

Benzene

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Benzene

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Preface

The National Environmental Health Forum has been established by the Directors of Environmental Health from each State and Territory and the Commonwealth with a secretariat provided by the Commonwealth Department of Health and Family Services.

The National Environmental Health Forum is publishing a range of monographs to give expert advice and guidance on a variety of important and topical environmental health matters. This publication is the second in the air series. A list of published monographs, and others in preparation, appears on page vi.

The Directors of Environmental Health, in expediting publication of this document, have undertaken targeted consultation only.

This booklet is one of a series of plain language summaries describing current knowledge on the occurrence and human health effects of a number of air pollutants. The series is currently being prepared by the Commonwealth Department of Health and Human Services as part of a process to explore options to reduce the exposure of Australians to these pollutants. Other booklets in the series are as follows:

- Ozone (photochemical oxidants)
- Nitrogen dioxide
- Sulfur dioxide

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Published monographs

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1. Guidance for the control of Legionella (1996)
2. Guidance on water quality for heated spas (1996)
3. Guidance on the use of rainwater tanks (1998)

Soil Series

1. Health-based soil investigation levels, 2nd edition (1998)
2. Exposure scenarios and exposure settings, 2nd edition (1998)
3. Composite sampling (1996)

Metal Series

1. Aluminium, 2nd edition (1998)
2. Zinc (1997)
3. Copper (1997)

Air Series

1. Ozone (1997)
2. Benzene (1997)
3. Nitrogen Dioxide (1997)
4. Sulfur dioxide (1999)

General Series

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2. Paint film components (1998)
3. Guidelines for the control of public health pests – Lice, fleas, scabies, bird mites, bedbugs and ticks (1999)
4. National Standard for licensing pest management technicians (1999)

Indigenous Environmental Health Series

1. Indigenous Environmental Health No. 1 (1999)

Exposure Series

1. Child activity patterns for environmental exposure assessment in the home (1999)

Counter Disaster series

1. Floods: An environmental health practitioner's emergency management guide (1999)

1. Introduction

Chemical nature and occurrence of benzene

Benzene is a hydrocarbon chemical consisting of six atoms each of carbon and hydrogen arranged in a ring structure. At normal ambient temperatures it is a liquid, but it evaporates rapidly at room temperature and is highly flammable. It has a characteristic aromatic odour and is slightly soluble in water (1.5 grams/litre at 20°C) but miscible with most other organic solvents.

Although benzene is a naturally-occurring chemical, found in crude petroleum at levels of up to 4 g/litre, almost all the benzene found at ground level comes from human activities. It is produced in extremely large quantities worldwide (14.8 million tonnes) and emissions arise during the processing of petroleum products, in the coking of coal, during the production of toluene, xylene and other aromatic compounds, and from its use as a component of petrol and in other consumer products as a chemical intermediate.

Benzene is naturally broken down by chemical reactions in the atmosphere. The length of time that benzene vapour remains in air varies between a few hours and a few days depending on environmental factors, climate and the concentration of other chemicals in the air, such as nitrogen and sulfur dioxides.

Benzene can be removed from air by rain, leading to the contamination of surface water and groundwater. However, due to its volatility, benzene does not remain in water for more than a few hours and it is not bound to sediments. If benzene is buried in soil, it may be transported into groundwater. In surface water it is rapidly degraded by aerobic bacteria (within hours), and in groundwater more slowly by anaerobic bacteria (weeks/months). It has not been shown to bioaccumulate in aquatic or terrestrial systems.

Human exposure and health effects

Benzene is a component of petrol; it is also formed in the motor engine combustion process and emitted in the exhaust. In newer cars with catalytic converters (manufactured in Australia since 1986) much of the benzene is removed from the exhaust gases but vehicle exhaust remains the major source of benzene in the air. Cigarette smoke contains benzene and is an important source of intake for active smokers. Some furnishings, solvents and adhesives also contain benzene and can contribute to the intake of benzene when used indoors. Trace amounts are found in food and drinking water.

It is known that benzene causes a number of adverse health effects. In the past, several groups of workers in the shoemaking, leather, rubber, adhesive and chemical industries were exposed to high concentrations of benzene. Current understanding of the health effects of benzene are mainly derived from follow-up studies of these workers supplemented with information obtained from studies of laboratory animals.

The most frequently reported health effect of benzene is bone marrow depression leading to anaemia (reduced red blood cells), leukopenia (reduced white cells) and thrombocytopenia (reduced blood platelets). In the workplace these effects have been recognised as the first stage of benzene toxicity and the higher the level of exposure the greater the likelihood of observing decreases in circulating blood cells (dose-response).

Benzene is also a well-established human carcinogen. Laboratory studies have shown that it exerts its effect by damaging the genetic material of cells. After long-term exposure at lower levels of benzene than those that cause reductions in blood cells, workers have shown a small but definite increase in the risk of developing certain types of leukaemia. Studies in laboratory animals have shown similar effects.

The observation of any of these effects, regardless of the level of exposure, indicates a need for control over benzene exposures. As with other genotoxic carcinogens, it is not possible to determine a concentration to which people might be exposed that carries absolutely *no risk*. As leukaemia is an uncommon disease, however (lifetime risk from all leukaemias of 1 in 135 persons and of acute myeloid leukaemia of 1 in 474, for Australia in 1990), it is possible to determine a level of benzene exposure that, for all practical purposes presents such a low risk to the population, effects are unlikely to be detected by any practicable method.

