69	0.54–0.61

These data are consistent with those in Nelson et al (1998), which also indicate ratios of acetaldehyde to benzene of 0.58 for a catalyst car and 0.14 for a non-catalyst car.

The Auckland inventory indicates that acetaldehyde from motor vehicle exhaust is 0.4% of total VOC emissions in the region. No data are provided for acetaldehyde emissions from domestic solid-fuel combustion. VOC speciation factors published by the California Air Resources Board (1998a) indicate an acetaldehyde to formaldehyde ratio for residential wood combustion of 0.93. On this basis, acetaldehyde emissions from domestic fuel consumption are around 0.75% of total VOC emissions in the Auckland region.

3.6 Benzo(a)pyrene

Benzo(a)pyrene (BaP) is a minor component of PAHs, but extremely important because of its highly toxic and carcinogenic properties. It is used as an indicator species for a wide range of compounds, some of which are also toxic or carcinogenic. PAHs arise from the incomplete combustion of solid and liquid fuels. They are semi-volatile compounds, and occur in both the gaseous phase or attached to fine particles.

There are few estimates of PAH emissions for New Zealand. Kvatch et al (1998) report PAH emissions from tunnel exhausts for the Mount Victoria tunnel in Wellington and the Lyttelton tunnel in Christchurch. Data are reported for five PAHs but do not include benzo(a)pyrene. Computed emission rates for the Mount Victoria tunnel are approximately half those for Lyttelton, which the authors attribute largely to the higher proportion of heavy-duty vehicles in the latter. Measurements were also taken in Christchurch in 1979 during periods of poor air-quality (days when air particulate matter exceeded 100 $\mu g/m^3$), and the particulate matter was analysed for PAHs (Cretney et al, 1985).

The Melbourne emissions inventory (Victorian Environment Protection Authority, 1998) estimates emission rates for total PAHs for the inventory region as 270 tonnes per annum, including 42% from motor vehicles and 50% from domestic solid fuel combustion. No specific estimates of benzo(a)pyrene are provided. Summary data from a draft report reviewing hazardous air contaminants in Australia and New Zealand (Victorian Environment Protection Authority, 1999a) indicate Benzo(a)pyrene to total PAHs ratios in ambient air for Melbourne of 0.07–0.08. Kuschel et al (1998) present summary data for Khyber Pass Road in Auckland which indicate a ratio of 0.11.

There is no reliable way of estimating Benzo(a)pyrene emissions and ambient levels in New Zealand from existing information. The above data are at best an indication.

3.7 Mercury, chromium and arsenic

There are no reliable estimates of metal emissions in New Zealand, and few in Australia. Emissions of metals are mainly associated with particles emitted from sources burning fossil fuels, including power stations, cars and trucks. Metal emissions largely depend on the metal content of the fuel, which varies with both the fuel type and source.

Specific sources of mercury include crematoria, waste incineration, gold-recovery plants, and chlor-alkali plants employing the mercury cell process (previously the case at Kinleith, which is now closed down). In New Zealand, volcanic and geothermal activities are probably the most significant sources of mercury, but emissions are largely unquantified.

Specific sources of chromium include metal smelting and foundries, cement production, pulp and paper mills, chrome plating, timber treatment using copper/chrome/arsenic preservatives, cooling towers, and leather tanning.

Specific sources of arsenic include timber treatment using copper/chrome/arsenic preservatives and previous pesticide application. Emissions are largely to land or water. Arsine can be released to atmosphere from old chemical landfill sites. The burning of treated timber releases volatile arsenic oxides, either in the gaseous form or associated with particle emissions. Health and environmental guidelines for selected timber treatment chemicals have been prepared (Ministry for the Environment / Ministry of Health, 1997).

The Melbourne emissions inventory indicates that annual emissions of compounds of mercury, chromium and arsenic are 0.21, 3.2, and 0.69 tonnes respectively. The Australian national pollutant inventory (NPI) trials (Victorian Environment Protection Authority, 1996) report emissions as shown in Table 3.3.

Table 3.3: Emission of metals at selected Australian sites (tonnes)

Compound	Dandenong (Victoria)	Port Pirie (South Australia)	Newcastle (New South Wales)	Launceston (Tasmania)
Mercury	0.0023	0.001	–	0.001
Chromium	0.321	<0.001	0.076	0.005
Arsenic	–	–	0.003	–

3.8 Lead

The chemical and physical properties of lead have made it useful as an ingredient in paint, batteries and most notably, automotive fuels. Historical use of organic compounds, tetra-ethyl and tetra methyl lead in petrol resulted in a widespread dispersion of lead into the environment.

Unlike the other hazardous air contaminants covered in this report, lead was covered in the 1994 Guidelines. The 1994 guideline value is the range 0.5 to 1.0 g/m^3 (3 month moving average, calculated monthly). In the 1994 guidelines, the recommended measurement method involved the determination of lead in suspended particulate using AS2800-1985. This method involves a high volume sampler for either total suspended particulate or with a size selective inlet for PM10 (AS27243 and 2724.6).

Prior to 1996, motor vehicle exhaust emissions were the major source of lead in New Zealand's environment. However, since the introduction of unleaded low octane petrol in 1987 and then unleaded premium petrol in 1996 the amount of lead in the air in urban areas has declined rapidly. However, there are still some industrial sources of lead such as secondary lead smelting, non-ferrous foundries and refining of aluminium and iron. These activities emit quantities of lead that need to be assessed, controlled and monitored.

Other sources of exposure to lead include lead based paint, lead pipes in household plumbing, and lead in solder. Although these uses have been phased out, not all the remaining sources have been removed. Paint removal from old houses continues to be a major potential health risk if not done appropriately. The Ministry of Health has developed guidelines on how to remove leaded paint safely.

Consequently, although lead levels have been reduced substantially in New Zealand to levels that are unlikely to cause adverse health effects, it is appropriate to continue to have a guideline value.

4 Ambient air levels

The dangers associated with exposure to high levels of hazardous air contaminants have been recognised for a long time, and well-established procedures are in place to limit worker exposure. Recognition that exposure to the generally much lower concentrations occurring in ambient air could also impact on public health has been much slower in developing. However, public concern about air pollution is now very strongly directed at hazardous air contaminants.

Monitoring of hazardous air contaminants in New Zealand has been relatively sparse and of variable quality. The reasons for this include the relatively recent recognition of the potential for these pollutants to adversely affect the community, and the high costs and technical difficulties of routinely measuring relatively low levels of a large number of chemicals. This situation is common to both Australia and New Zealand, to variable degrees. Measurements of hazardous air contaminants in Australia and New Zealand are summarised in a draft Victorian Environment Protection Authority report (1999a).

4.1 Benzene, toluene, xylene, and 1,3-butadiene

Measurements of benzene, toluene, xylene, and 1,3-butadiene have been reported by Stevenson and Narsey (1997 and 1998), NIWA (1997a) and Clarkson et al (1996).

During 1996–97 Stevenson and Narsey carried out a measurement programme for the New Zealand Ministry of Health for benzene, toluene, xylene, and 1,3-butadiene in Auckland (two locations), Christchurch (three locations), and Dunedin (two locations) (Stevenson and Narsey, 1997). Table 4.1 summarises the results for each city, presented as 24-hour maxima and minima and overall averages. For benzene, 24-hour samples were collected on a 1-in-6-day sampling programme for a 12-month period. For the other pollutants 4 to 6 months of data were obtained. The data appear to be referenced to 25°C.

Table 4.1: Concentrations of volatile hydrocarbons in New Zealand cities

City	µg/m^3, 24-hour average											
	Benzene			Toluene			Xylene			1,3-Butadiene		
	Max	Min	Ave	Max	Min	Ave	Max	Min	Ave	Max	Min	Ave
Auckland	21.7	0.3	2.6	67.6	0.2	8.8	26.1	0.6	5.6	0.13	0.00	0.015
Christchurch	67.6	0.2	9.7	224	1.4	34.0	132.5	0.7	59.4	1.03	0.00	0.14
Dunedin	14.0	0.1	4.2	52.6	1.7	12.9	43.6	1.6	10.8	0.09	0.00	0.04

The highest 24-hour average values in Christchurch were in a suburban shopping centre located on a busy arterial road.

The same authors conducted a further study for the Ministry of Health during 1997–98 (Stevenson and Narsey, 1998). Measurements of the aromatics benzene, toluene and xylene were undertaken in Auckland (five sites), Christchurch (six), Dunedin (three), Hamilton (two), and Paeroa (one). Table 4.2 shows the annual averages for each city/town, excluding locations heavily influenced by major roads (two in Christchurch, one in Dunedin) which are shown separately in Table 4.3.

Table 4.2: Concentrations of volatile hydrocarbons in New Zealand cities/towns

City/town	μg/m^3, annual average		
	Benzene	**Toluene**	**Xylene**
Auckland	1.3–3.8	3.6–11.6	3.9–10.2
Christchurch	2.9–6.2	7.4–17.1	6.8–14.3
Dunedin	1.6–2.6	4.0–6.9	3.1–6.6
Hamilton	1.0–2.6	3.4–6.9	2.3–6.6
Paeroa	1.8	3.9	3.6

Table 4.3: Concentration of volatile hydrocarbons in New Zealand near heavy traffic

City	μg/m^3, annual average		
	Benzene	**Toluene**	**Xylene**
Christchurch	11.6–20.1	30.0–49.9	31.1–52.9
Dunedin	6.3	20.0	17.0

The data above illustrate the influence of motor vehicles on local air quality. They also illustrate the role of meteorology on pollutant levels. Ambient levels of hazardous air contaminants in Christchurch are considerably higher than in Auckland, even though the population, and therefore, aggregate emission rates, are much lower. Because of the topography, ground-based inversions which trap pollutants and inhibit dispersion are much more common in Christchurch than in Auckland. New Zealand climate observations for the 1969 to 1998 period show that Christchurch has a lower annual mean daily temperature than Auckland (7.2 °C vs 12.4 °C), a higher number of ground-frost days (69 vs 6), and a lower mean wind speed 15 km/h vs 17 km/h).

For comparison, data for Australian cities (Victorian Environment Protection Authority, 1999a) and cities in the United States are given in Table 4.4.

Table 4.4: Concentrations of volatile hydrocarbons in Australian and US cities

City	µg/m^3, at 25^{o}C					
	Site type	Averaging time	Benzene	Toluene	Xylene	1,3-Butadiene
Melbourne	Urban	24-hour	0.64–7.3	3.4–11.7	>(1.7–4.3)	0.55
	Urban	Annual	≤(3.4–5.4)	≤6.4		
	Traffic	24-hour	2.2–8.0	4.5–18.1	0.87–2.6	
Sydney	Urban	48-hour	2.5–9.2	6.4–55.0	1.3–9.5	
	Traffic	1-hour	5.1–49.0	4.1–270		
Brisbane	Urban	30-min	0.03–1.7	0.15–41.5		
Perth	Urban	10–60-min	5.2	2.2	1.3	
	Traffic	10–60-min	16.0	34.3	25.6	
Adelaide	Traffic	3-minute	1.9–274	9.8–222	3.9–247	
	Traffic	1-hour	25.3	140		
Launceston		Annual	2.8	48	46	0.11
US cities		Annual	2.5–30			0.29–15.9
US average		Annual	6.8			0.64

As can be seen from the above tables, benzene concentrations (in µg/m^3) across New Zealand cities range from 0.10 to 68 (24-hour average) and 1.0 to 20 (annual average). The range across Australian cities is 0.64 to 49 (24-hour average) and across US cities 2.5 to 30 (annual average). The World Health Organization (1996) cite mean ambient concentrations (annual averages) for rural and urban areas of Europe as about 1 µg/m^3 and 5–20 µg/m^3 respectively.

4.2 Formaldehyde and acetaldehyde

The only available ambient air-quality data for formaldehyde in New Zealand are from a 17-day study of transport emissions by NIWA at Khyber Pass Road in Auckland (Kuschel et al, 1998). The 1-hour average formaldehyde concentration range was 7–29 µg/m3 and the 17-day average was 12 µg/m3. There are no available acetaldehyde data for New Zealand.

Data for Australian cities for both carbonyls are also limited. They have generally been collected over relatively short study periods (~1 to 6 months) using traditional and long-path UV monitoring. The available data (Victorian Environment Protection Authority, 1999a), are summarised in Table 4.5, as are data for cities in the United States (US Environmental Protection Agency, 1993b).

Table 4.5: Concentrations of carbonyls in Australian and US cities

City	µg/m^3 at 25^{o}C

	Site type	Averaging time	Formaldehyde	Acetaldehyde
Melbourne	Traffic	20-hour	0.5–7.5	0.7–4.7
Sydney	Landfill	24-hour max	9.4	
		Average	6.3	
Brisbane	Industry	30-min max	17.4	
		Average	9.2	
Adelaide	Traffic	1-hour max	24.6	
		Average	12.3	
	Industry	1-hour max	75.5	
		Average	22.0	
US cities		Annual range	1.8–5.8	2.4–3.9
		Overall average	3.15	2.4

4.3 Benzo(a)pyrene

There are few consistent measurements of benzo(a)pyrene (BaP) in either New Zealand or Australia. Measurements are reported for different types of locations, different averaging times, different sampling methods (gas, particle, or both), and different seasons, and are therefore difficult to compare. Kuschel et al (1998) report BaP levels of 1.3 ng/m^3 for Khyber Pass Road, a peak traffic site in Auckland, using continuous methods.

Ambient air 24-hour samples of particulate matter were obtained at three sites in Christchurch during July and August 1979 on days of poor air quality (days when air particulate matter exceeded 100 µg/m^3) and analysed for PAHs (Cretney et al, 1985). More than 40 species were identified, 26 of which were quantified. The ranges in reported pollutant levels were: 8 to 72 ng/m^3 for BaP; 110 to 871 ng/m^3 for quantified PAHs; and 107 to 373 µg/m^3 for air particulate matter. Principal component analysis and other methods using these and other data led the authors to conclude that domestic sources were the dominant contributor to PAHs on winter days.

For these data to be useful in assessing current ambient levels and relative source contributions, it would be necessary to determine what impact changes in fuel type and quality, better technology, and improvements in emission controls over the last 20 years, have had on emissions. This may be more difficult and less reliable than obtaining current data.

Levels (in ng/m^3) for various averaging times for Australian cities (Victorian Environment Protection Authority, 1999a) are shown in Table 4.6.

Table 4.6: Concentrations of benzo(a)pyrene in Australian cities

City and site information	ng/m^3	
	Sampling details	Result information
Melbourne, 1990/91, 1 site	24-hour, PM_{10} BaP	0.03–0.59, monthly

Melbourne, 1990/92, 2 sites	8-hour, $PM_{2.5}$ BaP, sampled on days of poor visibility	5.35–21.8, 8-hour
Sydney 1984/85, 4 sites	PM_{10} BaP	0.02–6.53, monthly
Brisbane 1994/95, 4 sites	PM_{10} and vapour BaP	0.05–1.95, 4-5 day
Perth 1994/95, 3 sites	24-hour, $PM_{2.5}$ BaP	0–19.7, 24-hour
Launceston 1991/93, 5 sites	24-hour, TSP and PM_{10} BaP	4.6–34.3, 24-hr maxima 0.5–3.8 (site means)

Sites in Launceston, Tasmania, which are heavily affected by wood smoke, are in the same range as the earlier Christchurch measurements.

In the US, several PAHs are routinely monitored by the state-wide Air Resources Board (ARB) air toxics network. The table below gives the network's mean concentration, in ng/m^3, of various PAHs from January 1996 through December 1996 (ARB, 1997c).

PAH Compound	Mean Concentration (ng/m^3)
Benzo[a]pyrene	0.194
Benzo[b]fluoranthene	0.245
Benzo[g,h,i]perylene	0.619
Benzo[k]fluoranthene	0.100
Dibenz[a,h]anthracene	0.031
Indeno[1,2,3-cd]pyrene	0.327

When benzo[a]pyrene was formally identified as a toxic air contaminant, the ARB estimated a population-weighted annual ambient concentration of 0.53 ng/m based on 1988 to 1989 monitoring data (ARB, 1994e).

Stevenson and Mills (1999) note that data from the U.K. monitoring network is similar to that from the Californian network. A rural site in the U.K. shows an average concentration of 0.2 ng/m^3 benzo(a)pyrene, while urban sites in Manchester, London and Middlesbrough give concentrations of about 0.5 ng/m^3 benzo(a)pyrene. An ambient air concentration of 0.2 ng/m^3 benzo(a)pyrene was used in their comparative assessment. Based on the California data, a concentration of 1 ng/m^3 is taken as the indoor exposure concentration.

4.4 Mercury, chromium and arsenic

A review of heavy metals in the New Zealand atmosphere was published over a decade ago (Steiner and Clarkson, 1985). This mainly focused on lead (which is now much less of an air-quality issue in New Zealand). Recent published data on ambient levels of mercury are limited for either Australia or New Zealand, although measurements have been made in areas of New Zealand in the past, mainly as part of geothermal investigations. Measurements of mercury vapour during the early 1980s at Ngawha Springs (a natural geothermal area at which a geothermal power station was planned) were in the range 13 to 47 ng/m^3, 7-day average, with a mean of 30 ng/m^3 (Brasell, 1982). Samples of mercury vapour taken in a "clean site" at Baring

Head, near Wellington, obtained concentrations of 0.5 to 1.3 ng/m^3, 7-day average, with a mean of 0.73 ng/m^3 (Bibby et al, 1988).). More recent but limited mercury monitoring has been undertaken in Ngawha and Rotorua using techniques that provide shorter averaging times. Results in Ngawha ranged from 2.8 to 21 ng/m^3 for averaging times of 5 to 8-hours (Timperley 1997-1998). In Rotorua levels were from < 0.6 to 1.5 ng/m^3 (0.0015 μg/m^3) in suburban areas and up to 20 ng/m^3 (0.02 μg/m^3) near areas of geothermal activity (Whakarewarewa) for 4 and 8-hour averages (Bates and Fellows 1998).

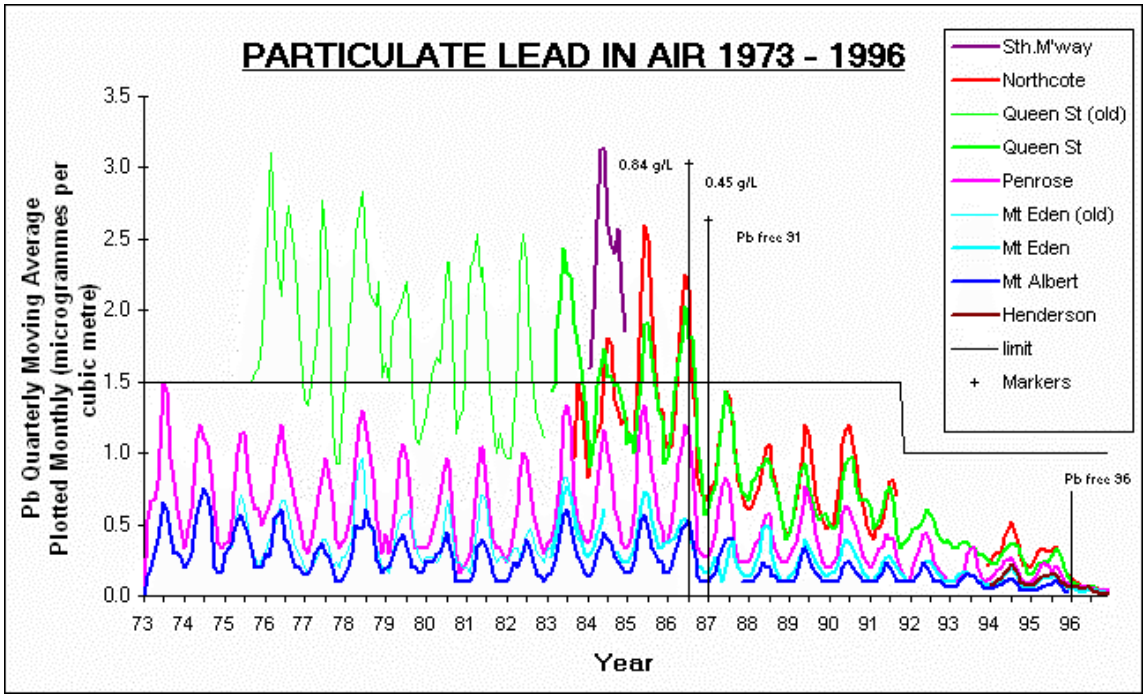
There is little New Zealand data available on ambient levels of chromium and arsenic, and few in Australia. Some data is available for two sites in Auckland, Penrose and Takapuna, covering a 6 month period. The levels of chromium and arsenic were very similar to data from the Californian and UK monitoring networks.