While the National Health and Medical Research Council (NHMRC) has not recommended any specific air quality goals for benzene, it is widely accepted that exposure should be minimised. This booklet summarises the scientific evidence on the health effects of benzene and possible sources of exposure of the Australian population. It will help health, education and environmental professionals to gain a better understanding of the issues relating to the need to minimise benzene exposure and the options for achieving this goal.

Box 1:

UNITS

Benzene concentrations are expressed as **micrograms per cubic metre ($\mu\text{g}/\text{m}^3$)** or as **parts per billion (ppb) or parts per million (ppm)** — which are measurements of the airborne concentration of a substance by volume.

1 ppb is one part, by volume, in one thousand million parts of air, or 1 in 10^9

The conversion of ppm to $\mu\text{g}/\text{m}^3$ is dependent on temperature and pressure.
At 20°C and 1013 Kpascals, 1 ppb is equivalent to 3.24 $\mu\text{g}/\text{m}^3$ of benzene.

2. Sources of benzene

Benzene is present, at least in trace levels, in all parts of the environment resulting in the exposure of all humans. Exposure in the general population is primarily to airborne benzene but small amounts may be ingested with food and water.

Atmospheric benzene

There are no well-defined natural sources of atmospheric benzene and almost all the benzene observed in air at ground level results from human activities, in particular from the use of petrol and oil.

Benzene is present in petrol and can escape into the air, particularly from vehicle fuel systems and from filling stations. The proportion of benzene currently in both leaded and unleaded petrol can be up to 5% by volume (which is the maximum allowable concentration in the European Union). Conversion from leaded to unleaded petrol over the last decade has not resulted in an increase in the amount of benzene in petrol.

The major source of exposure for the general population is from vehicle exhausts because, in addition to the benzene actually present in petrol, it is also produced by chemical reactions during combustion in the engine. In the United Kingdom in 1991, 78% of atmospheric benzene was derived from petrol engine exhausts (Table 1). A similar breakdown is not available for benzene in Australia. Emissions inventory data from the Sydney region in 1992 indicate that for total volatile organic compounds, 40% are derived from mobile (vehicular) sources. A study by CSIRO at Point Phillip Bay in 1988 also indicated that for benzene vehicular sources contributed 45% of the total emissions.

From this it is clear that if levels in ambient air are to be reduced, control of vehicle emissions is essential.

Table 1: Sources of benzene emissions in the United Kingdom, 1991

	<i>Tonnes/year^a</i>	<i>%</i>
Petrol engine exhausts	39250	78
Diesel engine exhausts	4550	9
Petrol evaporation from vehicles ^b	3350	7
Petrol refining and distribution	1350	3
Combustion of oil, wood, etc	950	2
Gas leakage	400	<1
Other industrial processes	16–350	<1

^a *Figures rounded to the nearest 50 tonnes*

^b *Evaporation is expected to be higher in Australia because of higher temperatures.*

Source: UK Department of the Environment, Expert Panel on Air Quality Standards, Benzene, HMSO, London, 1994.

Australian car fleet

Both combustion and evaporative emission rates of toxic hydrocarbon species, including benzene, have been measured in the Australian car fleet. The per vehicle drive-cycle (combustion) emission of benzene is shown in Table 2 as an average of the three phases of the Australian Design Rule (ADR) 37 prescribed drive cycle (cold transient, cold stabilised and hot transient). Overall, the average per vehicle emissions of benzene from post-1986 vehicles are about 30% of those from pre-1986 vehicles. Cold-start emissions have been shown to make the largest contribution to total emissions because the fuel-rich conditions that occur during ignition lead to a higher proportion of unburnt fuel in the exhaust. Also, catalytic converters generally have poorer efficiencies during the first few minutes after the engine is started and, for catalyst-equipped vehicles, the cold transient (start-up) emissions represent a larger proportion of the total emissions than for non-catalyst vehicles.

Evaporative emissions have been measured in standard sealed housing evaporative determination (SHED) tests in which evaporation due to the diurnal heating/cooling cycle ('heat build') and evaporation from the carburettor and petrol lines during the period immediately after a normal trip ('hot soak') were measured. For the cars tested in this way the pre-1986 cars showed higher evaporative losses than the newer cars

Table 2: Average emissions of benzene from Australian cars

<i>Age of vehicle</i>	<i>Number tested</i>	<i>Average emissions (mg/km/vehicle)</i>
Pre-1986	10	132
Post-1986	56	41
Ratio of post- to pre-1986 emissions		31

Source: CSIRO 1996.

Concern has been expressed that the use of unleaded petrol in older cars may lead to higher levels of toxic emissions, including benzene. However, tests have shown that when pre-1986 vehicles are refuelled with unleaded petrol, the levels of benzene emissions are reduced compared with those when leaded petrol is used (Table 3). These tests also showed that most of the increase in emissions with leaded petrol was in the cold transient phase of the drive cycle (double the emissions for leaded compared to unleaded petrol) with less difference in the other two phases.

Table 3: The effect on benzene emissions of leaded and unleaded fuel use in pre-1986 vehicles

<i>Fuel</i>	<i>No. of cars</i>	<i>Average benzene emission (mg/km/vehicle)</i>
Leaded	6	154
Unleaded	6	119

Source: CSIRO 1996.

Other sources of benzene

While petrol exhaust is the major source of benzene in outdoor air, especially in urban areas, there are other sources of human exposure, some of which contribute significantly to an individual's total intake of the chemical. Cigarette smoke contains benzene and may be the main source of exposure for a heavy smoker. The World Health Organization (WHO) has estimated a benzene intake of 30 µg per cigarette, so that people who smoke 20 cigarettes per day have a daily benzene intake of 600 µg. For these individuals cigarette smoking will be a major source of intake, exceeding intakes from ambient air and other sources (food and water). Passive smoking will also make a small contribution to benzene intake of non-smokers if they are exposed in confined spaces.

Occupational exposure

Occupational exposure to benzene occurs in the petroleum industry and other chemical and manufacturing industries where benzene-containing adhesives, solvents or other products are used. In recent years improved technology and worker practices have reduced the level of exposure to benzene in the industrial workplace. WHO has reported that when appropriate engineering controls are in place, exposure of workers involved in the production and handling of benzene and benzene-containing materials varies from non-detectable to 5 ppm during the working day.

3. The effects of benzene on human health

Several groups of workers in the shoe-making, leather, rubber, adhesive and chemical industries have, in the past, been exposed to high concentrations of benzene (several hundred ppm). Current understanding of the effects of benzene on human health is largely derived from follow-up studies of the effects of health of these cohorts of workers, supplemented with information obtained from studies with laboratory animals.

Benzene is readily absorbed into the body when breathed into the lungs or taken orally, about half of it being retained. As it is more soluble in fat than in water, it is distributed in the body to fatty tissues including the brain and the bone marrow. In the absence of further exposure, benzene is eliminated by chemical breakdown in the body or by metabolic excretion in the urine; 80% being eliminated within about two days.

Toxic effects of benzene

The distribution to fatty tissue is reflected in the toxic effects of benzene, which are shown in Table 4. Acute exposure to extremely high concentrations may cause narcotic or anaesthetic effects and deaths of workers have been recorded after exposures to concentrations of several thousand ppm. Very high levels of exposure (well over 5000 ppm) on repeated occasions have led to the development of severe and sometimes fatal damage to the blood-forming elements of the bone marrow, preventing the manufacture of essential blood cells. Such serious consequences are not, of course, a risk associated with exposure to the concentration

of benzene observed in ambient air and will not occur in workers except as a result of unforeseen and accidental exposure to very high concentrations.

Table 4: Toxic effects of benzene

<i>Concentration range of benzene</i>	<i>Toxic effects</i>
20 000 ppm; 5–10 minutes	Central nervous system depression, cardiac arrhythmia, respiratory failure and death
50–150 ppm; 5 hours	Headaches, lassitude and general weakness
> 120 ppm; 1–22 years	Increases in the number of chromosomal aberrations
> 50 ppm; 1 year (> 50 ppm–years)	Bone marrow toxicity (anaemia, leukopenia, thrombocytopenia)
> 30 ppm	Increasing susceptibility to allergens
20–50 ppm; 10 years (200–500 ppm–years)	Increased incidence of leukæmias (especially non-lymphocytic leukæmia)
3–6 ppm	Some evidence of decreased immune function
1 ppm; 40 years (40 ppm–years)	Determined by the WHO to be associated with no statistical increase in leukæmia deaths.

Source: Expert Panel on Air Quality Standards (UK) 1994; WHO 1993; Wadge 1996.

At concentrations occurring in the ambient atmosphere, benzene does not have short-term or acute effects. Of the effects of long-term exposure to benzene described above, however, the effect that is of most concern for human health is leukæmia, in particular, several types of leukæmia known collectively as the non-lymphocytic leukæmias (or myeloid leukæmias).

The mechanism of benzene-induced leukæmia in humans is not understood. Benzene or its metabolites cause chromosome aberrations in humans and laboratory animals and such chromosomal rearrangements are relevant steps in the carcinogenic process. Several types of neoplasms have been reported to be associated with benzene exposure in rats and mice after oral dosing or inhalation exposures. These tumours were primarily of epithelial origin (eg liver, mammary gland, nasal cavity) and lymphomas and leukæmias were observed less frequently in rats and mice.

The neurotoxicity and immunotoxicity of benzene have not been well studied in either experimental animals or humans. Benzene can pass through the placenta and cause fetal toxicity. However, there is no evidence that it is teratogenic (ie that it causes an increased risk of fetal abnormalities).

Leukæmia incidence in exposed workers

One of the first reports of a possible association between benzene and leukæmia was made in the late 1960s and early 1970s when a series of cases of leukæmia were seen from an estimated population of 28 500 shoemakers in Istanbul. The workers had been exposed for periods of 4 months to 15 years to adhesives and solvents containing high levels of benzene (reported to be 150–650 ppm). Bone marrow toxicity (ie reduced production of blood cells) was evident in the workers and a leukæmia incidence rate was estimated as 14 per 100 000 compared to 6 per 100 000 for the general population.

The reliability of the Istanbul shoemaker data is questionable because the degree of exposure to other chemicals was not monitored and there were other uncertainties about the methods used in the study. Since this time, however, a series of studies of groups of workers in the synthetic rubber and petroleum industries has been conducted, in which lower exposures to benzene were reported. These studies have confirmed an increased risk of cancer of the lymphopoietic and haematopoietic system at benzene levels that do not cause bone marrow toxicity. A summary of these studies is shown in Table 5.