Data that have been collected are for metal compounds associated with $PM_{2.5}$ (Victorian Environment Protection Authority, 1999a), and are measured in terms of the metal. In New South Wales, fine particle ($PM_{2.5}$) 8-hour samples were taken twice weekly over 18 months (1992–93), at 24 sampling sites in Sydney, Newcastle, and Wollongong, and analysed for metals, including chromium. Mean chromium levels were 0.5 ± 0.4 ng/m^3. In Melbourne, 24-hour $PM_{2.5}$ samples were taken on a 1-in-6-day cycle during 1990–91 at two suburban sites, and 8-hour samples ($PM_{2.5}$ and $PM_{2.5-10}$) were taken near an arterial road in 1994. Mean arsenic and chromium levels at the suburban sites were 0.002 ng/m^3 and 0.008 ng/m^3 respectively. Mean chromium levels for the arterial road site were 0.002 ng/m^3 and 0.004 ng/m^3 in the fine and course particle fractions respectively.

4.5 Lead

Following the removal of lead additives from all petrol in 1996, atmospheric lead in New Zealand is now well below guidelines to protect human health (although as discussed previously inappropriate removal of lead based paint is still a significant public health problem). Lead in general air concentrations have reduced to about 0.2 μg/m^3, three-month moving average, calculated monthly, and are decreasing. This trend is likely to continue for a while, as residual contamination in petrol distribution systems and environmental contamination, caused by the addition of alkyl lead compounds to petrol for more than 30 years, decrease. Figure 1 clearly demonstrates the impact of Government policies to require lead-free petrol on lead levels in ambient air. The continuing decline in lead levels between 1987 and 1996, may also be attributed, at least in part, to the gradual increase in the market share of 91 as people switched from 96 leaded petrol to the unleaded, lower octane fuel.

Figure 4.1: Particulate lead in air at several monitoring sites in Auckland (1973 to 1996)
Source: Auckland Regional Council



Although overall urban concentrations have decreased and no longer pose a threat to human health, there may still be some "hot spots" around particular industries that may have the potential to exceed health guideline values.

The MoH reports that the weekly dietary exposure to lead has also decreased by almost 50% for all age-sex groups over the past decade (MoH, 2000). They link this decrease to government strategies for encouraging the food industry to implement new canning technologies to eliminate the use of lead solder in canned food and the reduction in lead additives in petroleum products. The weekly ingested levels are now less than 5% of the international Provisional Tolerable Weekly Intake for young males and 12% of the Provisional Tolerable Weekly Intake for young children.

5 Health effects and evaluation criteria

5.1 Approaches to setting guidelines for hazardous air contaminants

Several approaches are used for setting guidelines for hazardous air contaminants and these are explained in more detail in Annex C. They include:

- those focusing on technological controls of sources as the primary emphasis
- those relying principally on health risk evaluation
- those adapting guidelines from other countries
- those adapting occupational exposure standards.

5.1.1 Technological controls

It is beyond the scope of this review to provide a detailed assessment of different technological controls for controlling specific hazardous air contaminants. These are usually industry-specific, include management practices, are subject to technological developments, and to some extent, depend on economic considerations. However, a brief discussion of technological controls for motor vehicles and solid-fuel heaters is included in Annex C.

5.1.2 Health risk evaluation

The use of health risk evaluations for setting guidelines depends on combining dose-response data derived from epidemiological and/or toxicological studies of humans or animals with uncertainty factors. The main problem in setting ambient guideline values is in determining what is an acceptable risk, and the level of uncertainty in the risk estimate that one is prepared to accept. The acceptability or otherwise of a risk and the uncertainty involved are usually decided by each country or jurisdiction in conjunction with its citizens. Common considerations include economic and cost-benefit factors, the perceived importance of the risk, equity principles (who bears the risk and who derives the benefit), and available technological and other options.

A further consideration in applying ambient air guideline values for a specific substance is estimating the effects of different exposure routes on the effective dosage. Substances for which the effect is dependent on cumulative dosage (as is generally accepted to be the case with carcinogens), need to be considered in terms of exposure via ingestion of food and fluids, skin absorption, and inhalation, and also their reliance on target organs and the body mass balance (inputs minus losses).

Estimates of dosage via ingestion need to consider dietary habits; biological processes controlling absorption and transport in the body; the initial form of the species; its transformation to other, sometimes more toxic, species in the body; as well as the levels in different food substances. A related consideration is the fate of a chemical in the environment. For example, the main risk of exposure via inhalation may be very low for a particular

substance, but have a very high risk of exposure via other exposure routes because of deposition from the air to land or water. This is particularly important for substances such as mercury which bioaccumulate in the environment and the food chain, or are transformed to more toxic species.

5.1.3 Adapting guidelines from overseas

The approach taken in deriving the draft guidelines has been to adapt overseas guidelines where this is considered appropriate, or derive an ambient guideline value by selecting from appropriate overseas unit risk factors. An initial judgement has been made about what may constitute acceptable risk and level of uncertainty in New Zealand. This has been based on the US EPA classification of substances in terms of potency (high, medium, low), the range of risks considered acceptable (generally in the range 10^{-4} to 10^{-7}, also referred to as 1 in 10,000 to 1 in 10,000,000), consideration of guidelines applying or being modified by other agencies, and consideration of ambient air and emissions data, where available. The "acceptable risk" values resulting will not be uniform for all substances because of these different considerations, and have only taken into consideration exposure through inhalation.

Establishing a New Zealand consensus as to what constitutes an "acceptable risk" is a priority task. Clearly the recommended draft guidelines are dependent on the outcome of such a consensus. It is also highly desirable to develop guidelines on how the ambient air guidelines should be used where exposure for other routes is important, and this may entail developing a total exposure model.

The acceptable risk approach for setting ambient guideline values is most commonly used for longer averaging times and chronic effects. It can also be used for setting guideline values for short averaging times (usually less than 24-hour) aimed at preventing acute (short-term) effects.[6] The shorter averaging time guidelines are used by control agencies as a method of assessing and controlling intermittent emissions from industrial sources, since long-term averages do not adequately cover intermittent peak concentrations potentially causing acute effects.

An alternative method used by many jurisdictions for setting short-term guideline values is to use a power law approximation for converting guideline values at the longer averaging time to statistically equivalent concentrations at the shorter averaging time, and vice versa.

The following equation can be used:

$C_{T1}/C_{T2} = (T2/T1)^n$,

where C_{T1} and C_{T2} are concentrations for averaging times T1 and T2

and n is an exponent, usually taken as 0.2 in the absence of other data.

Different exponents ranging from 0.1 to 0.5 have been proposed depending on the distribution, of sources, source characteristics, their elevation, and local meteorology (Hibberd, 1998). The Victorian Environment Protection Authority uses 0.2 in its plume calculation procedure with the power law applied to dispersion coefficients used in dispersion models. The value of 0.2 has been used in this review.

[6] See the discussion in Annex C on RfCs, RfDs and RELs as used by the US EPA, and the California Air Resources Board.

5.2 Benzene

5.2.1 Health effects

Adverse health effects arising from exposure to benzene have been well documented and summarised by the World Health Organization (1993 and 1996), the US Environmental Protection Agency (1998a), the California Air Resources Board (1998a) and the UK Expert Panel on Air Quality Standards (1998a). The most significant chronic adverse effects from prolonged exposure to benzene are haemotoxicity, genotoxicity and carcinogenicity (World Health Organization, 1996).

Haemotological effects of varying severity have occurred in workers occupationally exposed to high levels of benzene. Decreased red and white blood cell counts in humans have been reported above median levels of approximately 120 mg/m^3. There is only weak evidence for effects below 32 mg/m^3, and no reported effects at 0.03–4.5 mg/m^3. Haemotological effects have also been demonstrated in mice chronically exposed (25 weeks) to concentrations as low as 32 mg/m^3 (World Health Organization, 1996).

Data from both animal and human exposures indicate that benzene is both mutagenic and carcinogenic. A number of studies indicate that exposure to benzene induces chromosomal changes in experimental animals, while in humans chromosomal effects have been demonstrated at mean workplace exposures of 4–7 mg/m^3. Increased mortality from leukaemia has been demonstrated in occupationally exposed workers, while multi-site carcinogenic effects have been observed in rats and mice exposed to high levels of benzene (320–960 mg/m^3), including tumours, lymphomas, and leukaemias.

Benzene has been classified as a Group A carcinogen of medium potency by the US EPA, and a Group 1 carcinogen by IARC (see Annex C).

Exposure to high levels of benzene through inhalation can result in acute toxic effects in humans (US Environmental Protection Agency, 1998a). Neurological symptoms include drowsiness, dizziness, headaches and unconsciousness, and exposure to very high levels can result in death. Exposure to liquid and vapour may cause irritation of the eyes, skin, and upper respiratory tract, while dermal exposure can result in blisters and redness. Co-exposure to benzene with ethanol can increase benzene toxicity. The LC50 in rats and mice is around 32,000 mg/m^3 (US Environmental Protection Agency, 1998a). Based on animal studies, the US EPA has assessed acute toxicity from benzene exposure to be low for inhalation, moderate for ingestion, and low or moderate for dermal contact.

Estimates provided by the Expert Panel on Air Quality Standards of the UK Department of the Environment, Transport and Regions (UK Expert Panel on Air Quality Standards, 1998a) are shown in Table 5.1.

Table 5.1: Estimates of daily intake of benzene, by source

Exposure source	Estimated daily intake (μg)
Ambient air – rural environment	15 μg
Ambient air – urban environment	400 μg
Cigarette smoke (20 per day)	600 μg
Food	100–250 μg
Water	1–5 μg

These estimates are based on a rural daily mean of 1.6 $\mu g/m^3$ and an urban maximum daily mean of 40 $\mu g/m^3$ (London). The potential exposure can range from 120 μg/day for a non-smoker in an unpolluted rural environment, to 1,250 μg/day for a 20 cigarettes per day smoker living in a city. For Europe, WHO estimate extended automobile travel (1-hour travel time) can contribute around 30% of cumulative ambient benzene exposure (World Health Organization, 1996). Inhalation is considered the dominant pathway for benzene exposure in humans.

5.2.2 Unit risk estimates

WHO has recently re-evaluated the human health exposure data for benzene (World Health Organization, 1996). It notes that different unit risk estimates result from different estimates of exposure and from different risk models, and that, in particular, concentration-dependent models yield a much lower risk estimate than models giving equal weight to concentration and exposure duration. Because much of the essential data for the concentration-dependent model are preliminary and need to be further developed and peer-reviewed, the latter model has been preferred by WHO. On this basis, the range of estimates of excess lifetime risk of leukaemia at an ambient air concentration of 1 $\mu g/m^3$ (unit risk) is in the range 4.4×10^{-6} to 7.5×10^{-6} (World Health Organization, 1996). By comparison, the US EPA estimates the unit risk for benzene inhalation as 8.3×10^{-6}, and California Air Resources Board as 29×10^{-6}.

5.2.3 Guidelines and standards

WHO's approach is to recommend guidelines fully protective of human health. For toxic substances, this is usually the no observed effect level (NOEL) derived directly from human exposure data, or indirectly by applying uncertainty factors to the no observed adverse effect level (NOAEL) or lowest observed adverse effect level (LOAEL) from animal or human exposure data. Because benzene is a carcinogen with an inferred effects threshold of zero, WHO recommends that jurisdictions establish their own guideline value by considering the unit risk factors and the level of risk considered acceptable in the jurisdiction.

The UK Panel recommended a benzene standard of an annual running average equivalent to 18 $\mu g/m^3$ (at 0°C)[7] for the UK. The panel has concluded that on the basis of US data on workers in the rubber and chemical industries, the increased risks of leukaemia would be too small to detect by any feasible study of workers exposed for a lifetime to a benzene

[7] Henceforth in this review all concentrations are expressed at 0^oC, and to a maximum of two significant figures.

concentration of 1,800 μg/m^3. The recommended standard is arrived at by applying a safety factor of 100 to account for differences in chronological and working life, and in susceptibility (as described in Annex C). This implies an additional leukaemia risk range of 7 to 12 per 100,000 being acceptable, if the WHO unit risk estimates are accepted, reducing to 1.5 to 2.5 per 100,000 at the lower concentration.[8]

The UK went on to establish the air quality goal or 5 ppb (16μg/m^3) annual average recommended by the EPAQS as an air quality objective under the Air Quality Regulations 1997 (HSMO, 1997). This is part of the National Air Quality Strategy which also recognises that exposures to benzene should be kept as low as possible because it is a genotoxic carcinogen. A review of the National Air Quality Strategy (UK DETR, 1999) has recommended that the date for achievement of the 5ppb objective in all areas, including roadside sites, be bought forward from the original 2005 date to 2003. The EPAQS target of 1 ppb (3.2μg/m3) is recommended to become an indicative level to be achieved in all locations, as far as practicable, by the year 2005. It is considered that measures already in place will ensure that this indicative level will be achieved at all urban background sites by 2005, but there is likely to be a considerable number of busy roads where further measures will be needed to achieve the 1ppb annual average level.

The European Commission has declared its intention to issue a directive for an ambient air-quality limit for benzene. In effect, a maximum value of 10 μg/m^3, annual average, at the time the directive comes into force is proposed, with progressive reduction to 5 μg/m^3 by the year 2010 (European Commission, 1998) implying an acceptable risk of 40 (current) and 20 (final) in one million, using the WHO lower unit risk factor.

In New Zealand, NIWA has recommended a maximum ambient guideline concentration for benzene of 18 μg/m^3 as a running annual average concentration (NIWA, 1997a), which is the same as the proposed UK standard.

Currently, the reference concentration (RfC) and oral reference dose (RfD) (US Environmental Protection Agency, 1993a) for benzene are under review by the US EPA.

California has established draft reference exposure levels (RELs) for acute exposure to benzene (1-hour average concentration), but these need to be reviewed by the Scientific Review Panel on Toxic Air Contaminants before being considered further. The draft RELs are shown in Table 5.2.

[8] The risk of developing an effect from exposure to a pollutant in ambient air is calculated by multiplying the ambient concentration by the unit risk factor. Thus for an ambient guideline concentration of 18 μg/m^3 (at 0°C) = 16.5 μg/m^3 (at 25°C), and the lower WHO unit risk factor of 4.4 x 10^{-6}, compliance with the guideline implies a risk of developing an effect of 7.2 x 10^{-5}. Conversely, if an acceptable risk is specified, then an ambient concentration for which that risk will not be exceeded can be calculated by dividing the acceptable risk value by the unit risk.

Table 5.2: Draft RELs for benzene

Effect	µg/m^3	
	Severity	**REL (1-hour average)**
Host resistance to L. monocytogens; Numbers of lymphocytes in the spleen	Mild adverse effect	780
Teratogenicity, foetal or maternal toxicity, foetal weight	Severe adverse effect	320
Lethality	Life-threatening	620,000

Source: California, Air Resources Board, 1998a.

5.2.4 Recommended evaluation criteria

A combination of the EC and UK approach seems appropriate for New Zealand, as follows: year 2000: 10 µg/m^3, annual average; year 2010: 3.6 µg/m^3, annual average.

Compliance with the criteria would be assessed by monitoring at "residential" sites. Sufficient monitoring should be conducted and used along with atmospheric dispersion models or other assessment tools, to characterise population exposure adequately.

One way of ensuring that new stationary sources are adequately controlled is to specify technological requirements in the form of emission limits, and/or control equipment as well as environmental management systems. Because benzene is a known carcinogen, a best available control technology (BACT) approach would be desirable. Residual emissions, after the application of BACT, could then be modelled to ensure adequate dispersion.

A short-term evaluation criterion (1-hour average) against which the results of dispersion modelling could be evaluated can be devised in one of three ways:

- derive a design level protective against acute impacts the California Air Resources Board approach. This would result in a design value in the vicinity of 780 µg/m^3, 1-hour average.
- convert the ambient air criterion to a 1-hour average in accordance with the formula $C_{T1}/C_{T2} = (T2/T1)^{0.2}$, and convert the 2010 annual guideline value of 3.6 µg/m^3 results in a 1-hour value of 22 µg/m^3, which is the recommended design guideline value. This is the recommended approach. For comparison, the Victorian Environment Protection Authority has a 3-minute average criterion of 110 µg/m^3, the 1-hour equivalent of which is 60 µg/m^3.
- require a risk assessment to be undertaken. A risk value consistent with the implied risk accepted by the criterion value (between 1 and 3 per 100,000) would seem appropriate.

5.2.5 Implications for control strategies

Based on current monitoring data, most cities in New Zealand are in compliance with the year 2000 criterion value. However, there are hot spots adjacent to major roads in Christchurch, where levels have been measured close to double the criteria concentration, although it is not clear whether these would result in high population exposures.

Areas in Auckland, Christchurch, and possibly in other cities, do not appear to meet the proposed 2010 criterion value. This is particularly so in Christchurch. Based on the emissions inventory data, the most obvious sources to control are motor vehicles, motor vehicle fuels, and solid-fuel heating appliances.

A range of control measures for minimising the impact of motor vehicles on air quality, including emission standards, are discussed in a report by the Ministry of Transport on the air-quality impacts from the road transport sector (Ministry of Transport, 1998). Initiatives to address the air quality situation in New Zealand are currently being implemented by the Ministry of Transport. These initiatives were based primarily on air quality monitoring data for carbon monoxide and nitrogen dioxide. The initiatives include a basket of measures including: emission standards for new vehicles and a review of the Petroleum Fuel Specifications.

At the time of the VFECS there was no formal ambient air quality guideline value for any of the hazardous air contaminants. VFECS initiatives will be reviewed and revised over time as more monitoring data comes to light and guideline values are developed.

General VOC emission standards will only partially control vehicle benzene emissions. At present New Zealand petrol can have an aromatic content of 48%, which is higher than other countries, and a benzene limit of 5%. Therefore to reduce benzene emissions the total aromatic content of petrol may need to be reduced, as well as possible reductions in the benzene content and the fuel volatility. This may entail changing the configuration of the Marsden Point oil refinery, and possibly other refineries supplying the New Zealand petrol market. The feasibility and timeframe for this requires further analysis and will be considered as part of the review of the Petroleum Fuel Specifications by the Ministry of Economic Development.