The most commonly found association has been with increases in non-lymphocytic leukaemia. Excesses of other leukaemia cell types and multiple myeloma have also been reported although these have not always reached statistical significance. A possible role for benzene in other cancers has been suggested but evidence is limited.

Dose threshold for benzene effects

Although none of the published reports for leukaemia incidence in cohorts of workers are wholly reliable, it is evident that the risk of leukaemia in industrial workers has been related to their calculated lifetime exposure — the more benzene they were exposed to, the greater the risk. However, there are problems in extrapolating from the available human study data to possible effects on the general population since these studies have been carried out on workers exposed to benzene at concentrations considerably higher than those found in ambient air.

Exposure studies suggest that the lowest cumulative benzene exposure level at which statistically significant excesses of leukaemia deaths were observed was about 50 to 500 ppm-years, or about 2 to 17 ppm for a typical working life of 30 years. Exposure estimates from the Goodyear Pliofilm study (Table 5) have been interpreted by some authors to suggest effects at a time-weighted average exposure of about 2 ppm, while other authors have suggested that the effects occurred at exposures a magnitude higher.

Table 5: Leukæmia deaths in workers occupationally exposed to benzene

<i>Study</i>	<i>No. of subjects</i>	<i>Health outcomes</i>
Goodyear Pliofilm Plant (US rubber manufacturing plant)		
Departments using benzene, at least one day, 1940–65; deaths occurring after 1950.	1165	9 leukæmia deaths (4 myeloid leukæmia) with a dose-related significant excess of leukaemia among workers exposed above 200 ppm-years (SMR 1186, 95% CI 133 to 4285).
Chemical workers (Dow Chemical Co, US)		
Organic and resin synthesis	888	3 leukæmia deaths (all myeloid leukæmias) were recorded with an estimated benzene exposure of 1.5 ppm-years (SMR162, 95% CI 33 to 461).
Chemical workers		
At least 6 months exposure in 7 US chemical plants, 1946–75	3536	Significant dose-response relationship (cf an internal unexposed group of 3074 workers) for lymphatic and haematopoietic cancers (cumulative exposure categories of <15, 15–60, >60 ppm-years). In this study there were no myeloid leukæmias reported.
Chinese factory workers		
Painting, shoe-making, leather, rubber, adhesive and organic synthesis factories, 1972–81.	74 828	82 cases of haematopoietic neoplasms and related disorders, including 32 cases of acute leukaemia, compared to 13 haematological malignancies including 6 leukæmia cases in 35805 unexposed workers.
Petroleum industry workers		
US and UK, 1937–89	208 741	No increase in leukæmia(myeloid or other cell types) — attributed to relatively low levels of benzene exposure (81% below 5 ppm-years)
UK distribution workers, 1950–75	23 000	91 cases of leukæmia identified with no overall dose-response relationship; 32 cases of acute myeloid leukæmia(AML) with some evidence (not significant) of an increased risk for AML for cumulative exposures between 4.5 and 45 ppm-years compared to <0.45 ppm-years.
Oil and gas production workers, US		
Workers with exposure histories back to 1920s	353	69 leukæmia cases (OR 2.1, 95% CI 0.95–4.8 for all forms of myelogenous leukæmias; OR 3.2, 95% CI 1.1–9.2 for AML). Association with AML strongest in subjects exposed for 32 years or more. No information available on levels of benzene exposure.
Petroleum industry workers in Australia, 1937–89		
	15 000	12 leukæmia cases (cf 3.6 expected; SMR 340, 95% CI 170 to 590) of which 7 were myeloid leukæmias. 6 deaths from leukæmia(4 myeloid) cf 3.9 expected (SMR 160, 95% CI 60 to 340). Lower overall mortality rate with comparable incidence of all cancer compared to national rates but a statistically significant excess of deaths from multiple melanoma also observed.
Coke oven factory workers, UK, 1980s		
	6520	In this study fewer leukæmia deaths were observed (5) than expected (12). Mean time-weighted average concentrations for coal product workers was approximately 1.3 ppm
SMR:	Standardised mortality ratio: the ratio of the observed number of deaths for cases of disease in the occupationally exposed cohort to the expected number in a control group of similar unexposed persons, multiplied by 100. For control groups the studies have used either the national populations or workers in other industries. Values of >100 indicate an increased incidence of deaths in the exposed population, providing the lower value in the confidence interval is greater than 100.	
OR:	Odds ratio. Values greater than 1 indicate an increase in prevalence, providing the lower value in the confidence interval is >1.	
CI:	Confidence interval: 95% CI is the range in which, allowing for variability in study populations, there is a 95% chance of the true result falling.	

Both the Pliofilm cohort and the United States chemical workers showed evidence of increasing risk with increasing exposure, although the excesses of observed deaths were not always statistically significant among the lower exposure groups. The lowest exposure level in the chemical workers study was 0.5 ppm for a 30-year working life

The recent study of marketing and distribution workers in the petroleum industry in the United Kingdom (Table 5) provides a useful insight into leukaemia risks from relatively low-level exposures to benzene (815 of the subjects were exposed to less than 5 ppm-years). Although an excess risk of acute myeloid leukaemia (AML) was observed for cumulative exposures between 4.5 and 45 ppm-years, this did not reach statistical significance and doubt remains as to whether the risk of AML is increased by cumulative exposures less than 45 ppm-years. This equates to a time-weighted average concentration of 1.5 ppm for a typical 30-year working life.