Stevenson and Narsey (1999) note that measures agreed as part of the EC Auto-oil programme include new emission limits which will apply to cars, vans and heavy duty vehicles sold from 2001 and from 2006. This programme also provides for a reduction of the benzene and aromatics content of petrol from 2000, and reduction of the sulphur content of fuels from 2000 and again from 2005. The reduction of the sulphur content of petrol will reduce the deterioration of catalyst performance with age, which is one of the limits on achieving low benzene emissions (UK DETR, 1999).

Improving the combustion efficiency of domestic heating appliances through better design standards, including emission standards, and through better operating practices, will decrease emissions of various species including benzene, and substantially reduce operating costs. Introducing standards for new appliances would only improve emissions and air quality if these replaced older, more polluting units. An active policy for scrapping and replacing older units may be necessary to achieve improvements in air quality.

5.3 Toluene

5.3.1 Health effects

Adverse health effects arising from exposure to toluene have been well documented and summarised by World Health Organization (1996), the US Environmental Protection Agency (1998a) and the California Air Resources Board (1998a).

A range of health effects have been associated with chronic and acute exposure to toluene, the most significant being those on the central nervous system (CNS). CNS dysfunction (often

reversible), cardiac arrhythmia and narcosis have been observed in humans from acute inhalation exposure to low to moderate levels of toluene. Symptoms include fatigue, sleepiness, headaches and nausea, with CNS depression and death occurring at higher exposure levels. Results from animal studies have also shown CNS effects and decreased resistance to respiratory infection following acute exposure (US Environmental Protection Agency, 1998a).

At high chronic exposures CNS depression has been reported, with symptoms including ataxia, tremors, cerebral atrophy, involuntary eye movements, and impaired speech, hearing and vision. CNS effects have been replicated in chronically exposed experimental animals. Chronic inhalation and dermal exposure have been observed to cause respiratory tract and eye irritation, nausea, sore throat, skin conditions, dizziness, headaches, and sleep disturbance in humans. Chronic exposure to high levels of toluene has been associated with nasal, respiratory and pulmonary tissue lesions in rats and mice. Mild adverse effects on the liver and kidneys of humans, and on the liver, kidneys and lungs of rodents, have been noted in chronically exposed subjects (US Environmental Protection Agency, 1998a).

Exposure to toluene may cause developmental decrements and congenital abnormalities in humans, and these have also been observed in animal studies. Hormonal changes have been observed in men occupationally exposed to toluene at 19–94 mg/m^3. Reproduction and hormonal imbalance have been observed in occupationally exposed women, including higher rates of spontaneous abortion and menstrual function disturbances. Higher rates of spontaneous abortions were also noted in pregnancies where there had been paternal but no maternal occupational exposure. CNS dysfunction, attention deficits, minor craniofacial and limb abnormalities, developmental delays, growth retardation, and dysmorphism have been observed in children and infants of pregnant women exposed to toluene or to mixed solvents. However, study results may been affected by a number of confounding factors (US Environmental Protection Agency, 1998a; World Health Organization, 1996).

The US EPA concludes that toluene is a developmental but not a reproductive toxicant (US Environmental Protection Agency, 1998a). Both the US EPA and IARC consider that toluene is not classifiable as a carcinogen (International Agency for Research on Cancer, 1998).

As discussed earlier, motor vehicles are the major source of toluene in ambient air. Indoor and outdoor sources of toluene also include the use of paints, lacquers and varnishes, glues, and other household products where toluene is a solvent, domestic and industrial surface coating, petrol refuelling, chemical plants, and oil refining. Indoor exposure can be significant, as is the case for many solvents and chemical cleaners.

5.3.2 Unit risk estimates

Toluene is not considered carcinogenic and unit risk factors do not apply.

5.3.3 Guidelines and standards

Based on occupational studies, WHO has determined that the LOAEL for CNS effects in workers is 350,000 $\mu g/m^3$. After applying an uncertainty factor of 1,200, the recommended ambient guideline value is 290 $\mu g/m^3$ averaged over 1 week. This converts to an annual average of 120 $\mu g/m^3$. A 30-minute average guideline value of 1,000 $\mu g/m^3$ based on odour effects is also recommended.

The US EPA RfD is 200 μg/kg/day, and the RfC is 400 $\mu g/m^3$ (US Environmental Protection Agency, 1993a).

The California draft acute (1-hour) RELs, under review by the Scientific Review Panel on Toxic Air Contaminants, are given in Table 5.3.

Table 5.3: Draft acute (1-hour) RELs for toluene

Effect	Severity	REL (1-hour average)
Headache, dizziness, feeling of intoxication, and slight eye and nose irritation	Mild adverse effect	37,000 μg/m^3
Foetotoxic effects	Severe adverse effect	46,000 μg/m^3

No recommendation for a severe (life-threatening) adverse effect is made.

In New Zealand, NIWA has recommended a maximum ambient concentration of 190 μg/m^3, running annual average (NIWA, 1997a). This converts to a 1-week average of 420 μg/m^3.

The Victorian Environment Protection Authority 3-minute criterion, based on odour effects, is 710 μg/m^3, which is equivalent to 370 μg/m^3, 1-hour average.

5.3.4 Recommended evaluation criteria

The NIWA-recommended maximum ambient concentration of 190 μg/m^3, running annual average, falls between the WHO and US EPA guidelines (120 μg/m^3 and 400 μg/m^3, respectively), when converted to the same time and temperature bases. 190 μg/m^3, annual average, therefore seems reasonable as a criterion to assess the results of monitoring at residential sites.

The above data suggest a short-term (1-hour average) concentration for assessing the results of dispersion modelling of between 370 and 1,000 μg/m^3, based on odour. A 1-hour guideline value of 500 μg/m^3 may therefore be appropriate for evaluating any new or existing industrial operations involving toluene. It should be noted that an annual average of 190 μg/m^3 approximately equates to 1,200 μg/m^3, 1-hour average.

5.3.5 Implications for control strategies

Based on existing monitoring data, the proposed criterion value is unlikely to be exceeded except at some hot spots particularly in Christchurch. The potential control measures described for benzene will also control toluene emissions.

For any industrial sources, the application of good control practice for existing sources, or better for new sources (BACT), in combination with appropriate design practices, is likely to provide adequate compliance with the suggested short-term evaluation criteria.

5.4 Xylene

5.4.1 Health effects

Adverse health effects arising from exposure to xylene have been well documented and summarised by the World Health Organization (1996 and 1997), the US Environmental Protection Agency (1998a) and the California Air Resources Board (1998a).

Although there may be different toxicities for different isomers of xylene, mixtures and individual isomers are normally treated as equivalent. The range of health effects in humans associated with acute inhalation exposure to xylene include dyspnoea (difficulty in breathing); irritation of the nose and throat; gastrointestinal effects such as nausea, vomiting and gastric discomfort; transient eye irritation; neurological effects such as impaired reaction time, impaired short-term memory, and performance decrements in numerical ability; and changes in equilibrium and body balance. Co-exposure to benzene and toluene indicates an enhanced effect on respiratory and neurological toxicity (US Environmental Protection Agency, 1998a).

Chronic exposure to xylene has resulted in a range of neurological effects such as headaches, dizziness, fatigue, tremors, and poor coordination. Other symptoms include laboured breathing, impaired pulmonary function, increased heart palpitation, severe chest pain, and an abnormal EKG. Animal studies indicate effects on the liver, blood, and kidneys, and possible blood and kidney effects have been reported in humans (US Environmental Protection Agency, 1998a).

Developmental effects have been observed in humans in coexposure with other solvents making it difficult to draw conclusions. Developmental effects have been shown to occur in animals exposed to xylene through inhalation. These include delayed ossification, decreased foetal body weight, and haemorrhages in foetal organs. Maternal toxicity (that is, toxicity to pregnant animals) has also been observed (US Environmental Protection Agency, 1998a). WHO considers developmental toxicity to be the critical endpoint for determining guidelines (World Health Organization, 1996).

There are no data on the carcinogenic effects of xylene on humans (US Environmental Protection Agency, 1998a). Animal and laboratory studies indicate negative mutagenicity or carcinogenicity for xylene (World Health Organization, 1996). Both the US EPA and IARC consider that xylene is not classifiable as a carcinogen (International Agency for Research on Cancer, 1998).

As discussed above, motor vehicles are the major source of xylene in ambient air. Indoor and outdoor sources of xylene include the use of paints, lacquers and varnishes, glues, and other household products where xylene is a solvent, domestic and industrial surface coating, petrol refuelling, chemical plants, and oil refining.

5.4.2 Unit risk estimates

Xylene is not considered carcinogenic and unit risk factors do not apply.

5.4.3 Guidelines and standards

WHO has determined a LOAEL based on developmental toxicity in rats of 950,000 $\mu g/m^3$. After applying an uncertainty factor of 1,000, the recommended ambient guideline value is 950 $\mu g/m^3$, averaged over 1 year. Developmental toxicity is considered the critical endpoint. For CNS effects a 24-hour average guideline value of 5,200 $\mu g/m^3$ is proposed, based on a NOAEL of 330,000 $\mu g/m^3$ for human volunteers, and an uncertainty factor of 60. A 30-minute average guideline value of 4,800 $\mu g/m^3$ based on odour effects is also recommended. WHO notes that a sensitive subset of the population may find the odour annoying at that concentration.

The US EPA RfD is 2,000 µg/kg/day (US Environmental Protection Agency, 1993a), and the RfC is under review. The California Air Resources Board has established a chronic REL of 330 $\mu g/m^3$ and an acute REL (currently under review) of 4,800 $\mu g/m^3$. A draft acute (1-hour) REL is 2,200 $\mu g/m^3$, which has a mild adverse effect as seen in eye, nose and throat irritation.

No recommendations are made for severe and life-threatening adverse effects because of limitations in the database.

In New Zealand, NIWA has recommended a maximum ambient concentration of 2,400 $\mu g/m^3$, running annual average (NIWA, 1997a).

The Victorian Environment Protection Authority 3-minute average criterion is 370 $\mu g/m^3$, based on xylene's odorous properties. This is equivalent to an annual average concentration of 33 $\mu g/m^3$.

5.4.4 Recommended evaluation criteria

The New Zealand recommended maximum ambient concentration of 2,400 $\mu g/m^3$, running annual average, is less stringent than the WHO value, and much less stringent than both the California Air Resource Board REL and the Victorian Environment Protection Authority's odour-based design value. An annual average criterion, to assess the results of monitoring at "urban residential" sites, of 950 $\mu g/m^3$ (the WHO value) seems appropriate.

It is more difficult to recommend a short-term (1-hour or less) average design concentration for xylene based on odorous properties, because of different estimates of the odour threshold, large individual differences in odour sensitivity, and no consensus on an appropriate averaging time. A 1-hour average concentration of between 200 and 4,000 $\mu g/m^3$ (Victorian Environment Protection Authority and WHO 1-hour equivalents) based on odour is an appropriate range. A 1-hour guideline value of 1,000 $\mu g/m^3$ would therefore be reasonable.

5.4.5 Implications for control strategies

Existing monitoring data indicate that ambient xylene levels are currently much lower than the proposed criteria values. There are therefore no implications for control programmes, although measures adopted to reduce benzene levels will also reduce xylene levels.

For industrial sources, the application of good control practice for existing sources, or better for new sources, in combination with appropriate design practices, should provide adequate safeguards against local exceedences of criteria values.

5.5 1,3-Butadiene

5.5.1 Health effects

Adverse health effects arising from exposure to 1,3-butadiene have been well documented and summarised by the World Health Organization (1996), the US Environmental Protection Agency (1998a), the California Air Resources Board (1998a), and the UK Expert Panel on Air Quality Standards (1998b). WHO considers carcinogenicity as the critical effect for derivation of air-quality guidelines.

The main route for 1,3-butadiene exposure in humans is inhalation. Adverse health effects in humans resulting from acute exposure include irritation of the eyes, throat, lungs and nasal passages; and neurological effects such as blurred vision, fatigue, headache and vertigo. Chronic non-cancer effects in exposed humans include cardiovascular and blood diseases. Animal studies have reported effects on the respiratory and cardiovascular systems, blood, and liver (US Environmental Protection Agency, 1998a).

There are no data on the reproductive or developmental effects on humans. Animal studies have reported a range of reproductive and developmental effects, including increased incidence of ovarian and testicular atrophy, skeletal abnormalities, and decreased foetal weights (US Environmental Protection Agency, 1998a).

Several epidemiological studies of workers in the styrene-1,3-butadiene industry have shown an increased incidence of respiratory, bladder, stomach, and lymphato-haematopoietic cancers (US Environmental Protection Agency, 1998). The US EPA considers that these studies are insufficient to demonstrate causality because of simultaneous exposure to other chemicals. Animal studies have reported tumours at multiple sites including the heart, lung, mammary gland, ovaries, forestomach, liver, pancreas, thyroid, testes, and haematopoietic system (California Air Resources Board, 1998a). The board notes that 1,3-butadiene is one of only two chemicals known to induce cancer in the hearts of laboratory animals.

1,3-butadiene has been shown to be mutagenic in both bacterial and mammalian systems (World Health Organization, 1996). The data indicate that the induction of cancers requires the metabolic activation of DNA-reactive metabolites. The 1,3-butadiene metabolites, epoxybutene and diepoxybutene, are both genotoxic and carcinogenic *in vivo*. *In vivo* and *in vitro* studies indicate that rats produce much lower levels of epoxides than mice, and are relatively insensitive to 1,3-butadiene carcinogenicity compared to mice. Humans are considered to be more similar to rats than mice in their response (World Health Organization, 1996). WHO note that a group of 40 individuals occupationally exposed to 1,3-butadiene concentrations of 2.4–7.2 mg/m^3 showed no significant increases in chromosome aberrations and other measures of genetic impacts, compared to a control group of 30, whereas an increased occurrence of the same measures occurred in mice exposed to 15 mg/m^3.

The US EPA classifies 1,3-butadiene as a Group B2 carcinogen of medium potency. The IARC classification is Group 2A (International Agency for Research on Cancer, 1998). The UK Expert Panel accepts that 1,3-butadiene is a genotoxic carcinogen (UK Expert Panel on Air Quality Standard, 1998b).

5.5.2 Unit risk estimates

Unit risk factors for 1,3-butadiene inhalation exposure adopted by various groups are as follows:

2.8 x10^{-4} per µg/m^3	US EPA, 1998
1.7 x 10^{-4} per µg/m^3	CARB, 1998a
0.7 x10^{-5} – 3.3 x10^{-5} per µg/m^3	Netherlands National Institute of Public Health and Environment, 1994.

The US EPA has classified 1,3-butadiene as a Group B2, *probable human carcinogen* of medium carcinogenic hazard (US EPA, 1992), based on animal studies. In its draft 'Health Risk Assessment of 1,3-Butadiene" (US EPA 1998a) the EPA proposed to reclassify 1,3-butadiene as a *known human carcinogen* based on increased leukaemia rates among synthetic rubber workers exposed to 1,3-butadiene (and other volatile organic compounds), additional animal studies, and animal and human metabolic studies.

However, this US EPA proposal was not supported by the majority of the US EPA's Scientific Advisory Board Environmental Health Committee (US EPA, 1998c). The majority considered the weight of evidence, particularly from studies in humans to be insufficient. This was due to the lack of consistency between exposure response rates for leukaemia or lymphosarcoma when both the SBR (exposed to several chemicals) and monomer (exposed only to 1,3-butadiene) studies

were considered in total. The majority of the Committee considered that 1,3-butadiene should remain classified as a *probable human carcinogen*.

IARC has recently re-evaluated 1,3-butadiene and re-confirmed the earlier classification, as *probably carcinogenic to humans (Group 2A)*. IARC considered that the epidemiological evidence for increased risk of leukaemia or lymphoma in exposed workers strongly suggested a hazard. But the published evidence did not allow consistency of findings to be evaluated among two or more studies of adequate statistical power. Therefore, the epidemiological evidence was evaluated as *limited*. IARC concluded that other relevant published data did not compel reclassification of 1,3-butadiene (IARC, in preparation).

Mathematically extrapolated additional cancer risks from exposures to 1,3-butadiene

The current US EPA inhalation unit risk estimate is 2.8×10^{-4} per $\mu g/m^3$ (0.45 ppb) based on a NTP 1984 study in B6C3F1 mice using a linearised multi-stage procedure (US EPA, 1992).

The US EPA in their 1998 draft updated "Health Risk Assessment of 1,3-Butadiene", considered that the best estimate of human lifetime extra cancer risk from chronic exposure to 1,3-butadiene was 9×10^{-3} per ppm (4×10^{-6} per $\mu g/m^3$) (US EPA, 1998a). This estimate was based on a linear extrapolation of the increased leukaemia risks observed in occupationally exposed workers.

The US EPA's Scientific Advisory Board Environmental Health Committee (US EPA, 1998c), recommended a number of further considerations relating to the quantitative risk assessment in the 1998 draft. This, combined with the *probable human carcinogen* classification and the differences in the nature of the cancer response between rodents (a variety of cancers) and humans (only leukaemia suggested) indicate that both of the unit risk factors may best be regarded as tentative. However, the more recent, human leukaemia-based unit risk must be considered the more reliable estimate. The predominant uncertainties appear to relate to whether the increased leukaemia incidence found in the studies of synthetic rubber workers are attributable to 1,3-butadiene, rather than to other chemical exposures, and accordingly the unit risk estimate based on this study may greatly over-estimate the cancer risks from 1,3-butadiene. If the earlier, mouse cancer-based unit risk applied, a cancer incidence increase about 70 times higher than found would be expected in the synthetic rubber workers study.

5.5.3 Guidelines and standards

WHO considers that the uncertainties in current estimates of carcinogenic risks to humans do not allow a specific guideline to be recommended. Given that there is some equivocal evidence of carcinogenicity, prudence should be exercised in developing ambient air-quality guidelines/standards.

The UK Expert Panel has recommended a 1,3-butadiene standard of an annual running average of 2.4 $\mu g/ m^3$ (UK Expert Panel on Air Quality Standards, 1998b). The panel concluded that on the basis of current data, the increased risks of lymphomas and leukaemias would be unlikely to be detectable by any practicable means in workers from lifetime exposure to 2,400 $\mu g/m^3$ of 1,3-butadiene. The recommended standard is arrived at by applying a safety factor of 100 to account for differences in chronological and working life, and in susceptibility (as previously described). The panel believes that standards for genotoxic carcinogens should be set as low as practicable. Since ambient levels in the UK on current data have not exceeded 2.4 $\mu g/m^3$, the panel has recommended this level as the standard, implying an additional safety factor of 10. The additional carcinogenic risk using the US EPA unit risk factor is 6.2 per 10,000, which is

the implied acceptable risk. The risk value using the Netherlands unit risk factors ranges from 1.6 to 7.4 per 100,000.

The US EPA has not established an RfC or RfD for 1,3-butadiene. California has also not established an REL for acute exposure to 1,3-butadiene (1-hour average concentration) (see California EPA, 1997).

In New Zealand the recommended maximum ambient concentration for 1,3-butadiene derived by NIWA (1997a) is a running annual average concentration of 24 $\mu g/m^3$, which is 10 times the proposed UK standard.

The Victorian Environmental Protection Authority 3-minute average criterion, based on odour effects of 1,3-butadiene, is 1,100 $\mu g/m^3$. On an annual basis this equates to about 90 $\mu g/m^3$. (The Victorian EPA value is due for review.)