Evidence from Chinese factory workers suggests that statistically significant excesses of leukaemia have occurred at time-weighted average benzene exposures of about 2–5 ppm. This is not dissimilar to the time-weighted average exposure levels in the Pliofilm and chemical worker studies described above. However, given the high exposures for some members included in the Chinese studies, the implications for risks at lower levels need to be interpreted with caution.

WHO concluded that a time-weighted average of 1 ppm or less over a 40-year working life is not statistically associated with any increase in deaths from leukaemia. In the United Kingdom, the Expert Panel on Air Quality Standards (1994) determined that a level of 0.5 ppm over a working lifetime would not produce any detectable adverse health effect (see Section 4).

4. International standards, goals and guidelines

Standard setting for carcinogens

Two approaches have been used by regulatory authorities in different parts of the world to establish air quality standards for carcinogens. One is to develop a mathematical model of the exposure-response curve which can be used to extrapolate from the measured risks at occupational exposure levels back to estimate risks which might arise at much lower ambient levels. The model is then used to derive an exposure level that would result in a risk that is 'acceptable' to the regulatory authority. This quantitative approach relies upon assumptions about the shape of the dose-response curve and, although mathematical models appear to be precise, they can be quite misleading, since it is not uncommon for risk estimates to vary by orders of magnitude depending upon the particular mathematical model chosen. For these reasons, this approach is not recommended by the United Kingdom Committee on Carcinogenicity of Chemicals in Food, Consumer Products and the Environment.

Another approach to standard setting for carcinogens is to identify exposure levels in epidemiological studies that have not caused adverse effects upon health and to incorporate appropriate safety margins. This is the traditional approach for *non-carcinogenic* compounds, but it has been used in the United Kingdom to recommend an air quality standard for benzene (see below). This approach relies upon expert judgment, first to identify an exposure level at which effects are not expected, and second to apply appropriate safety margins.

United Kingdom standard

The safety margin approach has recently been used by the Expert Panel on Air Quality Standards (EPAQS) to recommend an air quality standard for benzene in the United Kingdom. EPAQS considered it was not possible to define an absolutely safe level of exposure to a genotoxic carcinogen (other than zero), and therefore identified an exposure

level that was considered to not cause any detectable effects over a working life in any feasible study. Safety margins were incorporated to reflect differences between working lifespan and chronological lifespan and also to protect potentially vulnerable members of society.

Using this method EPAQS has recommended an air quality standard in the United Kingdom for benzene of 5 ppb ($16 \mu\text{g}/\text{m}^3$) as a running annual average, with an objective of achieving the standard by 2005. This level was based on a determination that the increased risk of leukaemia in cohorts of workers exposed to 500 ppb (0.5 ppm) benzene over a working lifetime would be too small to detect in any feasible study (see Section 3). Benzene is currently measured (in the UK) with a sampling frequency and analysis of once per hour. The hourly values are then averaged for 365 days (366 in a leap year) ending at 23.59 on the previous day.

The United Kingdom panel pointed out that industrial workers studied for the effects of benzene exposure were generally fit, young and middle-aged males. However, the general population also includes children, pregnant women, the elderly and the sick, some of whom may be more sensitive to toxic chemicals. Also, the industrial cohort is potentially exposed to high concentrations for about 8 hours per day, five days per week for about 40 years but the general population is exposed to much lower concentrations throughout their lifetimes.

A safety factor of 10 was therefore applied to allow for whole-of-life exposure and a further factor of 10 to safeguard those people exposed to other causes of leukaemia, children, and those who may be more sensitive to toxic chemicals. This gives a value of 5 ppb, as a running annual average, which has been recommended as the air quality standard in the United Kingdom.

5. Benzene levels in Australia

Measurement and monitoring of benzene

The standard approach for measuring benzene in air is to test air sampled for specified time periods using gas chromatography. Another technique, known as differential optical absorption spectroscopy (DOAS), is also sometimes used. This method continuously records benzene concentrations across transects, which may be several hundred metres in length, to detect contributions from local sources. Despite the inherent differences between these two measurement techniques, concentrations in the same range have been recorded with each. It is possible to detect benzene at levels as low as 0.003 ppb in air ($0.01 \mu\text{g}/\text{m}^3$) and at even lower levels in soil and water. From a health point of view for benzene, short-term ambient concentrations are less meaningful than longer time-weighted averages, since risk of leukaemia is associated with lifetime accumulated exposure.

Unlike other pollutants, such as nitrogen dioxide and ozone, monitoring of benzene concentrations in ambient air in Australia is not carried out in a systematic way by environmental authorities. It has therefore not been possible to present a comprehensive picture of benzene concentrations and exposures in Australia. There is a clear need for more data on ambient levels and personal exposures in Australia, using nationally agreed methodology.

Benzene levels in Australian cities

Recent data collected from 17 urban background air quality monitoring sites in Sydney suggest a daily mean of about 1 ppb (Table 6). Higher mean concentrations (2–8 ppb) have been measured in the central business district and in spot samples near point sources of benzene pollution. Individuals would generally experience only intermittent exposure to levels higher than 1 ppb and time-weighted mean exposure to benzene in Sydney is likely to

be well below 5 ppb for most people. There is a slight seasonal variation in benzene levels with higher levels in winter than in summer.

Data from Melbourne (Table 7) all relate to samples near to known sources of pollution and show mean benzene levels of 2–8 ppb, although for some sampling sites, particularly near the Altona petrochemical complex, the range of concentrations show that 1-hour average levels as high as 39 ppb were recorded. Similar data were obtained from sites in Brisbane and Adelaide (Table 8). Fort Lytton in Brisbane was an exception and 30-minute average concentrations in excess of 400 ppb, and a mean of 5 ppb have been recorded using DOAS. This site is near an oil refinery and petrol storage tanks. Overall, levels close to roads and other sources generally exceed 1 ppb but rarely exceed 10 ppb.

Benzene concentrations in air were measured in 83 samples from 11 service stations in major cities across Australia in 1993, with 8-hour average samples collected adjacent to pumps. The mean concentration for these samples was 107 ppb (range 20–600 ppb). During re-fuelling, individuals will therefore be exposed briefly to relatively high concentrations of benzene.

Table 6: Summary of ambient concentrations of benzene in Sydney and surrounding districts

<i>Site</i>	<i>Date</i>	<i>Method</i>	<i>Sample size</i>	<i>Mean (ppb)</i>	<i>Range (ppb)</i>
5 sites near large point sources ¹	1992–93	GC (spot samples, 6–8 am)	37	2.5	0.1–6.8
17 EPA air quality monitoring sites ²	1995–96	GC (24 h samples)	49	1.0	0.1–6.5
On and near Castlereagh Waste Management Centre, Sydney outskirts ²	Aug–Oct 1995	DOAS (several weeks)	2 transects	< 1	–
George Street, Central Business District, 3 m above street level ³	Jan–Feb 1994	DOAS (24 h samples)	333 m transect	4.1	3.3–5.2
	June–July 1994		405 m transect	7.6	5.4–9.4
Kingsford Smith Airport, end of runway ⁴	15 May 1995	GC (spot samples)	6	5.6	4.4–6.6

GC: Gas chromatography

DOAS: Differential Optical Absorption Spectroscopy

¹ *Nelson, 1994*

² *Personal communication, M Dean, NSW EPA, 1996*

³ *Dawson et al, 1994*

⁴ *Duffy and Nelson, 1995*

Table 7: Summary of ambient concentrations of benzene in Melbourne and surrounding districts

<i>Site</i>	<i>Date</i>	<i>Method</i>	<i>Sample size</i>	<i>Mean (ppb)</i>	<i>Range (ppb)</i>
1200 m south of Altona Petrochemical Complex	March 1991	GC (spot samples over 2 days)	51	7.9	<5–20
100 m north of Altona Petrochemical Complex	March–May 1995	DOAS (500 m open path)	not specified	2.7 (3 month average)	0–39 (1 hour averages)
Tullamarine Freeway - Essendon Airport	Jun–July 1995	DOAS (500 m open path)	not specified	2.2 (2 month average)	0–11 (1 hour averages)
Spring Street, Melbourne, either 3 or 20 m from road	July–Aug 1994	GC (12 h means)	28 (duplicate samples)	3.0	0.2–8.5

GC: Gas chromatography

DOAS: Differential Optical Absorption Spectroscopy

Source: EPA Victoria

Table 8: Summary of ambient concentrations of benzene in Brisbane, Adelaide and near an oil refinery

<i>Site</i>	<i>Date</i>	<i>Method</i>	<i>Sample size</i>	<i>Mean (ppb)</i>	<i>Range (ppb)</i>
Fort Lytton National Park, Brisbane. Near 2 oil refineries and petroleum storage areas ¹	July–Oct 1992	DOAS (open path of 230 m; 0.5 h means)	4032	5.2	33–421 (20 highest 0.5 hour readings)
North Terrace, Adelaide ²	Dec 1994	DOAS (open path of 250 m; 1 h means)	13 days	8	0–26
Edwardstown industrial area, Adelaide ²	Nov–Dec 1994	DOAS (open path of 236 m; 1 h means)	34 days	19	0–80
Boundary, Shell oil terminal, Geelong ³	Feb and July 1995	not specified	9	1.9	<1–3

GC: Gas chromatography

DOAS: Differential Optical Absorption Spectroscopy

¹ *Wainwright and Williams, 1992*

² *Mitchell et al, undated*

³ *Personal communication, M Brown, Shell Australia Ltd, 1996*

Benzene levels inside vehicles

A series of determinations of benzene concentrations during commuter runs was carried out in Sydney between June and August 1995. A few tests were also conducted during freeway driving. Tests were conducted on pre-1986 vehicles without catalytic converters and on newer models with converters. Test runs took place with the windows closed and with vents partially open. The results are summarised in Table 9.

Table 9: Mean concentrations of benzene inside Sydney cars

<i>Test run</i>	<i>Catalytic converter</i>	<i>Mean in-vehicle conc.^a (ppb)</i>	<i>Mean ambient conc.^b (ppb)</i>	<i>in-vehicle/ambient ratio</i>	<i>In-vehicle conc. before ignition^b (ppb)</i>	<i>Mean in-vehicle conc. at midday^b (ppb)</i>
Commuter	No	48 (8)	1.8 (4)	27	9.5 (1)	32 (4)
	Yes	22 (16)	2.0 (8)	11	3.6 (5)	5.4 (8)
Freeway	No	6.2 (2)	2.0 (1)	3	nd	nd
	Yes	2.5 (4)	1.0 (1)	2.5	nd	nd

() *Number sampled*

nd *Not determined*

^a *Time-weighted average*

^b *Spot samples*

Note: Results are summarised from one non-catalyst-equipped and two catalyst-equipped vehicles on two commuter routes and one freeway run for each vehicle type. Tests were carried out between June and August 1995 with windows closed and vents partially open.