5.5.4 Recommended evaluation criteria

As discussed above, the evidence that 1,3-butadiene is a genotoxic carcinogen is ambivalent, but has been accepted by expert panels in the United Kingdom and in the US, and by IARC. A precautionary approach in setting ambient criteria values in New Zealand is therefore warranted. An annual average criterion of 2.4 $\mu g/m^3$, to be assessed by monitoring at "urban residential" sites, would be appropriate, and is consistent with the UK.

As for benzene, a way of ensuring that new stationary sources of 1,3-butadiene are adequately controlled may entail a combination of technological requirements and short-term (1-hour) design criterion levels. Because of its status as a possible carcinogen, it would be desirable to use a BACT approach for minimising emissions. The approach should apply to new plant, and be phased in over a period for existing industrial premises. Residual emissions after the application of BACT could then be modelled to ensure adequate dispersion.

As previously discussed, one approach for deriving a short-term criterion value, against which the results of dispersion modelling could be evaluated, is to convert the annual average value to a 1-hour value. This would result in a 1-hour concentration of 15 $\mu g/m^3$. The second approach would be to require a risk assessment be undertaken. A risk value consistent with the implied acceptable risk in the guideline value of ~6 per 10,000 would seem appropriate.

The former approach is recommended as being simpler and more appropriate, given the current ambiguities in the carcinogenic status of 1,3-butadiene.

5.5.5 Implications for control strategies

Based on the current very limited monitoring data, all cities in New Zealand appear to be well within the recommended criteria. The maximum 24-hour value, equivalent to 1.1 $\mu g/m^3$, recorded in Christchurch, is less than half the recommended annual average concentration, and one fourteenth the recommended 1-hour design value. That suggests the criteria should have no implications for regional strategies but, because the monitoring is very limited, caution is warranted.

Emissions of 1,3-butadiene from motor vehicle exhausts are believed to vary with the level of olefins in petrol (emissions of benzene vary with the aromatic content). In order for motor vehicles to run correctly, a minimum octane number is required. Since, the aromatic and olefin content of petrol increase its octane number, lowering the aromatic content of petrol to reduce emissions of benzene is in competition with the lowering the olefin content to reduce emissions

of 1,3-butadiene. One solution to the problem is to use catalytic converters, which are efficient at reducing 1,3-butadiene emissions. Future developments in other technologies for emissions abatement may also be effective at reducing 1,3-butadiene emissions.

Another approach may be to increase the octane number of petrol by increasing the branched chain alkane content. This requires a reconfiguration at an oil refinery and will be considered within the review of the Petroleum Fuel specifications. Although this would be contrary to the findings of Ye et al. (1997) who assessed the contributions from different petrol components to 1,3-butadiene formation, and concluded that over 90% of 1,3-butadiene emissions originate from the common alkane and aromatic fractions of petrol. Accordingly, they state that there is no easy way to manipulate fuel composition to reduce 1,3-butadiene concentrations in raw engine exhaust. This also means that the variations in fuel composition, such as the proportions of olefins have little effect on 1,3-butadiene concentrations in exhaust gases.

Actions to improve combustion processes for solid-fuel heating appliances would also reduce 1,3-butadiene emissions and reduce ambient levels.

5.6 Formaldehyde

5.6.1 Health effects

Adverse health effects arising from exposure to formaldehyde have been well documented by the World Health Organization (1989a and 1996), the US Environmental Protection Agency (1998a), and the California Air Resources Board (1998a).

The major route for exposure in humans is inhalation, and the main toxic effects for acute exposure to formaldehyde are eye, nose and throat irritation and effects on the nasal cavity. Other effects are coughing, wheezing, chest pains, and bronchitis. Chronic exposure has also been associated with respiratory symptoms and eye, throat and nose irritation; animal studies have reported effects on the nasal epithelium and lesions on the respiratory system. An increased incidence of menstrual disorders and pregnancy problems has been observed in women workers using urea-formaldehyde resins, but there may have been confounding factors involved. A study of workers exposed to formaldehyde through sterilising equipment did not note increased incidence of spontaneous abortions, and no developmental effects have been observed in animal studies (US Environmental Protection Agency, 1998a).

WHO notes that there is substantial inter-individual variability in human formaldehyde responses. Significant increases in signs of irritation occur at levels above 0.1 mg/m^3 in healthy subjects, and a progression of symptoms occur above 1.2 mg/m^3. No lung function alterations were noted in healthy non-smokers and asthmatics exposed to formaldehyde levels up to 3.7 mg/m^3, leading to the interpretation that the observed effects were related more to peak than to mean concentrations (World Health Organization, 1996).

A highly significant increase in nasal cancer was found in rats exposed to 17 mg/m^3 of formaldehyde, but the dose-response curve was non-linear. A range of other analyses and observations led to the conclusion that hyperproliferation induced by cytotoxicity is likely to play a significant role in the formation of nasal tumours by formaldehyde. WHO assesses the evidence that high concentrations of formaldehyde can induce nasal cancer in rats and possibly mice as convincing. Formaldehyde has been shown to be genotoxic in a variety of *in vitro* and *in vivo* systems. There is also epidemiological evidence associating relatively high occupational exposure with nasopharyngeal and sinonasal cancers. Simultaneous exposure of

humans to other respiratory tract toxicants such as acrolein, acetaldehyde and ozone may lead to additive or synergistic effects, particularly for sensory irritation and possibly cytoxicity to the nasal mucosa (World Health Organization, 1996).

Formaldehyde has been classified as a Group B1 carcinogen of medium potency by the US EPA, and a Group 2A carcinogen by IARC (International Agency for Research on Cancer, 1998).

5.6.2 Unit risk estimates

In reviewing the evidence, WHO concluded that it would be reasonable to assume that the response of the human tract mucosa to formaldehyde would be similar to that of the rat, and that, provided that the respiratory tract tissue is not repeatedly damaged, the cancer risk in humans from exposure to low, non-cytotoxic concentrations of formaldehyde is negligible. Accordingly, WHO does not specify a guideline unit risk value.

Unit risk factors for formaldehyde inhalation exposure adopted by other groups are as follows:

1.3×10^{-5} per $\mu g/m^3$	US EPA, 1998a
6.0×10^{-6} per $\mu g/m^3$	CARB, 1998a.

5.6.3 Guidelines and standards

The WHO ambient air-quality guideline value is 100 $\mu g/m^3$, 30-minute average, for protection of the general population. This is based on a NOAEL of 100 $\mu g/m^3$ and an uncertainty factor of 1. WHO also recommend that for groups within the general population that show hypersensitivity reactions without immunological signs, the formaldehyde concentration should be kept to a minimum and not exceed 10 $\mu g/m^3$, 30-minute average.

The US EPA has not established an RfC for formaldehyde. The current 1-hour average RELs established by the CARB are 3.6 $\mu g/m^3$ (chronic) and 36 $\mu g/m^3$ (acute). However, the acute exposure RELs are under review by the Scientific Review Panel on Toxic Air Contaminants. Draft values are as shown in Table 5.4.

Table 5.4: Draft acute exposure RELs for formaldehyde

Effect	**$\mu g/m^3$**	
	Severity	**REL (1-hour average)**
Mild and moderate eye irritation	Mild adverse effect	310
FEV1 decrements >20%	Severe adverse effect	2,000
Lethality	Life-threatening	13,000

In New Zealand, NIWA (1997a) concludes that there are insufficient data to develop a recommended maximum acceptable concentration, and instead recommend minimising all emissions where practicable. The Victorian Environment Protection Authority 3-minute average criterion is 55 $\mu g/m^3$.

5.6.4 Recommended evaluation criteria

The WHO recommendations seem appropriate for New Zealand. Converting the 30-minute average concentration of 100 $\mu g/m^3$ to a running annual average produces an evaluation criterion of 15 $\mu g/m^3$, to be assessed by monitoring at "urban residential" sites.

An alternative approach based on unit risk values and an acceptable risk of 1 in 10,000, results in an annual average concentration of 8–17 $\mu g/m^3$. The ambiguity indicated by WHO on the carcinogenic risk to humans of exposure to "non-cytotoxic concentrations" indicates that the previous approach is preferable, but similar criteria would ensue.

Ensuring that stationary sources of formaldehyde are adequately controlled may entail a combination of technological requirements and a short-term (1-hour) design ambient concentration level for ensuring adequate dispersion of residual emissions. A high level of control is desirable for new sources, given the possibility of adverse effects among highly sensitive groups, as highlighted by WHO. Existing industrial sources may require similar levels of control to be phased in over a suitable timeframe.

The above data suggest a short-term (1-hour average) design concentration of between 10 and 95 $\mu g/m^3$. Therefore, 20 $\mu g/m^3$, 1-hour average, may be an appropriate criterion for evaluating the results of dispersion modelling at industrial operations involving formaldehyde. For comparison, the Victorian Environment Protection Authority value is equivalent to a 1-hour concentration of 29 $\mu g/m^3$.

5.6.5 Implications for control strategies

The NIWA monitoring data for Khyber Pass Road in Auckland (Kuschel et al, 1998) are equivalent to 1-hour average formaldehyde levels in the range 8–30 $\mu g/m^3$, indicating an annual average of 1–5 $\mu g/m^3$, which are well below the proposed criteria values.

However, photochemical smog reactions can generate significant levels of formaldehyde, and the above data probably do not fully represent formaldehyde levels formed in the atmosphere, but only direct vehicle emissions. The CARB estimates that up to 88% of formaldehyde in California is photochemically formed, and also reports network average levels of 4 $\mu g/m^3$ (California Air Resources Board, 1998a). Given the very much lower photochemical smog potential in New Zealand cities, control of VOC emissions from motor vehicles will probably ensure that ambient air levels remain below the recommended criteria values, but this should be ascertained by further monitoring.

A high level of control technology for new and some existing stationary sources may be necessary to avoid or remedy local hot spots of formaldehyde. These are most likely to be associated with fibre board production, and some resin and latex manufacturing and applications plant.

Controls introduced to reduce VOC and PAH emissions from domestic solid-fuel appliances would also lower ambient formaldehyde emissions.

5.7 Acetaldehyde

5.7.1 Health effects

Adverse health effects arising from exposure to acetaldehyde have been well documented and summarised by the World Health Organization (1995 and 1996), the US Environmental Protection Agency (1998a), and the California Air Resources Board (1998a).

The major route for exposure in humans is inhalation. The major toxic effects for acute exposure to acetaldehyde are eye, nose, skin and respiratory tract irritation; erythema; coughing; pulmonary oedema; and necrosis. Extremely high concentrations can cause respiratory paralysis and death. Depressed respiratory rates and elevated blood pressure have been observed in animals exposed to high concentrations of acetaldehyde. Chronic intoxication of acetaldehyde in humans can produce symptoms resembling alcoholism.

Hamsters chronically exposed via inhalation to acetaldehyde displayed changes in the nasal mucosa and trachea, growth retardation, slight anaemia, and increased kidney weight. There are no available data on reproductive or developmental effects of acetaldehyde exposure in humans. However, animal studies have shown that acetaldehyde can cross the placenta to the foetus, and that acetaldehyde may be a potential developmental toxin. Human data regarding the carcinogenic potential of acetaldehyde are inadequate, but an increased incidence of nasal tumours in rats and laryngeal tumours in hamsters has been observed following acetaldehyde inhalation exposure (US Environmental Protection Agency, 1998a).

Acetaldehyde has been classified as a Group B2 carcinogen of low potency by the US EPA, and a Group 2B carcinogen by IARC (International Agency for Research on Cancer, 1998).

5.7.2 Unit risk estimates

Unit risk factors for acetaldehyde inhalation exposure adopted by various groups are as follows:

2.2×10^{-6} per $\mu g/m^3$	US EPA, 1998a
2.7×10^{-6} per $\mu g/m^3$	CARB, 1998a
$1.5\text{-}9 \times 10^{-7}$ per $\mu g/m^3$	WHO, 1996.

5.7.3 Guidelines and standards

The WHO ambient air-quality guideline value is 2,200 $\mu g/m^3$, 24-hour average, based on a NOEL for irritancy to humans of 45,000 $\mu g/m^3$ and an uncertainty factor of 20.

The US EPA RfC for acetaldehyde is 9 $\mu g/m^3$. The CARB chronic REL is also 9 $\mu g/m^3$. No acute REL has been established.

In New Zealand, NIWA (1997a) concludes that there are insufficient data to develop a recommended maximum ambient concentration (RMAC) for acetaldehyde, and recommend instead that all emissions be minimised where practicable. The Victorian Environment Protection Authority 3-minute average criterion, based on odour effects, is 82 $\mu g/m^3$. It is currently under review.

5.7.4 Recommended evaluation criteria

The WHO unit risk estimates are 2.5 to 14 times lower than those of the US EPA. However, the WHO guideline value does not consider cancer as the health endpoint. On the other hand,

the US EPA RfCs may be unduly conservative given the inadequate database. Using the WHO upper risk level of 9 x 10^{-7} per $\mu g/m^3$, and an acceptable carcinogenic risk of between 1 in 10,000 and 1 in 100,000, suggests annual average guideline values in the range 12–120 $\mu g/m^3$. An evaluation criterion value of 30 $\mu g/m^3$, annual average, would therefore seem appropriate for New Zealand.

An appropriate health-based design guideline value for assessing residual emissions would be a 1-hour average level of 180 $\mu g/m^3$. However, it may be more appropriate to establish a guideline concentration value based on odour. The Victorian Environment Protection Authority has a current 3-minute average criterion, subject to review, of 82 $\mu g/m^3$ based on odour, which equates to a 1-hour average equivalent design level of 45 $\mu g/m^3$. This is the recommended value for assessing the results of dispersion modelling.

5.7.5 Implications for control strategies

There are no available acetaldehyde data for New Zealand, but based on overseas data ambient levels of acetaldehyde should be around the same as those for formaldehyde. On this basis, it is unlikely that control measures, other than those necessary to reduce emissions of other VOCs and PAHs, would be required to maintain ambient levels of acetaldehyde below the proposed criteria.

Controls beyond those applied to stationary sources and domestic solid-fuel appliances for reducing formaldehyde emissions are unlikely to be required for controlling local impacts of acetaldehyde, since the major sources of each chemical are common to both.

5.8 Benzo(a)pyrene

5.8.1 Health effects

Adverse health effects arising from exposure to benzo(a)pyrene (BaP) have been well documented and summarised by the World Health Organization (1996 and 1998), the US Environmental Protection Agency (1998a), and the California Air Resources Board (1998a). WHO considers carcinogenicity as the critical endpoint for the derivation of air-quality guidelines.

There are no human data on the effects of acute exposure to BaP and other PAHs. Animal studies have reported effects on the gastrointestinal tract and increases in liver weights from oral exposure to several PAHs. Chronic exposure to BaP in humans has resulted in dermatitis, photosensitisation, eye irritation, and cataracts. Effects on the blood and liver from oral exposure, and on the immune system from dermal exposure, have been noted in animals. There are no data on reproductive or developmental effects in humans. Studies of oral exposure to BaP in animals have noted the induction of reproductive toxicity, including reduced fertility, and developmental effects such as reduced viability of litters and reduced mean pup weights (US Environmental Protection Agency, 1998a).

Epidemiological studies have reported increases in lung cancer in humans from exposure to coke oven and roof tar emissions and cigarette smoke, all of which contain a number of PAHs. Animal studies have reported respiratory tumours following inhalation exposure to BaP, and forestomach and lung tumours and leukaemia following oral exposure (US Environmental Protection Agency, 1998a).

The lung carcinogenicity of BaP can be enhanced by co-exposure to other substances such as cigarette smoke, asbestos, and probably airborne particles (World Health Organization, 1996). A number of studies have shown that the benzene-soluble fraction of condensates from petrol and diesel vehicle exhaust, domestic coal stove emissions and tobacco smoke, containing 4-7 ring PAHs, account for nearly all the carcinogenic potential of PAHs from these sources. WHO notes that the carcinogenicity of PAH mixtures may be influenced by other compounds emitted with PAHs during incomplete combustion, and also points out the poor quality of available data sets from which to derive a risk assessment for BaP (World Health Organization, 1996). WHO also notes that the carcinogenic 4-7 ring compounds in ambient air are preferentially bound to particles, that only a minor fraction (depending on temperature) exists as volatiles, and that some studies indicate that the toxicokinetics of inhaled BaP attached to particles and pure BaP are different.

The US EPA has classified BaP as a Group B2 carcinogen of medium potency. The IARC classification is Group 2A (International Agency for Research on Cancer, 1998).

5.8.2 Unit risk estimates

The US EPA has not determined an inhalation unit risk for BaP. A unit risk for ingestion in drinking water is 2.1 $x10^{-4}$ per µg/L (US Environmental Protection Agency, 1993b).

WHO has determined an inhalation unit risk of 8.7 x 10^{-2} per µg/m^3 BaP, based on interpolation from risk estimates for PAHs in coke oven emissions. WHO has also determined an inhalation unit risk from studies of animals exposed to complex mixtures of PAHs of 2 x 10^{-5} per µg/m^3 BaP 10^{-5} per ng/m^3 (World Health Organization, 1996).

5.8.3 Guidelines and standards

WHO recommends that unit risks be used as a basis for setting ambient air-quality guidelines. The value developed from human exposure, 8.7 x 10^{-2} per µg/m^3, is appropriate.

The US EPA has not established an RfC or an inhalation RfD for BaP. California has also not established an REL for acute exposure to BaP.

In New Zealand, NIWA (1997a) concludes that there are insufficient data to develop a recommended maximum ambient concentration (RMAC), but recommends minimising all emissions where practicable. The Victorian Environment Protection Authority does not have a 3-minute average criterion for BaP.

5.8.4 Recommended evaluation criteria

In order to establish an ambient criterion value, the risk that would be acceptable to the New Zealand community needs to be established. A level between 1 in 10,000 and 1 in 100,000 would seem appropriate. The risks implicit in the recommended concentration values for benzene and 1,3-butadiene lie within this range. Using the WHO unit risk values, this implies annual average ambient air concentrations for BaP in the range 0.12 to 1.2 ng/m^3. A criterion of 0.30 ng/m^3, annual average, for BaP is therefore recommended.

Based on the limited data for New Zealand and typical levels found elsewhere there is a significant chance that existing (possibly background) levels exceed this recommendation.

The main sources of PAHs in the New Zealand urban environment are domestic solid-fuel combustion and motor vehicles. A 1-hour design value would be of very limited use since

controls of these sources rely on emission and equipment design standards and not on stack dispersion criteria, and is probably not warranted. If required, a 1-hour design value for BaP could be established as for other pollutants.

5.8.5 Implications for control strategies

The limited recent data suggest annual average ambient air levels of BaP around a few nanograms per cubic metre at hot spots in New Zealand. Earlier data for Christchurch obtained during 1979 (Cretney et al, 1985) indicated worst-case, short-term (daily) concentrations ranging from 9 to 72 ng/m^3. The data are for high-pollution days (defined as days when particle concentrations were greater than 100 μg/m^3) and were largely attributed to residential sources. Clearly, reducing ambient levels necessitates better control of smoke and particle emissions from various sources. As indicated by the early Christchurch data, wood and coal heating appliances are the primary targets, but petrol and diesel vehicle emissions are also sources. Emission standards for motor vehicles and domestic heating appliances, as well as improved fuel quality and operation and maintenance, are all required to keep concentrations of BaP to a minimum. Industry controls via BACT for any new sources, and good practice for existing sources, are appropriate.