Source: Adapted from Duffy and Nelson, 1996.

During commuter runs there was a marked difference between the levels of benzene inside vehicles and the outside levels. The older, non-catalyst-equipped vehicles had benzene concentrations 27-times greater than ambient levels. In modern catalyst-equipped vehicles the levels were much lower than in the older vehicles, but were still 11-times greater than in the outside air. Cars fitted with catalytic converters may be better maintained than older vehicles so the possible role of the catalysts themselves in lowering benzene levels inside the vehicles is not clear. The fact that the benzene levels inside the vehicles before ignition were only slightly raised above ambient levels suggests that most of the benzene accumulating within these vehicles is derived from emissions (both from their own engines and other traffic) rather than from evaporative losses. The ambient levels for the commuter runs were taken from parks and are therefore more representative of 'background' urban levels rather than true concentrations in busy streets.

Benzene concentrations inside vehicles during freeway travel were about 10-times lower than those measured for the urban commuter trips during peak hour traffic. However, the mean levels were still about 3-times higher than spot measurements made at the roadside. Although this study only sampled a few vehicles (see Table 6), it agrees with findings from abroad that benzene can accumulate in vehicles. For the regular commuter, in-vehicle exposure may make an important contribution to daily and lifetime benzene exposure. This can be illustrated by calculating the lifetime exposure of an average person who commutes for one hour per day. Assuming the ambient level to be 1 ppb and the level inside their car during commuting to be 22 ppb then 25% of the lifetime exposure of 2 063 439 µg benzene will come from this source.

Data on benzene in other sources (eg food, water) are not available for Australia.

6. Population exposure estimates and risk assessment

Personal exposure estimation

The estimated daily intake of benzene in the United Kingdom is shown in Table 10. It varies widely between rural and city areas and for smokers and non-smokers.

Table 10: Estimated daily intake of benzene

<i>Source</i>	<i>µg/day</i>
Ambient air – rural	15
– urban	400
Cigarette smoke – 10 per day	300
– 20 per day	600
Food	100–250
Water	1–5

*Sources: ambient air: UK Department of the Environment
smoke, food and water: World Health Organization*

Similar figures are not available for Australia but using the figures shown in Section 5 on benzene levels in Australian cities and inside cars the daily exposure of Australians can be estimated for smokers and non-smokers.

Non-smoker spending a total of 1 hour per day commuting

Table 6 showed that at 17 sites in the Sydney Region, not adjacent to any point sources of emissions, the mean ambient concentration of benzene in 1995–96 as 1 ppb ($3.24 \mu\text{g}/\text{m}^3$). Assuming a mean concentration during commuting of 22 ppb ($71 \mu\text{g}/\text{m}^3$) (measured in Sydney with cars equipped with catalytic converters; see Table 9) and a respiration rate of $20 \text{ m}^3/\text{day}$ (or $0.83 \text{ m}^3/\text{h}$) the following calculations can be made:

(a) Intake from 1 h exposure to $71 \mu\text{g}/\text{m}^3$ (22 ppb) during commuting:

$$= 1 \text{ h} \times 71 \mu\text{g}/\text{m}^3 \times 0.83 \text{ m}^3/\text{h}$$

$$= 59 \mu\text{g benzene.}$$

(b) Intake from 23 h exposure to $3.24 \mu\text{g}/\text{m}^3$ (1 ppb) ambient benzene:

$$= 23 \text{ h} \times 3.24 \mu\text{g}/\text{m}^3 \times 0.83 \text{ m}^3/\text{h}$$

$$= 62 \mu\text{g benzene}$$

Thus, on working days, the daily intake of benzene from inhalation during the 1 hour of commuting will be similar to the total intake from the remaining 23 hours of the day whilst not travelling to or from work.

On a lifetime basis, assuming a working life of 40 years, with commuting for 5 days/week and 48 weeks/year:

(c) Intake during commuting throughout lifetime:

$$= 5 \text{ days/week} \times 48 \text{ weeks/year} \times 40 \text{ years} \times 59 \mu\text{g}$$

$$= 566\,400 \mu\text{g benzene.}$$

(d) Intake chronological life of 74 years, excluding commuting:

$$= [(24 \text{ h/d} \times 7 \text{ d/wk} \times 52 \text{ wk/y} \times 74 \text{ y}) - (1 \text{ h/d} \times 5 \text{ d/wk} \times 48 \text{ wk/y} \times 40 \text{ y})] \times 3.24 \mu\text{g}/\text{m}^3 \times 0.83 \text{ m}^3/\text{h}$$

$$= 1\,712\,655 \mu\text{g benzene}$$

(e) Total lifetime benzene intake for non-smoker spending 1 hour per day commuting (c + d)

$$= 2\,279\,055 \mu\text{g benzene}$$

Thus, for this individual, about 25% of the total amount of benzene inhaled over a lifetime would be derived from the relatively small amount of time spent commuting. Clearly, the

precise intake of individuals will vary considerably with the local conditions and no account is taken of reduced air intake during childhood or old age.