5.9 Mercury and mercury compounds

5.9.1 Health effects

Adverse health effects arising from exposure to mercury and its compounds have been well documented and summarised by the World Health Organization (1989b and 1996), the US Environmental Protection Agency (1998a), and the California Air Resources Board (1998a). Elemental mercury exists almost totally in the gas phase in the atmosphere, as does methyl mercury, while inorganic mercury compounds are usually particle-bound (California Air Resources Board, 1998a).

Acute inhalation exposure to high levels of elemental mercury in humans results in CNS effects such as hallucinations, delirium, and suicidal tendencies, and gastrointestinal effects and respiratory effects such as chest pains, dyspnoea, cough, pulmonary function impairment, and interstitial pneumonitis. Acute exposure to high levels of methyl mercury also results in CNS effects including blindness, deafness, impaired level of consciousness and death. The effects of chronic exposure to elemental mercury include CNS effects such as erethism, irritability, insomnia, severe salivation, gingivitis and tremor, kidney effects including proteinuria, and acrodynia in children. The primary effect of chronic exposure to methyl mercury is CNS damage, while chronic exposure to inorganic mercury induces kidney damage (US Environmental Protection Agency, 1998a).

Studies of the effects on human reproduction and development from exposure to inorganic mercury are ambivalent. There is no information on reproductive and developmental effects on humans, but animal studies have reported effects including testicular changes, and developmental abnormalities. Studies on the carcinogenic effects of elemental mercury on humans are inconclusive. Chronic exposure to inorganic mercury (mercuric chloride) resulted in an increased incidence of forestomach and thyroid cancer in rats and mice, and renal tumours in mice. No studies are available on the carcinogenic effects of methyl mercury on humans, but one animal study reported renal tumours in mice (US Environmental Protection Agency, 1998a).

The US EPA has classified inorganic and methyl mercury as Group C carcinogens, and elemental mercury as Group D (unclassifiable). IARC has classified methyl mercury compounds as a Group 2B carcinogen, and mercury and inorganic compounds as Group 3 (unclassifiable) (International Agency for Research on Cancer, 1998).

5.9.2 Unit risk estimates

No unit risk factors are available for mercury and mercury compounds. Their status as carcinogens is ambivalent.

5.9.3 Guidelines and standards

The WHO recommended guideline for inorganic mercury is 1 $\mu g/m^3$ as an annual average. This is based on a LOAEL for renal tubular effects on humans of 20 $\mu g/m^3$ and an uncertainty factor of 20.

The US EPA RfC for elemental mercury is 0.3 $\mu g/m^3$, and the RfD for methyl mercury is 0.3 µg/kg/day (US Environmental Protection Agency, 1993a).

The California Air Resources Board RELs are as follows:

Elemental mercury	0.3 $\mu g/m^3$ (chronic REL)
Inorganic mercury and mercury compounds	30 $\mu g/m^3$ (acute REL)
Methyl mercury	1 $\mu g/m^3$ (chronic REL).

The acute REL for inorganic mercury is under review, and a draft value of 1.8 $\mu g/m^3$ is to be reviewed by the Scientific Review Panel on Toxic Air Contaminants. Appears now to be the REL (California EPA, 1999).

In New Zealand, NIWA has recommended the following maximum running annual average ambient concentrations (RMACs), expressed as mercury (NIWA 1997a):

Alkyl compounds	0.025 $\mu g/m^3$
Elemental and inorganic compounds	0.025 $\mu g/m^3$
Aryl compounds	0.010 $\mu g/m^3$.

These appear to be at variance with the listing of intended changes to the New Zealand Workplace Exposure Standards 1994 (WES), and the methodology for deriving the RMACs. The RMACs derived from the WES values are 0.1, 0.25 and 1.0 $\mu g/m^3$ for alkyl, elemental, and aryl compounds, respectively. The occupational health standard for alkyl mercury in the USA is 10 $\mu g/m^3$, and 50 $\mu g/m^3$ for mercury vapour (US Environmental Protection Agency, 1998a).

The Victorian Environment Protection Authority 3-minute average criteria are 0.33 $\mu g/m^3$ for organic mercury and 1.8 $\mu g/m^3$ for inorganic mercury.

5.9.4 Recommended evaluation criteria

In order to make the interpretation simpler, it is proposed that criteria values be established for two groups of mercury substances: an inorganic group (including elemental mercury) and an organic group.

The US EPA RfC and the CARB REL values for elemental mercury are close to the New Zealand WES-derived RMAC value for inorganic mercury. A criterion for monitoring of 0.33 $\mu g/m^3$ is therefore appropriate as an annual average.

The monitoring criterion for organic mercury can be derived from the value for inorganic mercury by scaling according to the occupational health standards. Thus the appropriate concentration for organic mercury, based on New Zealand WES values, is 0.13 $\mu g/m^3$, annual average.

The ambient guideline concentrations can be used as the basis for short-term criteria, by converting these to the appropriate time period using the formula with the power of 0.2 factor. The 1-hour average values so derived are 2.0 $\mu g/m^3$ for inorganic mercury and 0.80 $\mu g/m^3$ for organic mercury. These fit well within the Californian acute REL for inorganic mercury and mercury compounds.

The above levels should be viewed as applicable where exposure to mercury is mainly through inhalation. They may need to be adjusted downwards where dietary intake is significant. Mercury in fish and in drinking water has proved significant in some locations, and dietary intake of mercury should be established before a final guideline value for air is adopted.

5.9.5 Implications for control strategies

There is limited published ambient air-quality data for New Zealand that could be used to assess the control implications of the proposed criteria. Data for the USA indicate overall mean ambient levels of 0.006 $\mu g/m^3$, and 0.002 $\mu g/m^3$ in California. On this basis it is unlikely that regional-scale levels of mercury in New Zealand would require any control action, but this would need to be confirmed by emissions inventories and monitoring.

Local issues could be dealt with through the application of good control practice or better technological approaches based on local industry information. Geothermal sources of mercury have been investigated in the past (Brasell, 1982), and may require on-going investigation. It may be desirable to continue a reasonable level of testing of New Zealand fuels (especially coal), and bottom and fly ashes for mercury (and other metals) to assess whether there are any potential problems associated with fuel combustion. It would also be desirable to maintain a database of mercury levels in fish and drinking water, particularly in areas impacted by current or historical sources of mercury.

5.10 Chromium and chromium compounds

5.10.1 Health effects

Adverse health effects arising from exposure to chromium and its compounds have been well documented and summarised by the World Health Organization (1996), the US Environmental Protection Agency (1998a), and the California Air Resources Board (1998a). Chromium VI compounds are much more toxic than chromium II and III, and so chromium VI (Cr^{+6}) will be the focus of the following analysis.

The respiratory tract is the major target organic for acute inhalation exposure to chromium VI. Dyspnoea, coughing, and wheezing in humans have been reported following exposure to very high levels. Gastrointestinal and neurological effects have also been reported. Chronic inhalation exposure has been associated with effects on the respiratory tract; perforations and ulcerations of the septum, bronchitis, decreased pulmonary function, pneumonia, asthma, and nasal itching and soreness have also been reported in humans following exposure. Other effects of chronic inhalation exposure have been shown to include the liver, kidney, gastrointestinal and immune systems, and possibly the blood. The highest tissue levels in rats

following inhalation exposure occurred in the lung and kidney (US Environmental Protection Authority, 1998a).

Complications during pregnancy and childbirth in humans have been reported following inhalation exposure. Reproductive effects have not been reported in animal studies, but oral exposure has been reported to cause severe developmental effects in mice. Epidemiological studies of workers have established that inhaled chromium is a human carcinogen, resulting in increased risk of lung cancer, although the studies were not able to differentiate between chromium VI and chromium III compounds. Lung tumours have also been reported in animals following inhalation exposure to chromium VI (US Environmental Protection Agency, 1998a).

The US EPA has classified chromium VI as a Group A carcinogen of high potency, and chromium III as not classifiable (Group D). IARC has classified chromium VI as a Group 1 carcinogen, and chromium III as a Group 3 (unclassifiable) (International Agency for Research on Cancer, 1998).

5.10.2 Unit risk estimates

Unit risk factors for chromium VI compounds for inhalation exposure adopted by various groups are as follows:

1.2×10^{-3} per $\mu g/m^3$	US EPA, 1998a
1.5×10^{-1} per $\mu g/m^3$	CARB, 1998a
$1.1\text{-}13 \times 10^{-2}$ per $\mu g/m^3$	WHO, 1996.

5.10.3 Guidelines and standards

Since chromium VI is considered a human carcinogen, WHO has not specified a guideline for ambient air quality, but recommends that unit risk factors be applied.

The US EPA specifies an RfD for chromium VI of 5 µg/kg/day, and 1,000 µg/kg/day for chromium III (US Environmental Protection Agency, 1993a). RfCs for both groups of compounds are under review.

The CARB specifies a non-cancer chronic REL of 2×10^{-3} $\mu g/m^3$ for chromium VI, considering effects on the respiratory tract, kidney, and gastrointestinal system as the toxicological targets. An REL has not been established for chromium III.

In New Zealand, the recommended maximum ambient concentrations (RMAC), derived by NIWA (1997a), are as follows:

Soluble chromium VI compounds	0.50 $\mu g/m^3$
Insoluble chromium VI compounds	0.10 $\mu g/m^3$
Other chromium soluble compounds	5.0 $\mu g/m^3$.

The Victorian Environment Protection Authority 3-minute criteria are 1.8 $\mu g/m^3$ for chromic acid and chromates (as CrO_3), and 18 $\mu g/m^3$ for chromium, soluble chromic and chromous salts (as chromium).

5.10.4 Recommended evaluation criteria

Since chromium VI is acknowledged as a human carcinogen of high potency, a risk level of 1 in 100,000, which is at the lower end of the range considered acceptable by the US EPA, seems appropriate for New Zealand. Ambient air concentrations corresponding to an individual risk

of 1 in 100,000 of developing cancer for lifetime exposure to chromium VI based on the various unit risk factors are as follows:

8.7 ng/m^3	US EPA
0.073 ng/m^3	CARB
0.99 ng/m^3	WHO (high end, lower unit risk factor)
0.085 ng/m^3	WHO (low end, higher unit risk factor).

An annual average criterion for chromium VI (to be assessed by monitoring) of 1.1 ng/m^3 is at the top end of WHO values but lower than the US value, and therefore seems to be appropriate for New Zealand. For chromium metal, and chromium II and III compounds, concentration values 100 times larger than those for chromium VI seem appropriate on the basis of their much lower toxicity and non-carcinogenicity.

The ambient criteria values can be used as the basis for a 1-hour design value, and converting it by the ratio of the time scales, using the formula with the 0.2 power factor. The resultant 1-hour averages are 6.7 ng/m^3 for chromium VI and 670 ng/m^3 for the other forms.

The recommended criteria are 1.1 ng/m^3 (annual average) and 6.7 ng/m^3 (1-hour modelling criterion for chromium VI; and 110 ng/m^3 (annual average) and 670 ng/m^3 (1-hour modelling criterion) for chromium metal and chromium II and III.

As is the case for mercury, these values may need to be adjusted downwards if dietary intake is significant.

5.10.5 Implications for control strategies

There are no ambient air-quality data for New Zealand that could be used to assess the control implications of the proposed concentration values. New South Wales data indicates total chromium levels of 0.5 ± 0.4 ng/m^3. Data for California give total chromium levels of 3.9 ng/m^3 and chromium VI levels of 0.13 ng/m^3 (network averages). On this basis it is unlikely that regional-scale levels of chromium in New Zealand would require any control action, but this would need to be confirmed by emissions inventories and monitoring.

Most of the chromium in air is in the fine particle mass (California Air Resources Board, 1998a). Thus control requirements that reduce fine particles will also reduce emissions of chromium (and other metals). Local issues can be dealt with through the application of good control practice or better technological approaches based on local industry information. As is the case for mercury, it may also be desirable to continue with a reasonable level of testing for chromium (and other metals) in New Zealand fuels (especially coal), and bottom and fly ashes, to assess whether there are any potential problems associated with fuel combustion. A potential problem is the combustion of timber that has been treated with chromium salts (via a copper-chrome-arsenic formulation), but because the chromium is retained in the ash (unlike arsenic, where the oxides are volatile), it is only associated with emissions of fly ash. Levels in the environment that may be potential sources of dietary chromium should also be established.

5.11 Inorganic arsenic

5.11.1 Health effects

Adverse health effects arising from exposure to inorganic arsenic and its compounds have been well documented and summarised by the World Health Organization (1996), the US Environmental Protection Agency (1998a), and California Air Resources Board (1998a).

Acute inhalation exposure to inorganic arsenic may result in gastrointestinal effects, haemolysis, and central and peripheral nervous system disorders in humans. Effects of acute exposure to arsine (a gaseous compound of arsenic) include haemolytic anaemia, haemoglobinuria and jaundice, and can lead to kidney failure. Acute inhalation exposure to arsine can lead to death: it has been reported that exposure to 87 to 170 mg/m^3 arsine for half an hour can be lethal. Chronic inhalation exposure to, and contact with, inorganic arsenic is associated with irritation of the skin and mucous membranes including dermatitis, conjunctivitis, pharyngitis, and rhinitis. Several studies of women working or living near metal smelters, and in the electronics industry, have associated exposure to arsenic and arsine gas with increased incidence of spontaneous abortions and lower birth weights. However, the studies have limitations due to simultaneous exposure to other pollutants, and small numbers in some studies. Human inhalation studies have reported that inorganic arsenic exposure is strongly associated with lung cancer. Human exposure by ingestion has also been associated with an increased risk of skin, bladder, liver, and lung cancer (US Environmental Protection Agency, 1998a).

The US EPA has classified inorganic arsenic as a Group A carcinogen of high potency, but it has not classified arsine. The IARC has not classified either inorganic arsenic or arsine (International Agency for Research on Cancer, 1998).

5.11.2 Unit risk estimates

Unit risk factors for inorganic arsenic for inhalation exposure adopted by various groups are as follows:

4.3 x 10^{-3} per μg/m^3	US EPA, 1998
3.3 x 10^{-3} per μg/m^3	CARB, 1998a
1.5 x 10^{-3} per μg/m^3	WHO, 1996.

5.11.3 Guidelines and standards

Since inorganic arsenic is considered a human carcinogen, WHO has not specified a guideline for ambient air quality, but recommends that unit risk factors be applied.

The US EPA specifies an RfD of 0.3 μg/kg/day for inorganic arsenic, but it has not established a RfC. For arsine, the EPA has not established an RfD, but it does specify a non-cancer RfC of 0.055 μg/m^3 (US Environmental Protection Agency, 1993a).

The CARB specifies a non-cancer chronic REL of 0.55 μg/m^3 for inorganic arsenic, considering blood disorders as the toxicological endpoint, and a non-cancer chronic REL of 140 μg/m^3 for arsine, for which the toxicological endpoints are considered to be the respiratory system, the central and peripheral nervous systems, and the skin (California Air Resources Board, 1998a).

In a review of its draft acute (1-hour) non-cancer RELs for inorganic arsenic and arsine, the CARB has proposed the following draft values, which are to be reviewed by the Scientific Review Panel on Toxic Air Contaminants:

Inorganic arsenic	0.41 μg/m^3	basis: decreased foetal weight in mice
Arsine	170 μg/m^3	basis: haemolysis of red blood cells.

In New Zealand, the recommended maximum ambient concentrations (RMACs), derived by NIWA (1997a), are as follows:

Inorganic arsenic	0.10 μg/m^3
Arsine	1.7 μg/m^3.

The Victorian Environment Protection Authority has not specified 3-minute average criteria values for inorganic arsenic and arsine.

5.11.4 Recommended evaluation criteria

As for chromium VI, ambient criteria for inorganic arsenic can be developed based on an acceptable risk value of 1 in 100,000 for a high-potency carcinogen.

Ambient air concentrations corresponding to an individual risk of 1 in 100,000 of developing cancer for lifetime exposure to arsenic based on the various unit risk factors are as follows:

0.0025 μg/m^3	US EPA
0.0033 μg/m^3	CARB
0.0073 μg/m^3	WHO.

An annual average criterion value for inorganic arsenic, to be assessed by monitoring, of 0.0055 μg/m^3 therefore seems appropriate. For arsine, the US EPA's RfC of 0.055 μg/m^3 seems an appropriate annual average concentration.

The ambient criteria can be used as the basis for 1-hour average concentrations for assessing the results of dispersion modelling, by converting them using the ratio of time scales, using the formula with the 0.2 power factor. The 1-hour average criteria so derived are as follows:

Inorganic arsenic	0.033 μg/m^3
Arsine	0.33 μg/m^3.

As is the case for mercury and chromium, these values may need to be adjusted downwards if dietary intake is significant. Contaminated soils may be a significant source of exposure for children.

5.11.5 Implications for control strategies

There are no published ambient air-quality data for New Zealand that could be used to assess the control implications of the proposed criteria values. Data for California indicate arsenic levels of 0.0015 μg/m^3 (network averages). On this basis it is unlikely that regional-scale levels of arsenic in New Zealand would require any control action, but this would need to be confirmed by emissions inventories and monitoring.

Most of the arsenic in the air is in the fine particle mass (California Air Resources Board, 1998a). Thus control requirements that reduce fine particles will also reduce emissions of arsenic (and other metals). Local issues can be dealt with through the application of good control practice or better technological approaches based on local industry information.

Emission of arsine from old landfills, which may have been used to dispose of arsenic residues, may warrant investigation. As is the case for mercury and chromium, it may also be desirable to continue a reasonable level of testing for arsenic (and other metal levels) in New Zealand fuels (especially coal) to assess whether any potential problem from fuel combustion exists. A particular potential problem is the combustion of timber that has been treated with arsenic salts (via a copper-chrome-arsenic formulation). Arsenic oxides are volatile, and so are more likely to be discharged to the air than are copper and chromium compounds. Controlled studies have shown that up to 90% of the arsenic in treated timber is emitted to air on combustion, whereas nearly all of the copper and chromium is retained in the ash (Ministry for the Environment / Ministry of Health, 1997).

5.12 Lead

5.12.1 Health effects

The biological effects of lead are related to the levels of lead in human blood. Although there are some differences in the bio-availability of different lead compounds, the health effects caused by increased blood lead levels are the same, regardless of the lead compounds causing the exposure (USEPA, 1994).

As discussed previously, the major areas where humans may be exposed to lead in New Zealand are around point discharges and around houses or other structures where lead-based paint is being, or has been, removed without the proper safety precautions. Exposure for both adults and children can arise from inhaling fine lead particles in the air or by ingesting soils or crops contaminated by lead deposition. Contaminated soils and dusts act as a continuous source of lead. Therefore where ambient lead levels are compared to the guideline value for lead in air, regard should also be had for any possible exposure from ingestion of lead.

The WHO has carried out a considerable amount of research on the health effects of lead. One of the most widely recognised effects of lead exposure is a decrease in intelligence and general academic performance in children especially when exposed to lead within the first 2 to 3 years of life. The LOAEL for haematological and neurological effects in adults and children are given in table 5.5 below (extracted from WHO (1999) and from Streeton (1997)).

Table 5.5 Table of haematological and neurological effects in adults and children.

Effect	Blood lead level	
	Children	Adults
Exhibition of frank anemia	700µg/l	800µg/l
Reduced haemoglobin production	250-300µg/l	500µg/l
Elevated urinary delta-aminolaevulinic acid (ALA) and coproporyphyrin caused by inhibition of delta-aminolaevulinic acid dehydrase (ALAD) – an enzyme involved in heme biosynthesis, resulting in an accumulation of its substrate, ALA, in blood, plasma and urine (WHO 1987)	400µg/l	400µg/l

Increased erythrocyte protoporphyrin	200-300µg/l (male adults)	150-200µg/l (female adults and children)
Reduction in vitamin D3	-	100- 150µg/l
Inhibition of ALAD (but not considered to be an adverse effect by the WHO)	100µg/l	100µg/l
Encephalophatic signs and symptoms do not appear to occur at levels below	1000-1200µg/l	800-1000µg/l
Cognitive effects in lead workers have not been observed at blood lead levels below	500µg/m3	-
Reductions in nerve condition velocity were found at concentrations as low as	300µg/l	-
Central nervous system effects appear to occur in children at levels below	-	200µg/l
Effects on intelligence quotient	-	100-150µg/l
Impairment of hearing (found in some epidemiological studies)	-	100µg/l

The sub-groups most vulnerable to lead are young children and developing fetuses. There is now clear epidemiological evidence of a close causal relationship between prenatal exposure to lead and early mental development indices. This effect has been attributed to lead present at blood levels as low as 10 µg/dL, but the absence of an identifiable threshold suggests that a deleterious effect may be produced at blood lead levels lower than 10 µg/dL.

5.12.2 Unit risk estimates

The USEPA has made lead a B2 carcinogen, however, it has not established a unit risk factor for lead in air.

5.12.3 Guidelines and standards

The guidelines for lead are based on the effects of lead in blood.

The existing New Zealand ambient air quality guideline for lead is the range 0.5 – 1.0 µg/m^3, 3-month moving average, calculated monthly. The recommended new method of measurement involves the determination of the lead content of PM10 (a modification of the methods recommended in the 1994 guidelines which also used the lead content of total suspended particulate). It was based on the 1987 WHO guideline value at the time of 0.5 – 1µg/m3, annual mean. This guideline value incorporated a safety factor of two and assumes that 98% of the population will maintain a blood lead level of below 200µg/l.

However, with no apparent threshold concentration, it is prudent to have the ambient air quality guideline for lead as low as possible. Most jurisdictions are now establishing

guidelines/standards for lead in air so as to achieve a blood lead level of less than 10 µg/dL (1dL = 0.1L) or 100µg/l in all sub-groups in the population.

The WHO (1999) recommend an annual ambient air concentration guideline value of 0.5µg/m^3, annual average. They considered the critical effects to be elevation of free erythrocyte protoporphyrin for adults and cognitive deficits, hearing impairment and disturbed vitamin D metabolism for children. A critical level of lead in blood is 100 µg/l. These values were based on population studies that provided group averages. They apply to the individual child only in a probabilistic manner.

In deriving this guideline value the WHO assumed inhalation of airborne lead is a significant route of exposure for adults (including pregnant women), but it is less significant for young children for whom other pathways of exposure, such as ingestion, are more important. Also, they note that 1 µg/m^3 of lead in air approximately equates to 19µg Pb/l of blood in children and about 16 µg Pb/l in adults. They recommend that efforts should be taken to ensure that at least 98% of an exposed population, including pre-school children, should have blood lead levels that do not exceed 100µg/l of lead in blood. This proposal is based on the assumption that the upper limit of nonanthropogenic blood lead is 30µg/l.

Finally, WHO recommend that to prevent increases of lead in soils and the consequent increases in exposure of future generations the levels of lead in air should be kept as low as possible.

5.12.4 Recommended evaluation criteria

The lower of the current range, 0.5 µg/m^3, is appropriate at this time, but consideration should be given to lowering it even further to 0.2 µg/m^3. (A recent recommendation of the United Kingdom Expert Panel on Air Quality Standards is for 0.25 µg/m^3, annual average.) As with ambient concentrations of particles in New Zealand, there is seasonal variation in lead levels, with highest concentrations occurring during winter months. This is principally caused by poorer atmospheric dispersion provided by meteorological conditions in winter, especially the increased frequency of temperature inversions. This degree of seasonal variation means that the use of a three month (rather than a one year) averaging period for the ambient air quality guideline is appropriate; a one year average may not adequately show peak concentrations for what are still relatively long term exposure times.

The recommended monitoring criteria for the lead content of PM10 is 0.5 µg/m^3, 3-month moving average, calculated monthly, with sampling in accordance with the standard method specified in US 40 CFR Part 50, Appendix J, and analysis in accordance with the standard method specified in US 40 CFR Part 50, Appendix G, or an equivalent analytical method. It is also recommended that consideration be given to reducing the concentration to 0.2 µg/m^3.

It must be stressed that where there is the likelihood of ingestion from deposited lead, this must be taken into account in conjunction with inhalation exposure, when considering the total body burden, especially for any children living in the area, and when assessing potential health effects of a discharge containing lead.

5.12.5 Implications for control strategies

With the removal of lead from petrol the levels of lead in the ambient air are below the recommend guideline value, therefore there are no major implications for fuel specifications. However, there may be implications for some point source discharges.

6 Monitoring methods

There are a variety of different methods currently being used for the determination of hazardous air contaminants in ambient air, although most of these have yet to be adopted as formal standard monitoring methods. In fact, the only published standard method for any of the hazardous air contaminants, is a draft ISO document (DIS12884) for the determination of gas- and particle-phase polycyclic aromatic hydrocarbons.

No standard methods have yet been set by the US EPA, despite the comprehensive efforts being made towards control of hazardous air contaminants in that country. However, the USEPA has published a Compendium of recommended methods for organic pollutants* downloadable from These methods can be downloaded from the USEPA website at http://www.epa.gov/ttn/amtic/airtox.html. This document contains 17 peer reviewed, standardised methods for the determination of volatile, semi-volatile, and selected toxic organic pollutants in the air. The methods have no official regulatory status, but can be taken as representing the current "best practice" for the determination of organic hazardous air pollutants in ambient air. The relevant methods from this report have been used in developing the recommendations given below.

6.1 Volatile hydrocarbons

Examples of volatile hydrocarbons are benzene, toluene, xylene and 1,3-butadiene. The following US EPA methods are suitable for determining these pollutants:

- Method TO-1. Tenax absorption followed by thermal desorption and gas chromatography/mass spectrometry analysis.
- Method TO-2. Absorption on carbon molecular sieves followed by thermal desorption and gas chromatography/mass spectrometry analysis.
- Method TO-3. Cryogenic preconcentration followed by gas chromatography with flame ionisation and electron capture detection.
- Method TO-14A. Collection in specially-prepared canisters followed by cryogenic pre-concentration and analysis by gas chromatography.
- Method TO-15A. Collection in specially-prepared canisters followed by solid absorbent pre-concentration and gas chromatography/mass spectrometry analysis.
- Method TO-17. Multi-bed absorbent followed by thermal desorption and gas chromatography/mass spectrometry analysis. (This method is intended for use with automated analysers.)

All of the above methods are suitable for the determination of a range of volatile hydrocarbons in air. They differ mainly in the degree of complexity of both the sampling equipment and the analytical procedures. These generally increase as one moves down the list. The range of

* Methods for the Determination of Toxic Organic Compounds in Air – Second Edition. US EPA, Office of Research and Development, Cincinnati. Report No. EPA/625/R-96/010b, January 1999.

volatile hydrocarbons able to be covered by the different methods also increases down the list. However, none of these refinements is necessary for benzene toluene, xylene or 1,3-butadiene, which can all be measured quite adequately using method TO1. This is therefore the recommended method, but with provision for the use of other equivalent systems as appropriate.

6.1.1 Recommended method

The recommended method for benzene, toluene, xylene and 1,3-butadiene is US EPA method TO-1, or an equivalent procedure.

6.2 Carbonyls

The following US EPA methods are suitable for formaldehyde and acetaldehyde, the two most common carbonyls:

Method TO-5. Absorption in a solution of dinitrophenylhydrazine followed by analysis using high performance liquid chromatography with UV detection.

Method TO-11A. Sampling through packed tubes coated with dinitrophenylhydrazine followed by followed by analysis using high performance liquid chromatography with UV detection.

These two methods are essentially the same, but the second one has some operational advantages because of the use of packed tubes. These are a lot easier to handle and present fewer transportation difficulties than the bubbler solutions required for method TO-5. Both methods are in regular use in New Zealand, although TO-11A is generally the preferred approach.

6.2.1 Recommended method

The recommended method for formaldehyde and acetaldehyde is US EPA method TO-11A.

6.3 Polycyclic aromatic hydrocarbons

There is only one method recommended for PAHs, of which benzo(a)pyrene is the usual indicator, US EPA method TO-13A. This involves sampling through a glass fibre filter and a polyurethane foam or XAD-2 absorbent cartridge, using a modified high volume air sampler. The samples are extracted with solvent and then analysed using GC/MS. The method is much the same as that covered in the proposed ISO standard, DIS12884. Both methods allow for separate determination of gaseous and particulate fractions, if required. The EPA method has been used in New Zealand for the measurement of other semi-volatile organics such as PCBs and dioxins.

6.3.1 Recommended method

The recommended method for benzo(a)pyrene is US EPA method TO-13A.

6.4 Lead

Airborne lead is normally determined by laboratory analysis of the filters collected during sampling for inhalable or suspended particulate matter using a gravimetric method. Each of these will give a measure of "lead". However, one of the questions that needs to be addressed is which size fraction should be used for this determination?

The air quality guideline for lead is set on the basis of protection against lead exposure by inhalation. By definition, this means only the lead that is present in particles fine enough to be inhaled – i.e., inhalable particles, or PM10. It is therefore recommended that PM10 monitoring should be the method used for the determination of airborne lead. This should also provide practical advantages given that PM10 is already commonly measured throughout the country.

The Australian standard procedure for lead is given in AS2800-1985. However, this allows for monitoring of total suspended particulate using the high volume sampler. Similarly, the US EPA reference method is based on the analysis of filters from high volume sampling (40 CFR Part 50, Appendix G). However, both of these methods are equally as applicable to filters collected with a PM_{10} monitor. A similar procedure is also specified in ISO 9855:1993, but this method has been written without qualification as to the type of equipment used for sampling.

The analytical procedures specified in the above methods are all based on acid extraction of the lead followed by analysis using flame atomic absorption spectroscopy. This is a well-established laboratory method and is well suited to the task. However, the method has been superseded in many laboratories by other analytical instruments which are sometimes more sensitive and nearly always more cost effective than atomic absorption spectroscopy. These methods include flameless atomic absorption spectroscopy, inductively coupled argon plasma-optical emission spectrometry, inductively coupled argon plasma-mass spectrometry, and x-ray fluorescence spectrometry. All of these analytical procedures are suitable for lead analysis, and have been recognised as such by the US EPA (eg. Federal Register, v45, p14648, 6 March 1980, v54, p20193, 10 May 1989, and v61, p11404, 20 March 1996).

6.4.1 Recommended method

The recommended method for inhalable lead is PM_{10} sampling, in accordance with 40 CFR Part 50, Appendix J and lead analysis in accordance with 40 CFR Part 50, Appendix G, or an equivalent analytical method.

6.5 Mercury, chromium and arsenic

Most metals can be measured by the filter collection method described for lead. This method is effective for other toxic metals, such as mercury, chromium and arsenic, when these are present as components of suspended or inhalable particulate. However, this method will be ineffective for

mercury in its vapour (elemental) form. Arsenic can also be a problem if present as the moderately volatile trioxide, although this is not a common component of environmental samples.

Mercury vapour can be measured using commercially available mercury monitors, which use preconcentration by absorption onto a gold film and desorption into a gas cell. The mercury concentration is measured by absorption of UV light. A number of laboratory-based variations on this method are also available. However, neither these nor the portable monitors have been subjected to any detailed performance studies.

6.5.1 Recommended methods

The recommended method for mercury, chromium and arsenic, as particulates, is: PM_{10} sampling, in accordance with 40 CFR Part 50, Appendix J, followed by analysis using atomic absorption spectroscopy or an equivalent method. It is not possible to recommend a method for mercury vapour at this time.

7 Recommendations

For the each of the hazardous air contaminants assessed in this review, evaluation criteria are given. These are of two types: annual averages for assessing the results of ambient air monitoring at residential sites, and 1-hour average concentrations against which the results of dispersion modelling can be assessed. These criteria are given in Table 6.1.

Table 6.1: Recommended evaluation criteria for hazardous air contaminants

Hazardous air contaminant		**µg/m^3**	
		Monitoring criteria (annual average)	**Modelling criteria (1-hour average)**
Benzene		Year 2000 10 Year 2010 3.6	22
Toluene		190	500
Xylene		950	1,000
1,3-Butadiene		2.4	15
Formaldehyde		15	20
Acetaldehyde		30	45
Benzo(a)pyrene		0.0003	n.a.
Mercury	inorganic organic	0.33 0.13	2.0 0.80
Chromium	chromium VI other forms	0.0011 0.11	0.0067 0.67
Arsenic	inorganic Arsine	0.0055 0.055	0.033 0.33
Lead		Year 2001 0.5 Future 0.2	

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Annex A: Glossary of terms used

Acute exposure	Exposure of short duration, usually in the order of hours or minutes.
Acrodynia	A rare syndrome found in children exposed to elemental mercury or inorganic mercury compounds, characterised by severe leg cramps, irritability, prickling sensation on the skin, painful fingers, and peeling hands, feet and nose.
Adverse effect	Any biochemical change, functional impairment, or pathological lesion which impairs and reduces the ability of an organism to respond to additional change.
Ataxia	Muscular incoordination.
BACT	Acronym for "Best Available Control Technology".
Carcinogenic agent	Any substance that incites or produces cancer.
Critical effect	The first adverse effect, or its known precursor, that occurs as the dose rate increases.
Chronic exposure	Exposure which occurs over a long time, usually one or more years.
Cytoxic agent	Any substance that destroys cells or prevents cell multiplication.
Dose	The amount of a substance available for interaction with metabolic processes or biologically significant receptors after crossing the outer boundaries of an organism. The potential dose is the amount inhaled, ingested, or absorbed through the skin.
Dose response curve	A graphical representation of the quantitative relationship between the dose and a specific biological response.
Dose rate	The dose received per unit time, e.g. mg/day.
Dysmorphism	Abnormality in form.
Dyspnoea	Air hunger resulting in laboured or difficult breathing.
Erethism	Abnormal mental excitability or sensitivity to sensory stimulation.
Exposure concentration	The concentration of a substance in its transport or carrier medium to the point of contact. Thus the potential dose for an airborne substance is the concentration in air multiplied by the amount of air breathed.
Exposure route	The way a chemical or pollutant enters an organism after contact, e.g., by ingestion, inhalation, or dermal exposure.
Genotoxic agent	Any substance that interferes with genes or gene structure.
Haematopoietic system	The blood-making organs, particularly the bone marrow and lymph nodes.
Haemolysis	Destruction of the red blood cells with the liberation of haemoglobin into the surrounding fluid.

Haemoglobinurea	The presence of haemoglobin in the urine, but free from red blood cells.
Hyperproliferation	Excessive rate of increase.
LOAEL	Acronym for "lowest observed adverse effect level": the lowest exposure level at which there are statistically or biologically significant increases in the frequency or severity of adverse effects between the exposed population and its appropriate control group.
LOEL	Acronym for "lowest observed effect level": the lowest exposure level at which there are statistically or biologically significant increases in the frequency or severity of an effect between the exposed population and its appropriate control group.
Lymphatic system	All structures involved in conveying lymph from the tissue to the bloodstream.
Mutagen	Any substance that causes genetic mutations.
Necrosis	Death of areas of tissue or bone.
NOAEL	Acronym for "no observed adverse effect level": the exposure level at which there are no statistically or biologically significant increases in the frequency or severity of adverse effects between the exposed population and its appropriate control group. Some effects may be produced at this level but they are not considered as adverse nor precursors to the adverse effects.
NOEL	Acronym for "no observed effect level": the exposure level at which there are no statistically or biologically significant increases in the frequency or severity of any effect between the exposed population and its appropriate control group.
Pneumonosis	Any non-infective disease or disorder of the lung.
Pulmonary oedema	Escape of fluid into the air vesicles and into the interstitial tissue of the lungs; build-up of fluid in the lungs.
REL	Short for "reference exposure level".
RfC	Short for "reference concentration": an estimate (with uncertainty spanning perhaps an order of magnitude) of continuous inhalation exposure to the human population (including sensitive sub-groups) that is likely to be without appreciable risk of deleterious effects during a lifetime.
RfD	Short for "reference dose": an estimate (with uncertainty spanning perhaps an order of magnitude) of continuous exposure to the human population (including sensitive sub-groups) that is likely to be without appreciable risk of deleterious effects during a lifetime).
Risk	The probability of injury, disease or death under specific circumstances. In quantitative terms, risk is expressed in values ranging from zero (representing absolute certainty that harm will not occur) to one (representing absolute certainty that harm will occur).

Target organ	The organ or system of the body that is generally affected first as the dose of a substance is increased from zero.
Threshold	The dose or exposure below which an adverse effect is not expected.
TLV	Short for "Threshold limit value": the concentration of a substance to which most workers can be exposed without adverse effects.
TWA	Short for "the time weighted average": an allowable exposure concentration averaged over a normal 8-hour workday or a 40-hour work week.
Uncertainty factor	Factors representing specific areas of uncertainty inherent in the available data.
Unit risk	The upper-bound excess lifetime cancer risk estimated to result from continuous exposure to an agent at a concentration of 1 µg/L in water, or 1 µg/m3 in air.