Intake of benzene from cigarette smoke

The WHO has estimated an intake of 600 µg benzene among people smoking 20 cigarettes per day, assuming an intake of 30 µg per cigarette. For these individuals, cigarette smoking will be the major source of intake, exceeding intakes from ambient air and other sources. Assuming a smoker carries on the habit for 40 years, the total lifetime intake of benzene from cigarette smoking would be:

$$600 \mu\text{g} \times 365 \text{ d} \times 40 \text{ y} = 8\,760\,000 \mu\text{g benzene.}$$

This amount would be added to the lifetime accumulated total as calculated above.

In the UK, it has been estimated that a non-smoker living in an unpolluted rural area may be exposed to as little as 120 µg benzene daily from all sources, while a 20 cigarettes per day smoker living in a city may be exposed to as much as 1250 µg daily.

Occupational exposure not causing any detectable health effects

The data summarised in Section 3 supports the United Kingdom expert panel's view that 500 ppb (1620 µg/m³), averaged over a working lifetime, is a concentration at which it would not be possible to detect effects in any practicable epidemiological study.

Assuming 30-years of working life, the following calculation shows the exposure that would occur among a population exposed occupationally to an average concentration of 500 ppb (1620 µg/m³) benzene:

$$\begin{aligned} &\text{Occupational exposure to 500 ppb (1620 } \mu\text{g/m}^3\text{) for 30 years:} \\ &= 8 \text{ h/d} \times 5 \text{ d/wk} \times 48 \text{ wk/y} \times 30 \text{ y} \times 1620 \mu\text{g/m}^3 \times 0.83 \text{ m}^3\text{/h} \\ &= 77\,448\,960 \mu\text{g benzene.} \end{aligned}$$

Therefore non-smokers exposed over a lifetime to benzene concentrations at ambient levels in Australian cities would accumulate a lifetime exposure to benzene more than an order of magnitude lower than that observed among workers exposed at a level not thought to be associated with detectable effects. Smokers of 20 cigarettes per day, although accumulating a considerably higher lifetime exposure than non-smokers, would only accumulate approximately 14% of the occupational exposure level not thought to be associated with detectable effects.

Control of carcinogens

Benzene is a genotoxic carcinogen. One of the fundamental principles of regulatory toxicology concerning the control of carcinogens in the environment is that any increase in population exposures to genotoxic carcinogens should be avoided and, where practicable, action should be taken to reduce exposure. Therefore all practicable measures should be taken to reduce ambient benzene levels in Australia to as low as possible.

For practical purposes, however, a concentration can be proposed at which the risks are exceedingly small and unlikely to be detected by any practicable epidemiological method. A safety factor can then be applied and a concentration derived, at which, risks are negligible. This is the approach taken by the Expert Panel on Air Quality Standards in the United Kingdom in recommending an air quality standard of 5 ppb as a running annual mean, for benzene.

7. Population exposure reduction options

It would appear from the limited environmental data on benzene concentrations in Australian cities that most people are exposed to concentrations below 5 ppb as an annual average.

However, there is a paucity of reliable data and further work is needed to:

- establish standard procedures for sampling and monitoring of benzene in air;
- establish representative levels of benzene in ambient air in urban and rural areas, and near 'hot spots' such as industrial sources and large petrol stations; and
- establish levels of benzene in indoor air and in vehicles.

A study of personal exposure in Australia that identifies the relative importance of different sources of benzene to the total exposure (ie traffic, within vehicles, refuelling vehicles, indoors, smoking) is also needed.

Continued work on quantifying past exposures in the study of Australian petroleum industry workers is important and will assist in estimating risks for both workers and the general population. In addition, study of cancer incidence data for Australia to investigate temporal and geographical trends in the incidence of leukaemias will assist in determining possible associations with benzene exposure.

Above all, measures should be taken to ensure that exposure to benzene does not increase in the general population and, where practicable, action should be taken to reduce exposure.

Control of vehicle emissions

As has already been discussed (Section 2) a major source of benzene emissions is in motor vehicle emissions. Benzene reduction options will need to focus on transport issues, although the attainment of reduced benzene levels will require the efforts of other industry sectors as well.

In the past, motor vehicles have been progressively targeted for precursor emissions control under the Australian Design Rules (ADRs). The ADRs are standards set for new vehicle compliance. A major design rule for motor vehicle emissions (ADR 37) was introduced in 1986, setting national emission limits for hydrocarbons and other pollutants. These standards must be met by all new vehicles and maintained for up to 5 years or 80 000 km. In 1995 ADR 37 was modified and the new standard (ADR 37.01) applies to new passenger vehicles from January 1997, new light commercial vehicles from January 1998 and all vehicles from January 1999. The revised limits in ADR 37.01 are still above the current United States limits but as most manufacturers employ 3-way catalysts (including a reduction phase) vehicles easily meet the ADR limits.

The attainment of lower emissions levels from motor vehicles will have social as well as economic impacts. A high degree of public support and participation is necessary to achieve this, as well as public education to increase understanding of the problem, reduce the level of car use and promote the use of models consuming less energy.

WHO recommendations

WHO has made the following recommendations for the protection of human health from benzene.

- Benzene and benzene-containing products, including petrol, should never be used for cleaning purposes.
- Systematic information on occupational and non-occupational exposure should be collected using the total human exposure approach, where possible.

- The health risk of low-level benzene exposure is not clearly understood. Exposure should, therefore, be avoided as much as possible.
- The occurrence of benzene in environmental media such as air and water should be evaluated.
- A search for less toxic solvents to replace benzene in industrial processes should be encouraged.

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