Annex B: Priorities for hazardous air contaminants

Compound	US EPA		NPI Trials[2]		NPI Technical Advisory Panel		ANZECC Review		NIWA (N)	
	HAPs list[1]	High priority	Included in trials	Emission estimate (tonnes/A)	Risk[3]	Rank[3]	Risk	Staff rating	Bingham (B)[5]	
Acetaldehyde	Y	Y	Y		3.5	58		2.4	B	N
Acetamide	Y									
Acetone					5.5	22				
Acetonitrile	Y				3.6	56				
Acetophenone	Y									
2-Acetylaminofluorene	Y									
Acrolein	Y	Y						1.2	B	
Acrylamide	Y				2.8	80				
Acrylic acid	Y				3.8	49				
Acrylonitrile	Y	Y	Y		3.3	62		8.8	N	
Allyl chloride	Y				0	207				
4-Aminodiphenyl	Y									
Ammonia	E				4.0	45		5.3		
Aniline	Y				3.1	69				
o-Anisidine	Y									
Antimony – elemental	Y									
Antimony – elemental and compounds	Y				2.6	84				
Arsenic – elemental and soluble compounds	Y		Y		2.0	9	8	0.6	B	N
Arsine	Y				0.8	173				
Asbestos	Y				2.3	97		4.1		
Benzene	Y	Y	Y	300	6.7	14	9	20	B	N
Benzidine	Y				1	164				
Benzoyl chloride	E				2.1	102				

Compound	US EPA		NPI Trials[2]		NPI Technical Advisory Panel		ANZECC Review		NIWA (N)	
	HAPs list[1]	High priority	Included in trials	Emission estimate (tonnes/A)	Risk[3]	Rank[3]	Risk	Staff rating	Bingham (B)[5]	
Benzyl chloride	Y									
Benzyl trichloride	Y									
Berilium – elemental and compounds	Y				2.9	75			B	
Biphenyl	Y				3.7	53				
Bis(2-ethylhexyl)phthalate (DEHP)	Y	Y			3.1	66				
Bis(chloromethyl)ether	Y									
Bromoform	Y				1.9	116				
1,3-Butadiene	Y	Y	Y	50	6.7	12	8	12.4	B	N
Butyl acrylate	E									
n-Butyl alcohol	E									
Cadmium – elemental and compounds	Y	Y	Y	0.6	7.6	6	11	3.5	B	N
Calcium cyanamide	Y									
Caprolactum – dust	Y									
Caprolactum – vapour	Y									
Captan	Y									
Carbaryl	Y									
Carbon disulphide	Y				3.6	56				
Carbon tetrachloride	Y	Y			0	207		1.2	B	
Carbonyl sulphide	Y									
Catechol	Y									
Chloramben	Y									
Chlordane	Y				0	207		0		
Chlorinated camphene (toxaphene)	Y									
Chlorine	Y				4.0	42		3.5		

Compound	US EPA		NPI Trials[2]		NPI Technical Advisory Panel		ANZECC Review		NIWA (N)	
	HAPs list[1]	High priority	Included in trials	Emission estimate (tonnes/A)	Risk[3]	Rank[3]	Risk	Staff rating	Bingham (B)[5]	
Chlorine dioxide	E				4.2	40				
Chloroacetic acid	Y				0	207				
2-Chloroacetophenone	Y									
Chlorobenzene	Y				0	207		0.6		
Chlorobenzilate	Y									
Chloroform	Y	Y			3.1	66		0.6	B	
Chloromethyl methyl ether	Y				1	164				
β-Chloroprene (2-chloro 1,3-butadiene)	Y									
Chromium – (VI) insoluble compounds	Y	Y	Y		9.6	2			B	N
Chromium – (VI) soluble compounds	Y	Y	Y		9.6	2			B	N
Chromium – elemental and (II), (III) compounds	Y	Y	Y	0.3	3.5	60	8	2.4	B	N
Cobalt – elemental and inorganic compounds	Y				5.0	27				
Cobalt – organic compounds	Y				5.0	27				
Copper – elemental and compounds (dusts/mists)	E									
Copper – elemental and compounds (fume)	E				4.2	40		0.6		
Cresol (cresylic acid)	Y				4.2	40				
m-Cresol	Y									
o-Cresol	Y									
p-Cresol	Y									
Cumene	Y				3.1	70				
Cumene hydroperoxide										
Cyanide – salts	Y				5.0	70		2.4		

Compound	US EPA		NPI Trials[2]		NPI Technical Advisory Panel		ANZECC Review		NIWA (N)
	HAPs list[1]	High priority	Included in trials	Emission estimate (tonnes/A)	Risk[3]	Rank[3]	Risk	Staff rating	Bingham (B)[5]
Cyanogen									
2,4-D – salts and esters	Y								
DDE (1,1-dichloro-2,2-bis) p-chlorophenyl)ethylene)	Y								
Diazomethane	Y								
Dibenzofurans	Y		Y		2.6	83		0.6	
1,2-Dibromo-3-chloropropane	Y				0	207			
1,2-Dibromoethane (ethylene dibromide)	Y	Y			4.5	36			
Dibutyl phthalate	Y				3.1	66			
p-Dichlorobenzene	Y	Y	Y		0.7	172	3	2.9	
3,3-Dichlorobenzidine	Y				1.1	167			
1,1-Dichloroethane	Y	Y			0	207			
1,2-Dichloroethane (ethylene dichloride)	Y				2.8	77		1.8	N
Dichloroethylether									
1,1-Dichloroethylene (vinylidene chloride)	Y				2.0	106			
1,2-Dichloropropane (propylene dichloride)	Y	Y							
1,3-Dichloropropene	Y	Y			0	207			
Dichlorvos	Y								
Diethanolamine	Y								
Diethyl phthalate									
Diethyl sulphate	Y								
3,3-Dimethoxybenzidine	Y								
Dimethyl carbamoyl chloride	Y								
Dimethyl formamide	Y								
1,1-Dimethyl hydrazine	Y								

Compound	US EPA		NPI Trials[2]		NPI Technical Advisory Panel		ANZECC Review		NIWA (N)
	HAPs list[1]	High priority	Included in trials	Emission estimate (tonnes/A)	Risk[3]	Rank[3]	Risk	Staff rating	Bingham (B)[5]
Dimethyl sulphate	Y								
4-Dimethylaminoazobenzene	Y								
N,N-Dimethylaniline	Y								
Dimethylnitrosoamine (N-nitrosodimethylamine)	Y				0	212			B
Dimethylphthalate	Y								
Dinitro-o-cresol	Y								
2,4-Dinitrophenol	Y								
2,4-Dinitrotoluene	Y				1.7	126			
1,4-Dioxane (1,4-dithyleneoxide)	Y				2.0	113			
1,2-Diphenylhydrazine	Y				1.8	118			
Epichlorohydrin	Y				2.1	102			
1,2-Epoxybutane	Y								
1,2-Epoxypropane (propylene oxide)	Y				2.0	106			
2-Ethoxyethanol					6.0	19			
2-Ethoxyethyl acetate					6.0	19			
Ethyl acrylate	Y				1,8	123		5.3	B
Ethyl benzene	Y				2.5	86		2.4	
Ethyl carbamate (urethane)	Y								
Ethyl chloride	Y				3.0	71			
Ethylene glycol	Y				5.5	23			
Ethylene oxide	Y	Y			4	48		1.8	B
Ethylene thiourea	Y								B
Ethylenimine	Y				0	207			
Fine mineral fibres (synthetic)	Y								
Formaldehyde	Y	Y	Y	100	3.6	55	5	12.4	B N

Compound	US EPA		NPI Trials[2]		NPI Technical Advisory Panel		ANZECC Review		NIWA (N)	
	HAPs list[1]	High priority	Included in trials	Emission estimate (tonnes/A)	Risk[3]	Rank[3]	Risk	Staff rating	Bingham (B)[5]	
Heptachlor	Y				2.3	97		0		
Hexachloro1,3-butadiene	Y				1.7	128				
Hexachlorobenzene	Y				0	207		0	B	
Hexachlorocyclopentadiene	Y				0	207				
Hexachloroethane	Y				1.5	145				
Hexamethyl phosphoramide	Y									
Hexamethylene-1,6-diisocyanate	Y									
Hexane (n-hexane)	Y				2.5	86		5.3		
Hydrazine	Y	Y								
Hydrogen chloride	Y				4.3	39		2.9		
Hydrogen cyanide					2.4	91				
Hydroquinone	Y									
Isophorone	Y				1.3	156				
Isopropyl alcohol	E									
Lead – elemental and compounds	Y	Y	Y	40	6.9	11		4.9	B	
Lindane	Y				0	207				
Maleic anhdride	Y				1.8	123				
Manganese – elemental and inorganic compounds	Y	Y	Y	5	3.8	47	3			
Mercury – alkyl compounds	Y	Y	Y		4.6	35	*	*	B	N
Mercury – aryl compounds	Y	Y	Y		4.6	35	7	4.7	B	N
Mercury – elemental and inorganic compounds	Y	Y	Y		4.6	35	*	*	B	N
Methanol	Y				5.3	24		0.6		
Methoxychlor	Y									
Methyl acrylate					1.4	149				

Compound	US EPA		NPI Trials[2]		NPI Technical Advisory Panel		ANZECC Review		NIWA (N)
	HAPs list[1]	High priority	Included in trials	Emission estimate (tonnes/A)	Risk[3]	Rank[3]	Risk	Staff rating	Bingham (B)[5]
Methyl bromide	Y				2.0	106			N
Methyl chloride	Y	Y			1.6	134			
Methyl chloroform	Y								
Methyl ethyl ketone (2-butanone)	Y		Y	50	6.0	21	0	10	
Methyl hydrazine	Y								
Methyl iodide	Y								
Methyl isobutyl ketone (hexanone)	Y		Y	150	5.0	27	0	9.4	
Methyl isocyanate	Y								
Methyl methacrylate	Y				5.3	26	7.1		
Methyl-tert butyl ether	Y				2.3	96			
4,4-Methylene dianiline									
4,4-Methylene bis (2-chloraniline)	Y				4.0	44			
Methylene chloride	Y	Y	Y	200	7.8	5	5	3.5	N
4,4-Methylenediphenyl diisocyanate (MDI)	Y	Y			3,5	58		8.8	
Naphthalene	Y								
Nickel – elemental & compounds	Y	Y	Y	0.1	3.6	54	8	2.9	N
Nitric acid	E				4.0	42			
Nitrobenzene	Y				0.7	180			
4-Nitrodiphenyl	Y								
2-Nitropropane	Y								
N-Nitroso-N-methylurea	Y								
N-Nitrosomorpholine	Y								
PAHs as benzo(a)pyrene	Y	Y	Y	24	6.9	18	8	2.9	B N
Parathion	Y								

Compound	US EPA		NPI Trials[2]		NPI Technical Advisory Panel		ANZECC Review		NIWA (N)
	HAPs list[1]	High priority	Included in trials	Emission estimate (tonnes/A)	Risk[3]	Rank[3]	Risk	Staff rating	Bingham (B)[5]
Pentachloronitrobenzene (quintobenzene)	Y								
Pentachlorophenol	Y								
Perchloroethylene	Y	Y	Y	25	6.4	16	3	2.4	N
Peroxyacetic acid	E								
Phenol	Y				3.8	49		7.6	
p-Phenylene diamine	Y				0.7	181			
o-Phenylphenol	E								
Phospgene (carbonyl chloride)	Y				2.3	94			
Phosphine	Y		Y	0.6					
Phosphoric acid	E				4.3	38			
Phosphorus (yellow)	Y								
Phthalic anhydride	Y				1.6	132		5.3	
Polychlorinated biphenyls (PCBs, aroclors)	Y								B
Propane sultone	Y								
β-Propiolactone	Y								
Proprionaldehyde	Y								
Propoxur (Baygon)	Y								
1,2-Propylene imine	Y								
Quinoline	Y	Y							
Quinone (p-benzoquinone)	Y								
Radionucleides (including radon)	Y								
Selenium – elemental and compounds	Y				2.5	85			
Styrene	Y		Y	55	3.8	49	5	9.4	N
Styrene oxide	Y								

Compound	US EPA		NPI Trials[2]		NPI Technical Advisory Panel		ANZECC Review		NIWA (N)
	HAPs list[1]	High priority	Included in trials	Emission estimate (tonnes/A)	Risk[3]	Rank[3]	Risk	Staff rating	Bingham (B)[5]
Sulphuric acid	E				7.3	8			
2,3,7,8-Tetrachlorodibenzo-p-dioxin	Y	Y	Y		2.6	83	7	5.9	B N
1,1,2,2-Tetrachloroethane	Y				2.5	86		2.9	
Thiourea (isothiourea)	E				1.7	126			
Titanium tetrachloride	Y				1.0	164			
o-Tolidine (3,3-dimethyl benzidine)	Y								
Toluene	Y		Y	800	4.7	33	1	8.8	N
2,4-Toluene diisocyanate (TDI)	Y		Y	<0.1	4.7	34	2	13.5	
2,4-Toluene diamine	Y								B
o-Toluidine	Y				1.9	84			
1,2,4-Trichlorobenzene	Y				0.9	172			
1,1,2-Trichloroethane	Y				3.5	61		2.9	
Trichloroethylene	Y	Y			6.7	12	2		N
2,4,5-Trichlorophenol	Y				0.6	194			
2,4,6-Trichlorophenol	Y								
Triethylamine	Y				2.4	90			
Trifluralin	Y								
2,2,4-Trimethylpentane	Y								
Valbazen (albendazole)	Y								
Vinyl acetate	Y								
Vinyl bromide	Y								
Vinyl chloride	Y	Y	Y		3.2	65	1	6.5	B N
m-Xylene	Y				7.0	9			N
Xylenes –mixed isomers	Y		Y	800	7.0	9	1	11.8	N
o-Xylene	Y				7.0	9			N

Compound	US EPA		NPI Trials[2]		NPI Technical Advisory Panel		ANZECC Review		NIWA (N)
	HAPs list[1]	High priority	Included in trials	Emission estimate (tonnes/A)	Risk[3]	Rank[3]	Risk	Staff rating	Bingham (B)[5]
p-Xylene	Y				7.0	9			N
Zinc – elemental	E				3.8	48		*	
Zinc chloride fume	E				3.8	48		4.1	
Zinc oxide fume	E				3.8	48		*	
Hydrogen fluoride	Y				5.0 (fluorides)	27			B
Hydrogen sulphide					4.4	37			N
2-Methoxy ethanol (methyl cellosolve)					2.9	73			B

Notes to Table C1:

1. "HAPs list": this is taken from the 1990 amendments to the US Clean Air Act and (with the exception of caprolactum, which was subsequently removed) they are the hazardous air pollutants (air toxics) established by the US EPA. The "high priority" hazardous air pollutants are the US EPA urban air toxics (US Environmental Protection Agency, 1998a).
2. "NPI trials": this column lists pollutants selected for the trials conducted by the Victorian Environment Protection Authority (EPA) on the basis of professional judgement. The emission rates quoted are a combination of estimates reported by industry and estimates by the Victorian EPA.
3. The pollutant "rank" and "risk" estimates are taken from the database maintained by Environment Australia, which reflects subsequent amendments the Technical Advisory Panel made to remove duplications, but differences are minor.
4. The formal ranking system was applied to a limited number of substances, based on priorities for urban monitoring. A wider but still limited range of substances were selected and ranked on the basis of professional judgement from the point of view of potential for emission.
5. The different list of substances ranked by the two groups reflects, in part, different perspectives.

NB. Unless otherwise indicated, priorities for different substances within the one chemical group are treated as identical for ranking purposes.

Annex C: Approaches to setting guidelines for hazardous air contaminants and emission control technology

Introduction

Several different approaches are employed by different countries for controlling the impacts of hazardous air contaminants: a technology-based standards approach, characterised by the use of best available control technology; a health-based standards approach, employing health risk assessments; or a combination of the two. These approaches can be illustrated by reviewing recent developments in the United States nationally, and in the state of California.

Technology-based approach

In the US, the 1990 amendments to the Clean Air Act identify 189 (later amended to 188) priority air toxics (that is, hazardous air contaminants), and require the Environmental Protection Agency to develop emission standards based on available technology, irrespective of the degree of risk, for large stationary sources of these substances. Stationary sources are those emitting more than 10 tons per year of any individual air toxics, or more than 25 tons per year of a combination of air toxics. The performance-based standards are known as maximum achievable control technology (MACT), which is taken to mean the emission standards being achieved by the best performing sources in each source category.

The Clean Air Act amendments also require the US EPA to develop a strategy to address area sources of air toxics, and mobile sources. For area sources, emission standards can be MACT where warranted on the basis of community risk, or GACT (generally achievable control technology) otherwise. The Clean Air Act amendments require performance-based emission standards to be developed and implemented in accordance with a 10-year schedule. For area sources, the EPA is also required to regulate source categories accounting for 90% of emissions of the high-priority pollutants, and to achieve a 75% reduction in cancer risk.

Emissions post-MACT implementation are to be assessed to determine whether further action is required on the basis of residual risk to human health and the environment. The US EPA is required to develop a methodology for assessing residual risks, the public health significance of these risks, and technical and economic issues associated with controlling the risks.

The US EPA has established benchmark concentrations with which to compare outdoor ambient concentrations derived from modelling the 1990 air toxics emissions inventory. The benchmark concentrations for carcinogens were set to provide upper-bound lifetime excess cancer risks of 1 in 1 million. For non-carcinogens, the benchmark concentrations were set so that lifetime health impacts were not significant.

Based on data from the National Toxics Inventory for 1993, the EPA estimates that of the 40 air toxics initially identified, 23% come from point (major) sources, 40% from area sources, and 37% from mobile sources. The draft strategy for urban air toxics lists action taken to meet the requirements of the Clean Air Act. As of 1998, 175 source categories of air toxics are listed for regulation (167 for major sources, and 8 for area sources with some categories including both), and MACT or GACT standards have been promulgated for 47 of these categories.

For motor vehicles, existing exhaust and evaporative emission standards as well as fuel standards provide some degree of control of air toxics. Because of the importance of mobile sources, the EPA is currently considering further regulating mobile sources and fuels for specific air toxics. The aim is to achieve the greatest degree of reductions, considering factors such as availability and cost, and will at least include control of benzene and formaldehyde emissions, predicted ambient concentrations of which exceed benchmark concentrations by factors of more than 100 in some locations.

Clearly, the control technology based approach does not guarantee that emissions remaining after the application of controls will result in uniformly acceptable air quality, and other strategies need to be implemented to address local problems. Further possible action involves working with the states and local authorities to reduce residual risks, and may include tighter controls and national air-quality standards for specific hazardous air contaminants.

Relevance to New Zealand

In New Zealand, with the possible exception of specific hazardous air contaminants in a few places, area and mobile sources of hazardous air contaminants may be relatively more important for urban areas than major industrial sources. This is because the industrial base and the industry scale are smaller than in the US. In addition, although urban areas are also correspondingly much smaller, the presence of congested traffic conditions and lack of emission standards so far, will result in higher emissions per unit of distance travelled.

Favourable meteorology may lessen the impact of higher unit emissions on ambient air levels, but there are few data to quantify the overall impact. Air-quality data for Christchurch indicate relatively high levels of particles from solid-fuel combustion, which is indicative of relatively high levels of associated air toxics. Given the above, technological approaches for controlling air toxics would probably not be effective in reducing air-quality impacts in New Zealand urban environments, unless they include controls on mobile sources and domestic solid-fuel combustion.

Health-based approach

In addition to complying with Federal MACT and GACT regulations, California has a number of other approaches for limiting the ambient air-quality impacts of hazardous air contaminants (actually referred to as air toxics). These include tight vehicle-emission standards, which go beyond federal requirements, and volatile organic compounds (VOCs) controls on various sources (including area sources), which, while primarily aimed at reducing photochemical smog, have the added benefit of reducing emissions of hazardous air contaminants.

For stationary sources, the California Air Resources Board operates a "hot spots" programme, the details of which are specified in the Air Toxics "Hot Spots" Information and Assessment Act, and its various amendments. This legislation requires stationary source facilities emitting specified air toxics to compile emission inventories of those air toxics. The California Air Resources Board is also required to compile emission inventory data for mobile, natural, and area sources. Both major sources (defined in terms of the emission rate of criteria pollutants and total organic gases) and smaller sources are included, and priorities developed for assessing whether risk assessments are required, and subsequently whether risk-reduction plans are developed and implemented. All risk assessments are to be publicly available to the community. Inventories and risk assessments follow guidelines provided.

The approach to risk assessment follows the classical paradigm, which can be summarised as follows:

- hazard identification to determine the potential for adverse effects from a substance

- dose-response evaluation for the relevant health (or environmental) endpoints
- exposure assessment to determine the extent of exposure
- risk characterisation to determine the probability of an adverse outcome.

Two approaches are followed in the dose-response evaluation: a threshold approach, and a non-threshold approach. In the threshold approach, it is assumed that a level exists below which no adverse effects are likely to occur (NOAEL), and above which the effect is related to dose. The aim of the dose-response evaluation is to identify the NOAEL for each health endpoint being considered.

Because data to derive a NOAEL are generally limited for most chemicals, the US EPA derives it by using available data in combination with uncertainty factors. The derived value is known as the Reference Dose (RfD), and this can be converted to an ambient air concentration (RfC) where inhalation is the exposure route. The RfDs and RfCs are levels for which lifetime (or chronic) exposures will not result in appreciable risk of adverse effect.

The uncertainty factors used for chronic exposure to chemicals are given in Table C.1.

Table C.1: Uncertainty factors used for chronic exposure to chemicals

Area of uncertainty	Uncertainty factor
Extrapolation from LOAEL to NOAEL	10
Extrapolation from short-term exposure studies to chronic effects	10
Extrapolating animal data to human (interspecies variability)	10 (or 3)
Variability in sensitivity in the population (intraspecies variability)	10
Limitations in the database	10
Non-specific areas of uncertainty (modifying factor)	> 0 – 10

The combined uncertainty factor is the product of all the uncertainty factors, and can range from 10^6 (maximum uncertainty) to 1, where NOEL is derived from multiple high-quality human studies which include the most sensitive subjects.

The RfCs are considered to provide upper bound estimates of ambient concentrations that will be protective of the population (including sensitive subgroups, for the health endpoint being considered) and are therefore conservative. Exposure to ambient levels above the RfCs does not mean adverse health effects will occur, but the risk of an adverse effect increases above the RfCs. Conversely, given the very wide range in sensitivity in the community, hypersensitive individuals may not be fully protected. Because of the various uncertainties and different approaches for dealing with uncertainties, concentrations considered "safe" can differ widely between jurisdictions. This variation can increase further when the severity of effect is considered in deriving acceptable levels.

Table C.2 shows the 10-point scale employed by the US EPA for assessing the severity of adverse health effects. Also shown are the descriptors proposed for use in California for acute exposures to air toxics.

Table C.2: US national and California effects and severity levels

US EPA severity level	Effect category	Effect	California severity level
0	NOEL	No observed effects	Mild

1	NOAEL	Enzyme induction or other biochemical change consistent with possible mechanism of action, with no pathological change in organ weights.	Mild
2	NOAEL	Enzyme induction and subcellular proliferation or other changes in organelles, consistent with possible mechanism of action, but no other apparent effects.	Mild
3	NOAEL	Hyperplasia, hypertrophy, or atrophy, without changes in organ weight.	Mild
4	NOAEL/ LOAEL	Hyperplasia, hypertrophy, or atrophy, with changes in organ weight.	Mild
5	LOAEL	Reversible cellular changes including cloudy swelling, hydropic change, or fatty changes.	Mild/severe
6	(LO)AEL	Reversible or necrotic tissue changes with no apparent decrement in organ function.	Severe
7	(LO)AEL/ FEL	Reversible slight changes in organ function.	Severe
8	FEL	Pathological changes with definite organ dysfunction that are unlikely to be fully reversible.	Severe
9	FEL	Pathological changes with severe organ dysfunction and long-term sequelae.	Severe
10	FEL	Life-shortening or death.	Life-threatening

The Californian equivalent of the RfCs are the reference exposure levels (RELs) used by the California ARB. These have been developed for both chronic exposure, as well as for acute exposure.

The acute REL as used by the California Air Resources Board is the concentration that is not likely to cause adverse effects in a human population (including sensitive subgroups) for a 1-hour exposure on an intermittent basis. California follows the US EPA in considering intermittent exposures as exposures of less than 24 hours duration and occurring no more frequently than once a month.

In determining the acute RELs, factors that need to be considered are the duration of exposure, the frequency and pattern of exposure (how often and at what intervals is exposure likely to occur), and the background or chronic levels of exposure. In the absence of data to the contrary, some common assumptions made are that:

- each exposure can be considered an independent event, that is, no sensitisation occurs
- chronic exposure levels are negligible compared to acute levels
- the relationship between exposure duration and exposure level is described by "Haber's Law": $C^n \times T = K$, where n is a constant (ranging from 0.8 to 4.6) characteristic of the air toxic being considered. (This allows RELs derived from different exposure duration to be standardised to a single exposure duration (averaging time).

Health data can come from animal studies, opportunistic data from accidental or occupational human exposures, controlled human exposure studies, or epidemiological studies. Data on the combined effects of simultaneous or sequential exposure to different chemicals (synergistic, additive, or antagonistic) are rarely available and difficult to interpret. Therefore, in the absence of data to the contrary, the assumption made in the "Hot Spots" programme in California is that the effects of chemicals that affect the same biological system are additive and a hazard index (HI) is derived. The HI is the sum of Ci/RELi, where Ci is the ambient concentration of the ith species. The target systems considered in the hazard

index are the haematological system, cardiovascular system, the nervous system, the eyes, the alimentary tract, the immune system, the reproductive system, the respiratory system, and the skin.

Benchmark concentrations (BCs) are sometimes used to describe dose-response relationships. The BC is defined as the 95% lower confidence limit concentration expected to produce a response in 5 out of every 100 subjects exposed to that dose. The BC can be derived from the LOAEL by assuming a log-normal dose-response curve. RELs can then be derived by applying the relevant uncertainty factor. Distributions other than log normal are used by different groups, as are different definitions of BC (1%, 10%, or other response intervals), leading to different BCs.

In the absence of data, and sometimes for practical reasons, occupational exposure limits are used to derive "acceptable" concentrations by applying various factors to the occupational exposure limits. Occupational exposure limits represent time-weighted average concentrations (TWA), which workers can be repeatedly exposed during a 40-hour working week without adverse effect. In New Zealand, workplace exposure standards (WES) are set by the New Zealand Department of Labour.

The method for deriving "acceptable" concentrations from the WES-TWA is outlined in the NIWA Hazardous Ambient Air Contaminants (HAAC) database. Following the method developed by the UK Expert Panel on Air Quality Standards, NIWA derives Recommended Maximum Ambient Concentrations (RMAC) by dividing the WES-TWA (or the American Conference of Government Industrial Hygienists TWA where these are lower) by:

- a factor of 10 to extrapolate from a working lifetime (~77,000 hours) to a chronological lifetime (~660,000 hours)
- a second factor of 10 to compensate for differences in response between healthy workers, and sensitive individuals.

The RMAC is the maximum running annual average concentration that is considered protective against adverse health effects. They are intended for use as an indicator of environmental effect after the best practicable means for mitigating emissions has been applied. NIWA notes that meeting the RMAC does not guarantee protection for all exposed people, because of the very wide range in susceptibility of individuals in the community, but the risk is considered minimal.

In similar fashion, the Victorian Environment Protection Authority uses values of one-thirtieth of the occupational exposure standards as 3-minute average design ground level concentrations (DGLCs) in assessing potential emission sources. The DGLCs are not strictly comparable to the RMACs because of the different averaging times.

The non-threshold approach assumes that there exists no threshold for adverse effects: there is a finite probability that some adverse effect will occur as a result of exposure at any level. This approach is usually applied to carcinogens. The aim of the dose-response evaluation is to determine a curve that relates the exposure dose or concentration to the probability of an effect occurring at that dose. The results of evaluations of carcinogenic risks are usually expressed as unit risk factors. The unit risk is the additional risk of cancer incidence in a population from continuous lifetime exposure to a concentration of 1 $\mu g/m^3$ in the air breathed.

As is the case for non-threshold effects, data used for deriving unit risk factors can come from animal studies, opportunistic data from accidental or occupational exposures, or epidemiological studies. The basic assumption made in estimating cancer risks is that the risk increases cumulatively with dose; that is, exposure for 10 years to 10 $\mu g/m^3$ of pollutant increases the risk by the same amount as exposure for 5 years to 20 $\mu g/m^3$. Differences in unit risk factors derived by different groups arise from differences in the:

- treatment of, and weight given to, different studies
- methods used for extrapolating from animal to human responses
- assumptions made about the shape of the dose-response curve, which is normally only available for a narrow dose range at high dose levels
- base incidence of cancer, which can be different in different populations.

The International Agency for Research on Cancer (IARC) classifies the cancer potential of chemicals, based on the weight of evidence. The IARC classification is shown in Table C.3. Also shown is the equivalent US EPA classification. Because IARC and US EPA assess the weight of evidence in slightly different ways (different descriptors), the equivalence is approximate only. Chemicals classed as 1 and 2A, or A and B1 are normally considered carcinogens with zero threshold for effects.

Table C.3: IARC classes of carcinogenic chemicals and US EPA equivalents

IARC class	Descriptor (IARC)	US EPA class
1	The agent (mixture) is carcinogenic to humans; the exposure circumstances entail exposures that are carcinogenic to humans.	A
2A	The agent (mixture) is probably carcinogenic to humans; the exposure circumstances entail exposures that are probably carcinogenic to humans.	B1
2B	The agent (mixture) is possibly carcinogenic to humans; the exposure circumstances entail exposures that are possibly carcinogenic to humans.	B2, C
3	The agent (mixture or exposure circumstances) is not classifiable as to humans.	D
4	The agent (mixture) is probably not carcinogenic to humans.	E

The US EPA also categorises the cancer hazard of chemicals from inhalation as high, medium and low, by considering its carcinogenic potency and weight-of- evidence grouping (US Environment Protection Agency, 1998a). The carcinogenic potency, ED10, of a chemical is the estimated dose associated with an increased cancer incidence 10% over background. Table C.4 shows the rating system.

Table C.4: US EPA cancer hazard ranking

US EPA (weight-of-evidence) class	Potency group 1 (1/ED10 > 100)	Potency group 2 (1/ED1 to 100)	Potency group 3 (1/ED < 1)
A	High	High	Medium
B1 or B2	High	Medium	Low
C	Medium	Low	Low
D	No hazard ranking	No hazard ranking	No hazard ranking
E	No hazard ranking	No hazard ranking	No hazard ranking

Practical application

For pollutants for which a threshold exists for given health endpoints, it is possible (at least in theory) to establish pollutant levels at which everyone is protected. The various guidelines (RfCs, RELs, RMACs, etc) reflect more or less conservative assumptions in interpreting and extrapolating health data pertinent to those health endpoints.

By contrast, for pollutants where there is no identifiable threshold level, it is not possible to provide an absolute level of protection for everybody. In developing guidelines for these pollutants the risk estimates from the risk assessment are compared with a nominal "acceptable risk". It follows that such guidelines entail the acceptance of some level of adverse effects. Different guideline values arise not only because different risk assessment methodologies are used, but also because there are no universally accepted "acceptable risk" levels. The US EPA considers the acceptable risk to lie in the range 10^{-4} to 10^{-7}, and different values are used for different programmes. For example, the US EPA Office of Air and Radiation Safety uses 10^{-4} for individual risk and 10^{-6} for population risk.

Emission control standards

Although the amount of data is not extensive, monitoring in New Zealand indicates that levels of PM_{10} and of some VOCs, notably benzene, are close to or above international guidelines and standards in some major cities, and possibly in others. Based on emissions inventories and other data, it is clear that domestic solid-fuel combustion and motor vehicles are the major contributors, and therefore the major targets for enhanced control programmes.

Most international control programmes for motor vehicles are aimed at reducing photochemical smog. Since photochemical smog is formed by reactions involving volatile organic compounds (VOC) and oxides of nitrogen, control of either or both of these precursors is required to achieve smog reductions. Numerous atmospheric reactions involving many intermediate species are involved in smog formation. The process is driven by ultraviolet energy, and is highly non-linear.

Initial approaches for reducing smog levels focused on controlling VOC emissions, as this was considered more cost-effective than controlling emissions of nitrogen oxides. As the gains in smog reductions were eroded due to increases in the number of vehicles and kilometres travelled, it became necessary in some locations to control both precursors to maintain and increase the gains achieved. Control of smog precursors are now largely achieved through catalytic converters. Two-way catalytic converters which only tackled the VOC emissions have been superseded by three-way catalysts (which reduce both VOC and nitrogen oxides), which are now standard on new vehicles built for the United States, Japanese, and European markets. Table C.5 lists current and existing vehicle emission standards (Motor Vehicle Environment Committee, 1999). Other technologies are also emerging such as hybrid vehicles, that use both petrol or diesel fuel and electricity.

The VFECS outlines the initiatives for tackling vehicle emissions that are being pursued in New Zealand.

Table C.5: Emission standards for petrol vehicles

Current and future standards	Date of implementation	Limits on emissions			
		Carbon monoxide (g/km)	Hydrocarbons* (exhaust) (g/km)	Nitrogen oxides (g/km)	Hydrocarbons (evaporative) (g/test)
ADR37/01	1997–9	2.1	0.26	0.63	2
UN ECE			Combined hydrocarbons and nitrogen oxides 0.5		
Euro 2	1996	2.2			2
Euro 3	2000	2.3	0.2	0.15	2
Euro 4	2005	1.0	0.1	0.08	2
US EPA					Complex

Tier 1 Tier 2	1994-6 2004	2.1 1.0	0.25 0.08	0.25 0.12	requirements are being progressively introduced

* The original table has been simplified by treating hydrocarbons, NMHC, and VOC as equivalent, and omitting other detailed notes. For full details, see the Motor Vehicle Environment Committee (MVEC) report cited.

Diesel heavy vehicle standards are listed in Table C.6 (Motor Vehicle Environment Committee, 1999). For details of the units used and how they compare refer to the MVEC report.

Table C.6: Emission standards for diesel vehicles

Standard	Gross vehicle mass/ engine category	Oxides of nitrogen			Particulates (g/kWh)		
		ADR 70/00	1996	2000	ADR 70/00	1996	2000
ECE 49/02 (Euro 1,2,3)	>85 kW	8.0	7.0	5.0	0.36	0.15	0.10
US EPA 91, 94, 98	> 3.9 tonnes	6.7	6.7	6.7	0.33	0.13	0.13
Japan 94, 94, 97–2000	> 2.5 tonnes	7.8	6.8	6.8	0.96	0.96	0.25

Emissions of PM_{10} from wood heaters and open fire places (Victorian Environment Protection Authority, 1999b) are as follows:

Domestic home heating method	**PM_{10} emissions (g/kg of wood burned)**
Open fires	17.3
Older wood heaters	12.0
Heaters complying with the Australian Standard	5.5

As can be seen from the above data, replacing older inefficient heaters with modern heaters built to appropriate standards can reduce particle emissions by two-thirds, other things being equal. The quality of wood used (dry, well seasoned, low-resin), also affects emissions, as does the method of operation. The most effective control starts with a well-designed heater, but must also be reinforced by proper operation.

Annex D: Background information on hazardous air contaminants

Contaminant	Health Effects	Classification		Unit Risk x 10-6			Various Guidelines (μg/m3) (annual averages unless otherwise stated)				
		IARC	US EPA (potency)	WHO	USEPA	CARB	UK	EC	TWA/100	WHO	US
Benzene	Haemotoxic, Genotoxic, Carcinogenic	1	A (M)	4.4 – 7.5	8.3	29	18 (now) 3.6 (goal)	10 (now) 5 (2010)	18	-	-
Toluene	CNS, Irritation of eyes, skin, & respiratory system	3	D	-	-	-	-	-	190	290 (1-week) 1000 (30-min)	400 (RfC)
Xylene	Neurological, Irritation of eyes, nose, & throat, Gastrointestinal	3	D	-	-	-	-	-	2400	950 4800 (30-min)	-
1,3 – Butadiene	Neurological, Irritation of eyes, throat, lungs, & nose, Mutagenic, Carcinogenic (?)	2A	B2 (M)	(RIVM: 7 – 33)	280	170	2.4	-	24	-	-
Formaldehyde	Irritation of eyes, throat, nose, & respiratory symptom, Nasal cancer	2A	B1 (M)	very low	13	6	-	-	9.2	100 normal 10 hypersensitive (30-min)	-
Acetaldehyde	Irritation of eyes, throat, nose, & respiratory system, Nasal cancer.	2B	B2 (L)	15 - 90	2.2	2.7	-	-	3600	2400 (24-hour)	9 (RfC)
Benzo(a)pyrene	Dermatitis, Photosensitisation, Eye irritation, Cataracts, Lung cancer (?)	1	B2 (M)	87000	-	-	-	-	-	-	--

Mercury*	CNS, Gastrointestinal, Respiratory system, Kidney	2B(m) 3(I)	C(m) D(I)	-	-	-	-	-	0.1(al) 0 25(I) 1.0(ar)	1(I)	0.3(I) (RfC) 0.3(I) (REL)
Chromium (VI) Chromium II,III	Respiratory, Gastrointestinal, Liver, Kidney, Immune system, Blood	1 3	A (H) D	11000 – 130000 -	1200 -	150000 -	- -	- -	0.1 – 0.5 5.0	- -	0.0023 -
Arsenic (Inorg) Arsine	Gastrointestinal, Haemolysis, Central & Peripheral NS, Eyes, Skin, Mucous membranes	1	A (H)	1500	4300	1500	-	-	0.1 1.7	- -	0.41 (REL) 0.055 (RfC)

* Abbreviations for mercury: Organic (o) = {methyl (m), aryl (ar), alkyl (al)}; Inorganic (I) = elemental and other inorganic compounds

Annex E: Guidelines and unit risk levels for hazardous air contaminants

Contaminant	**Recommended Guideline Values (μg/m^3)**				**Implied Risk (per 10^6)**	**Levels for risk of 1 in 10^6 (μg/m^3)**	**Ambient levels (annual average, or as specified)**	**Comment**
	Ambient (annual average)	Basis	**Design (1-hour average)**	Basis				
Benzene	10 (now) 3.6 (2010)	EC(now) UK (long term goal)	22	Annual (2001) converted	44 – 75 (WHO) 16 (WHO)	0.13 – 0.23	~ 7 (urban) 20+ (traffic)	
Toluene	190	TWA/100	500	Odour	-	-	<20 (urban) ~50 (traffic)	
Xylene	950	WHO (health)	1000	Odour	-	-	<20 (urban) 50+ (traffic)	
1,3 –Butadiene	2.4	UK	15	Annual converted	17 – 72 (RIVM) 670 (US EPA)	0.03 – 0.14 0.0036	~1 (24-hour)	
Formaldehyde	15	WHO (health) converted	20	WHO < design < Vic EPA	196 (US EPA)	0.077	12 (17-day) ~30 (1-hour)	
Acetaldehyde	30	WHO (health) converted	45	Odour (Vic EPA)	450 - 2700 (WHO) 66 (US EPA)	0.001 - 0.067 0.45	No NZ data US ~(2 – 4)	

Benzo(a)pyrene	0.0003	Risk of 2 - 3 in 10^5 assumed acceptable	Not specified	-	26 (US EPA)	0.00001	7 – 72 (24-hour)	
Mercury (organic) Mercury (inorganic)	0.13 0.33	TWA/100 TWA/100	0.8 2.0	Annual converted Annual converted	- -	- -	No urban data <50 (7-day)	
Chromium (VI) Chromium (other)	0.0011 0.11	Assume Risk of 1 in 10^5 is acceptable (between WHO & USEPA) 100 x Cr (VI)	0.0067 0.67	Annual converted Annual converted	12 – 140 (WHO) 1.3 (US EPA) -	0.000007 – 0.00009 0.00083	No NZ data	Ignores dietary intakes
Arsenic (Inorg) Arsine	0.0055 0.055	Risk of 1 in 10^5 assumed acceptable (between WHO & USEPA) RfC (US EPA)	0.033 0.33	Annual converted Annual converted	8.3 (WHO) 24 (US EPA) -	0.00067 0.00023 -	No NZ data	